Teamwork in diagnosing and treating peripartum cardiomyopathy

James D Fett*
Peripartum Cardiomyopathy Projects, 2331 Mt. Hood Ct. SE, Lacey, WA 98503, USA

Introduction

When the diagnosis of peripartum cardiomyopathy (PPCM) can be made very early in its course the systolic heart function will be better preserved [1]. When the echocardiographic left ventricular ejection fraction (LVEF) is better, recovery outcomes will also be better [2]. Earlier diagnosis depends upon earlier recognition of symptoms that may be very similar to normal term pregnancy symptoms. When all of those involved in the care of new mothers, including the subjects themselves, are aware of a possibility for the development of pregnancy-associated heart failure in someone who has previously had perfectly normal heart function, then earlier diagnosis can easily be confirmed by echocardiography.

PPCM is still one of the leading causes of maternal mortality [3-5]. Unrecognized, it progresses at variable rates into severe heart failure threatening the life of both mother and unborn child or neonate. While not a common condition, it is also not rare. In the USA, incidence varies from approximately 1 case per 1500 live births in those mothers with African heritage to 1 case per 3000 in mothers without African heritage. Delay in diagnosis may lead to maternal mortality, newborn fatality, or survival of a mother with chronic cardiomyopathy and varying severity of heart failure for the rest of her life. Nevertheless, PPCM is a form of dilated cardiomyopathy with the greatest potential for full recovery, particularly when diagnosed early and treated appropriately, following evidence-based guidelines [2,6].

How can an earlier diagnosis of PPCM be made?

Greater awareness is already having an impact, leading to improving outcomes. Increasing attention must be given to enhance this awareness among all subjects with pregnancy as well as all their medical caregivers, including birthing center personnel, obstetrical nurses and aides, primary care physicians, emergency room physicians, obstetricians and cardiologists.

A self-test for heart failure in pregnancy is available for quantification of common symptoms [7]. Scores of 5 and higher continue to be validated as indicating the need for carrying out additional testing, including serum B-type Natriuretic Peptide (BNP) and/or echocardiography. It is important to work quickly in this assessment because, once triggered, the PPCM process may move very rapidly in which case the LVEF falls to levels that risk the subject’s susceptibility to ventricular tachyarrhythmias and sudden cardiac arrest. Once recognized, early treatment reverses the cardiomyopathic process and gives the greatest potential for avoiding those dangerous levels of systolic dysfunction, providing the greatest potential for subsequently returning to normal heart function.

What is the treatment of PPCM?

Evidence-based “Guidelines” for the initial treatment of heart failure with reduced LVEF include diuretics, beta-blockers (BB) and ACE-inhibitors or angiotensin receptor blockers (ACEI/ARB) in tolerable dosages as “Class I (“should use”) recommendations [6]. Usually, ACEI are started first followed by BB when there is hemodynamic stability; however, reverse order has been used and is also effective. The combination of ACEI + BB seems to have a synergistic effect that is beneficial.

We do not yet know if the new dual angiotensin receptor blocker (ARB) and nepriysin inhibitor (ARNI) will be more effective than an ACEI, but it does show promise of benefit for some [9]. We do know that newer intervention trials are needed to help those who currently are the most resistant to full recovery; namely, those who at diagnosis have LVEF < 0.30 and left ventricular end-diastolic diameter (LVEDD) ≥ 6 cm [2].

Thus far, inhibition of the lactating hormone, prolactin, with the use of bromocriptine has neutral or disappointing results; and continuation of breastfeeding has not been shown to be detrimental to recovery [2,10,11]. More work needs to be done on the prolactin theory of causation to be sure that findings on the mouse model can indeed translate to the human model, in which there may be more resistance to cleavage of normal prolactin into a cardiotoxic metabolite (genetically determined?) [11-13].

