Arrhythmias in Peripartum Cardiomyopathy

Michael C. Honigberg, MD, MPP[a],
Michael M. Givertz, MD[b,*]

KEYWORDS

• Peripartum cardiomyopathy • Pregnancy • Maternal-fetal health • Sudden cardiac death
• Antiarrhythmic medications • Implantable cardioverter-defibrillator • Arrhythmias

KEY POINTS

- Peripartum cardiomyopathy (PPCM) is a complication of late pregnancy and the early postpartum period characterized by dilated cardiomyopathy and heart failure with reduced ejection fraction.
- Although the prevalence of specific arrhythmias in PPCM is unknown, an estimated 1 in 4 deaths in women with this condition is sudden and presumed secondary to ventricular tachyarrhythmia.
- Management of PPCM entails standard treatment of heart failure with reduced ejection fraction and prevention of sudden cardiac death (SCD) in patients at increased risk, with special considerations for women who are predelivery or breastfeeding.

INTRODUCTION

PPCM is a rare, dilated cardiomyopathy of unknown cause characterized by heart failure with reduced ejection fraction.[1] According to the US National Heart, Lung, and Blood Institute, PPCM is diagnosed when a woman develops heart failure in the last month of pregnancy or up to 5 months postpartum, with a left ventricular (LV) ejection fraction less than 45% and no other identifiable cause of heart failure.[2] Alternative criteria allowing for earlier and later diagnosis have also been proposed.[3] Pregnancy-associated heart failure is a term used to describe women who develop signs and symptoms of heart failure at any time during pregnancy.[4]

Clinical Presentation

In the United States, a majority of women with PPCM are diagnosed in the early postpartum period (**Fig. 1**).[5,6] Patients typically present with heart failure symptoms, although rarely they may present with symptomatic or even unstable arrhythmias.[1,7,8] More common electrocardiographic findings include nonspecific ST-T wave changes, occasionally with conduction abnormalities, such as bundle branch block. Echocardiography shows varying degrees of depressed ventricular systolic function and ventricular dilatation, often with biatrial enlargement as a consequence of valvular regurgitation.[5] Chest radiography may show cardiomegaly, pulmonary edema, and/or pleural effusions. Natriuretic peptide levels (eg, B-type natriuretic peptide (BNP) and N-terminal pro-BNP) may be markedly elevated, in contrast with normal pregnancies.[9] Low-level troponin elevations also may be seen and predict adverse remodeling.[4]

Epidemiology

Well-established risk factors for PPCM include African American race,[10] older maternal age (with increased risk above age 30), chronic hypertension,

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[a] Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA 02115, USA; [b] Cardiovascular Division, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA 02115, USA
* Corresponding author.
E-mail address: mgivertz@partners.org

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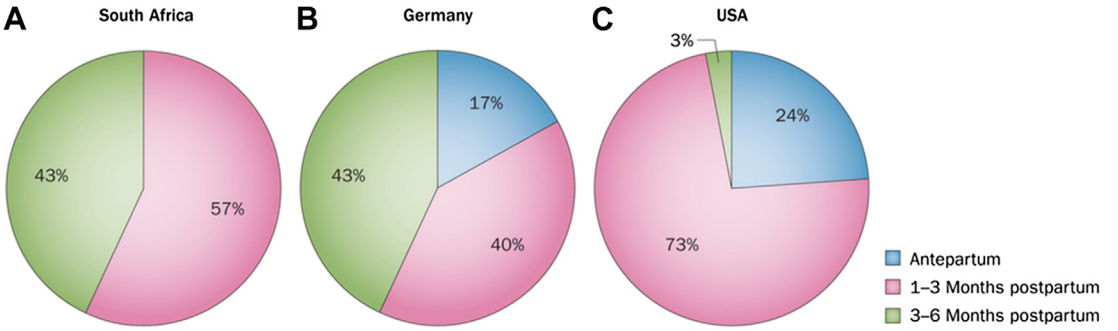

Fig. 1. Timing of presentation in patients with PPCM in (*A*) South Africa, (*B*) Germany, and (*C*) USA. (*From* Hilfiker-Kleiner D, Sliwa K. Pathophysiology and epidemiology of peripartum cardiomyopathy. Nat Rev Cardiol 2014;11(6):365; with permission.)

pregnancy-associated hypertensive conditions (eg, preeclampsia and eclampsia), and multifetal gestation as well as prolonged use of tocolytics in labor.[11–13] Hispanic women seem to have the lowest incidence.[12]

The incidence of PPCM in the United States seems to be increasing (Fig. 2),[14] with an average rate of 1 in 968 live births between 2004 and 2011.[15] During this period, incidence rose from 8.5 to 11.8 per 10,000 live births.[15] Proposed explanations for this trend include climbing rates of advanced maternal age and preeclampsia (secondary to increasing rates of obesity, diabetes, and chronic hypertension), more multifetal gestations due to increased use of assisted reproductive technologies, and growing recognition of PPCM as a disease entity.[5,6,15]

Approach to Management

Standard medical therapies for heart failure are used to manage PPCM, with some exceptions for women who have not yet delivered. For example, diuresis in pregnant women should be undertaken cautiously to avoid maternal hypotension and uterine hypoperfusion. With respect to afterload reduction, angiotensin-converting enzyme inhibitors and angiotensin-receptor blockers are teratogenic and are contraindicated in pregnancy. The combination of hydralazine and isosorbide dinitrate may be used instead. β_1-Selective blockers are preferred to nonselective β-blockers due to risk of uterine stimulation via β_2-sympathetic innervation. For pregnant women with severely depressed LV ejection fraction at high risk of thrombus formation, unfractionated heparin and low-molecular-weight heparin are preferred, because warfarin crosses the placenta and may cause fetal hemorrhage.[5]

For women with acute decompensated heart failure refractory to medical management, mechanical support with an intra-aortic balloon pump, ventricular assist device, or extracorporeal membrane oxygenation may be necessary as a bridge to recovery or transplant.[16]

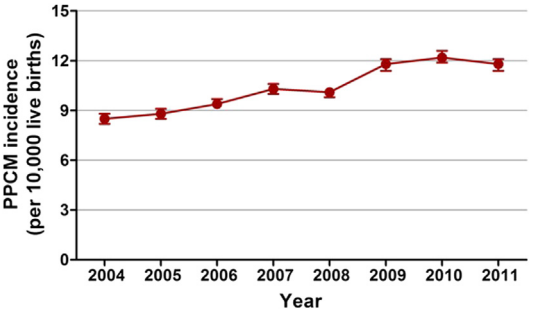

Fig. 2. Temporal trends in incidence rate of PPCM in the United States. (*From* Kolte D, Khera S, Aronow WS, et al. Temporal trends in incidence and outcomes of peripartum cardiomyopathy in the United States: a nationwide population-based study. J Am Heart Assoc 2014;3(3):e001056; with permission.)

Prognosis

Recovery of LV function occurs in approximately half of women, although there is considerable variation reported across studies likely due to selection bias. African American race seems to be associated with decreased likelihood of LV recovery.[17] A wide range of mortality rates, as high as 20%, has been reported in US series, with most mortality occurring within 6 months of diagnosis.[5] A recently published study reported that in-hospital mortality secondary to PPCM in the United States was 1.3%.[15] Other major adverse events associated with PPCM are the need for mechanical circulatory support (1.5%), heart transplant (0.5%), and cardiac arrest (2.1%).[15] Among women requiring mechanical circulatory support for PPCM, approximately half ultimately require cardiac transplantation; 2-year survival in this group is reported to be 83%.[16]