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No Mother Should Die of Peripartum Cardiomyopathy

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a unique form of heart failure characterized by systolic dysfunction developing during or shortly after pregnancy in a subject with a previously healthy heart; and is one of the leading causes of maternal mortality world-wide. Ventricular tachyarrhythmias with sudden cardiac arrest (SCA) and sudden cardiac death (SCD) do occur in peripartum cardiomyopathy subjects; but the occurrence is infrequent and most often only where there is severe systolic dysfunction with LVEF less than 0.35. Earlier diagnosis, with better-preserved LVEF, is the best way to prevent either SCA or SCD. Those with LVEF less than 0.35 can be protected by continuous cardiac rhythm monitoring with a wearable cardiac defibrillator (WCD) in the early stages; or with implantable cardiac defibrillator (ICD) if the LVEF does not rise above the level of 0.35 after 5 to 6 months. "Evidence-based" treatment utilizing beta-blockers (BB) and angiotensin converting enzyme inhibitors (ACEI) or angiotensin releasing hormone blockers (ARB) also increases the threshold, helping to prevent these malignant arrhythmias from occurring. Controlled studies are still needed to compare use vs. non-use of prolactin inhibition treatment. At this time, we are on the cusp of recognizing the mothers who are at greatest risk for the development of PPCM as well as those who, although apparently fully recovered, are still at greatest risk for the development of heart failure in a post-PPCM pregnancy. "State-of-the-art" knowledge about PPCM and its diagnosis/management obligates us all as medical caregivers, as well as all subjects in pregnancy, to make sure that any loss of life due to PPCM becomes rare or non-existent. No mother should die of PPCM. I foresee the day when that will become fact, as well as full recovery for almost all.

BRIEF CASE SUMMARY

24 year old gravida 2, para 2. First pregnancy uncomplicated, vaginal birth, healthy male newborn. Current pregnancy received regular prenatal care and experienced increasing discomfort during the last month of pregnancy. Attempted pitocin drip induction of labor at 40 weeks gestation intermittently over a 24 h period. Persistent tachycardia observed during pitocin infusion. Cardiology consultation requested, subsequent echocardiography revealed severe dilated cardiomyopathy, left ventricular ejection fraction (LVEF) 0.35, left ventricular internal diameter diastole (LViDd) 7.l7 cm. Treatment initiated for heart failure with immediate Cesarean section under epidural anesthesia on 8 December, healthy female newborn. Experienced some improvement, with brief discharge from hospital, but required readmission in NYHA Class III heart failure. Repeat echocardiography on 9 December, LVEF 0.30; and again on 9 January, LVEF 0.15. Despite standard heart failure therapy, deterioration continued with development of cardiogenic shock and acute renal failure, died on 13 January.

COMMENTARY

Peripartum cardiomyopathy (PPCM) is a unique form of heart failure characterized by systolic dysfunction developing during or shortly after pregnancy in a subject with a previously healthy heart; and is one of the leading causes of maternal mortality world-wide [1,2]. Over the past two decades, maternal mortality rates have diminished from over 50% to less than 5%, with variation world-wide. With appropriate timely diagnosis and treatment, no mother should die of PPCM.

In the USA, the incidence of PPCM in mothers of African heritage is double that of those who do not have African heritage [3]. The reasons for this are not yet clear, but genetic factors, higher incidence of hypertension in

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pregnancy and later post-partum recognition of PPCM appear to play a role **(Figure 1)**.

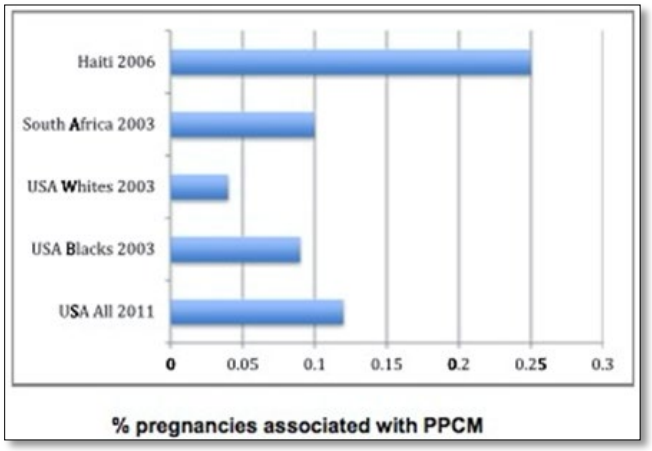

Figure 1. Comparative incidence of PPCM.