First priority is to initiate the recovery phase. The issue of safety for subsequent pregnancies can be considered later

It is helpful to indicate to the new mother that the safety of future pregnancies depends upon achieving full recovery of heart function. We now know that most women who experience full recovery are indeed able to safely have a subsequent pregnancy [8,14,15]. We are still learning about the risks for relapse of heart failure in subsequent pregnancies. This type of relapse is still a possibility in some of those

Correspondence to: James D. Fett, MD, Peripartum Cardiomyopathy Projects, 2331 Mt. Hood Ct. SE, Lacey, WA 98503, USA; Co-Director and Steering Committee, Peripartum Cardiomyopathy Network (PCN), IPAC Investigations in Pregnancy-Associated Cardiomyopathy, (Principal Investigator and Co-Director, Dennis McNamara, MD), Tel: 360-438-5270; E-mail: fett.sprunger@comcast.net

Received: November 06, 2015; Accepted: December 07, 2015; Published: December 10, 2015
who appear to have regained normal function. Nevertheless, it is also reassuring to know that even when relapse occurs in these apparently recovered women there is an effective treatment that still favors good outcomes. Part of the reason for this is that close observation leads to early recognition, early institution of effective treatment and thus stabilization with subsequent good response.

A new era

This is a new era for PPCM. We are poised to make much more progress. Working together as a team we are now in a much better position to help every PPCM mother fully recover.

References

1. Fett JD (2013) Earlier detection can help avoid many serious complications of peripartum cardiomyopathy. Future Cardiol 9: 809-816. [Crossref]
2. McNamara DM, Elkayam U, Alharehi R, Damp J, Hsich E, et al. (2015) 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. [Crossref]
3. James D Fett (2014) Peripartum cardiomyopathy: A puzzle closer to solution. World J Cardiol 6: 87-99. [Crossref]
4. Elkayam U (2011) Clinical characteristics of peripartum cardiomyopathy in the United States: diagnosis, prognosis, and management. J Am Coll Cardiol 58: 659-670. [Crossref]
5. Goland S, Modi K, Bitar F, Janmohamed M, Mirocha JM, et al. (2009) Clinical profile and predictors of complications in peripartum cardiomyopathy. J Card Fail 15: 645-650. [Crossref]
6. American Heart Association (2009)The AHA Guidelines and Scientific Statements Handbook. Fuster V (Ed.). Wiley-Blackwell, Oxford, UK.
7. Fett JD (2011) Validation of a self-test for early diagnosis of heart failure in peripartum cardiomyopathy. Crit Pathw Cardiol 10: 44-45. [Crossref]
8. Fett JD, Shah TP, McNamara DM (2015) Why do some recovered peripartum cardiomyopathy mothers experience heart failure with a subsequent pregnancy? Curr Treat Options Cardiovasc Med 17: 354. [Crossref]
9. Sabe MA, Jacob MS, Taylor DO (2015) A new class of drugs for systolic heart failure: The PARADIGM-HF study. Cleve Clin J Med 82: 693-701. [Crossref]
10. Saffrinstein JG, Ro AS, Grandhi S, Wang L, Fett JD, et al. (2012) Predictors of left ventricular recovery in a cohort of peripartum cardiomyopathy patients recruited via the internet. Int J Cardiol 154: 27-31. [Crossref]
11. Haghikia A, Podewski E, Libhaber E, Labidi S, Fischer D, et al. (2013) Phenotyping and outcome on contemporary management in a German cohort of patients with peripartum cardiomyopathy. Basic Res Cardiol 108: 366. [Crossref]
12. Patten IS, Rana S, Shahul S, Rowe GC, Jang C, et al. (2012) Cardiac angiogenic imbalance leads to peripartum cardiomyopathy. Nature 485: 333-338. [Crossref]
13. Piwnica D, Touraine P, Struman I, Tabruyn S, Bolbach G, et al. (2004) Cathepsin D processes human prolactin into multiple 16K-like N-terminal fragments: study of their antiangiogenic properties and physiological relevance. Mol Endocrinol 18: 2522-2542. [Crossref]
14. Fett JD, Fristoe KL, Welsh SN (2010) Risk of heart failure relapse in subsequent pregnancy among peripartum cardiomyopathy mothers. Int J Gynaecol Obstet 109: 34-36. [Crossref]
15. Elkayam U, Tummala PP, Rao K, Akhter MW, Karaa1p IS, et al. (2001) Maternal and fetal outcomes of subsequent pregnancies in women with peripartum cardiomyopathy. N Engl J Med 344: 1567-1571. [Crossref]