CLINICAL VIGNETTE

Cardiac Arrhythmia Complicating Postpartum Cardiomyopathy

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Introduction

Peripartum cardiomyopathy is usually defined as the development of heart failure with reduced ejection fraction at any point in the month prior to delivery date up to 5 months postpartum, without other identifiable causes of heart failure. It is a relatively unknown disease with a lot of the current data derived from small, nonrandomized studies. Readmission rates for patients with peripartum cardiomyopathy and dilated cardiomyopathy are very similar, and about half of those admissions are due to cardiac causes, and about 15% of them are due to cardiac arrhythmias.[1] Prognosis varies by geographical region with most reports indicating lower mortality rates in the US.[2] We present a case of a woman with peripartum cardiomyopathy who developed polymorphic VT/VF.

Case Report

A 45-year-old Caucasian female presents with a history of postpartum cardiomyopathy two months after her third pregnancy. She was 40 years old and her left ventricular ejection fraction decreased from normal to 15%. She was started on goal-directed medical therapy, including carvedilol 37.5mg twice daily and losartan 50mg daily, and after one year her left ventricular function improved to 50%, with residual mild mitral and aortic regurgitation.

Subsequently she had four episodes of syncope due to ventricular tachycardia (VT) as well as cardiac arrest which ultimately led to ICD implantation. Three of the four syncopal episodes occurred during intercourse. She was also started on Amiodarone but 18 months requested discontinuation due to concerns of long term toxicities. She had no recurrence of VT while she was on Amiodarone.

Six months after stopping Amiodarone she again reported episodes of presyncope and syncope during intercourse. Device interrogation revealed polymorphic ventricular tachycardia and ventricular fibrillation. She did not have any episodes with exercise. In total she had 6 episodes of PMVT/VF within one year, and 3 of them resulting in ICD shock therapy. Her cardiologist increased her carvedilol to 50mg BID and she was referred for Electrophysiology evaluation.

She was offered sympathetic denervation versus starting dofetilide for treatment. The patient declined both options and is currently continuing with goal-directed medical therapy for heart failure.

Discussion

Cardiomyopathy in the peripartum period was first described in 1937.[2] The term peripartum cardiomyopathy was not used until 1971 after Demakis et al[3] published a series of 27 patients and officially termed their clinical presentation as "peripartum cardiomyopathy." PPCM is a diagnosis of exclusion and very little is known about the etiology of the disease. Although many potential theories have been explored, none have ever been proven. Several risk factors have been cited in the literature. These include advanced maternal age (older than age 30), black race, multiparity, multiple gestations, obesity, smoking, chronic hypertension, cocaine abuse, low socioeconomic status, preeclampsia and eclampsia to name a few.[2]

Patients are treated with goal directed medical therapy for heart failure, with bromocriptine emerging as a promising adjunct therapy during the acute phase of PPCM.[4] It is important to take into account possible adverse effects to the fetus in patients who develop PPCM during the antepartum period. About half of the patients recover normal left ventricular function, while the remaining half has varying recovery and mortality rates.[1,5] Determining when to stop medical therapy is controversial. There are no clear guidelines regarding duration of therapy, although studies suggest tapering medications gradually, with one study suggesting to continue ACE-inhibitors and beta-blockers for at least 2 years after complete recovery.[2,6]

Complications of PPCM include cardiogenic shock, cardiopulmonary arrest, arrhythmias and thromboembolism. These patients are at particularly higher risk for developing thromboembolism compared with patients with dilated cardiomyopathy. One study reporting an LV thrombus in 17% of women at the time of diagnosis.[7]

There is limited data regarding the prevalence of ventricular arrhythmias in PPCM. In general patients with severely reduced left ventricular systolic function are considered high risk for developing life-threatening arrhythmias, however one study seemed to suggest that the risk is actually lower in patients with PPCM. Saltzberg et al[8] did a retrospective analysis of 107 patients with PPCM versus 159 matched nonpregnant patients with nonischemic DCM (NIDMC). All patients wore wearable cardioverter/defibrillators (WCD) and they compared the incidence of ventricular arrhythmias in these groups. They found that no shocks were delivered to the patients with PPCM versus 2 shocks in the NIDMC patients. They also found a significantly lower mortality rate in the patients with PPCM (2.8%)

versus those with NIDCM (6.9%). In contrast, the mortality risk in patients with PPCM has been noted to be higher in other studies, with rates as low as 7.1% in some studies and as high as 15% in one study.[9-12]

Another study found that the incidence of ventricular arrhythmias is higher in PPCM. Duncker et al[13] followed 12 women with PPCM and found 3 women had 4 ventricular defibrillation (VF) events over a 2.5 year period, all were successfully shocked by the WCD. One women had VF despite her EF improving to 45%. Although this study has its limitations due to small sample size, the authors concluded that PPCM carries a high risk of ventricular arrhythmias, especially in the early stages of the disease. They also noted that a low EF may not be an indicator of the risk of developing ventricular arrhythmias in these patients.

There is still a lot we do not know about PPCM, and more specifically about the incidence and the risk of arrhythmias in these patients. The use of WCDs can be useful in patients with PPCM and can help guide management.

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