Ventricular tachyarrhythmias with sudden cardiac arrest (SCA) and sudden cardiac death (SCD) do occur in peripartum cardiomyopathy subjects; but the occurrence is infrequent and most often only where there is severe systolic dysfunction with LVEF less than 0.35 [4]. Earlier diagnosis, with better-preserved LVEF, is the best way to prevent either SCA or SCD. Those with LVEF less than 0.35 can be protected by continuous cardiac rhythm monitoring with a wearable cardiac defibrillator (WCD) in the early stages; or with implantable cardiac defibrillator (ICD) if the LVEF does not rise above the level of 0.35 after 5 to 6 months [1,5].

"Evidence-based" treatment utilizing beta-blockers (BB) and angiotensin converting enzyme inhibitors (ACEI) or angiotensin releasing hormone blockers (ARB) also increases the threshold, helping to prevent these malignant arrhythmias from occurring. Guidelines are provided in the American Heart Association/American College of Cardiology publication recommendations for the treatment of heart failure with reduced LVEF [6,7]. If the diagnosis of PPCM is made antenatal, it is safe to use hydralazine in place of ACEI/ARB. The use of hydralazine can be accompanied by the use of nitrates, depending upon possible heart rate increase from the hydralazine. BB treatment is safe to use during breastfeeding; but of the ACEI group, only enalopril is safe to use while breastfeeding, in which case ARBs should also be avoided. Likewise, the newer Entresto (valsartan/sacubitril) is not deemed safe to use while breastfeeding. "Controlled studies are still needed to compare use vs non-use of prolactin inhibition treatment.

Recent reports from the Peripartum Cardiomyopathy Network of North America, through the Investigations of Pregnancy-Associated Cardiomyopathy (IPAC) study, have clearly confirmed that adverse events, including chronic heart failure, following a diagnosis of PPCM, correlate with diagnosis not made until there is already severe systolic dysfunction, LVEF<0.30-0.35 [1].

Increasing awareness of PPCM enables earlier recognition, with a diagnosis made closer to the time of delivery, even antenatal. It appears that this earlier diagnosis is associated with better-preserved cardiac function, in which case there is better response to the effective treatment available, with fewer adverse events and fuller recoveries.

What other adverse events threaten the life of the new mother with PPCM? The most serious are major thromboembolic phenomena and severe heart failure transitioning to chronic cardiomyopathy requiring eventual cardiac transplantation; an option not available to many PPCM subjects in the "developing world" [8]. These adverse events can also be avoided if diagnosis can be made with relatively intact systolic function, i.e., LVEF>0.35 at diagnosis [8,9].

At this time, we are on the cusp of recognizing the mothers who are at greatest risk for the development of PPCM as well as those who, although apparently fully recovered, are still at greatest risk for the development of heart failure in a post-PPCM pregnancy [10]. This recognition will come through continuing development of a "genetic risk profile" [11,12], a "hypertensive risk profile" [1] an "angiogenic risk profile" and a "contractile reserve risk profile" **(Figure 2)** [13].

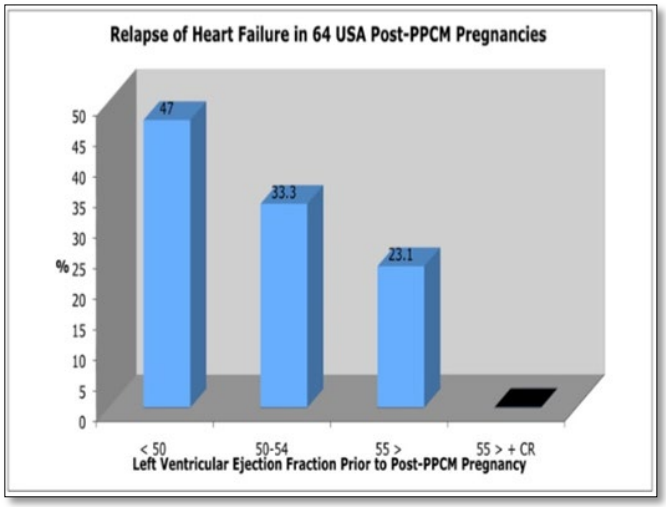

Figure 2. Risk for relapse of heart failure in a post-PPCM pregnancy [14,15].
CR: Contractile Reserve on Exercise Stress Echo

"State-of-the-art" knowledge about PPCM and its diagnosis/management obligates us all as medical caregivers, as well as all subjects in pregnancy, to make sure that any loss of life due to PPCM becomes rare or non-existent. No mother should die of PPCM. I foresee the day when that will become fact, as well as full recovery for almost all.

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