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EMERGENCY DEPARTMENT EVALUATION AND MANAGEMENT OF PERIPARTUM CARDIOMYOPATHY

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□ **Abstract—Peripartum cardiomyopathy (PPCM) affects 1000–1300 women in the United States each year. We present three cases of PPCM seen in our Emergency Department (ED) that cover the entire spectrum of disease from mild heart failure to sudden cardiac death. Without previous heart disease, these women develop cardiomyopathy with impairment of left ventricular function in the last month of pregnancy, or during the first 5 months postpartum. The etiology of PPCM is not clear, although various mechanisms have been proposed, including infection, autoimmune response, prolonged tocolysis during labor, and maladaptive responses to the hemodynamic changes of pregnancy. The initial presentation of these patients is frequently to the ED. The differential diagnosis and key characteristics of PPCM are discussed. ED management should focus on three elements: reduction in pre-load, reduction in afterload, and increase in inotropy. Key differences between the antepartum and postpartum states are highlighted. © 2009 Elsevier Inc.**

□ **Keywords—cardiomyopathy; peripartum; heart failure; pregnancy; postpartum**

INTRODUCTION

Postpartum or peripartum cardiomyopathy (PPCM) is a clinical entity whose etiology remains unclear. PPCM is associated with 1 of every 3000–4000 live births in the United States (US), thereby affecting 1000–1300 women annually (1). The prognosis of women who develop PPCM varies, with an alarmingly high mortality rate of 20–50% (2,3). In those who survive, 50% will have improvement of left ventricular (LV) function, and of these women, 10% to more than 50% will have complete recovery (3–6). Women with PPCM may present to the Emergency Department (ED) for initial evaluation and management of their acute symptomatology. A review of the emergency medicine literature noted few reports on the evaluation and management of this disease entity within the past two decades (7–10). Here, three cases of women presenting to the ED with PPCM are discussed, followed by the diagnosis and management of this potentially life-threatening disease.

CASE REPORTS

Case 1

A 23-year-old, previously healthy woman, gravida 3 para 2 (2 full-term live births, 1 medical abortion), 28 days postpartum, presented to the ED with the initial complaint of a headache. The headache was frontal, throbbing, and consistent with similar headaches in the past. On further review of systems, she reported intermittent episodes of dyspnea. The dyspnea began late in the third

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trimester, and was thought to be within the spectrum of normal third trimester dyspnea. However, over the 2 days before ED evaluation, she developed increased orthopnea, new paroxysmal nocturnal dyspnea (PND), and worsening dyspnea on exertion. She denied any chest, left arm, neck, or jaw pain. She had not noted any peripheral edema or swelling. She did not have any hemoptysis.

The prenatal history was notable for normal blood pressures and no proteinuria. She had gone into labor at term, and had had an uncomplicated spontaneous vaginal delivery without tocolytics. Family and social history were non-contributory.

On ED presentation, the temperature was 36°C (96.4°F), pulse was 119 beats/min, blood pressure was 148/91 mm Hg, respiratory rate was 18 breaths/min, and oxygen saturation was 96% on room air. The physical examination was significant for bilateral diffuse rales and an S4 gallop. There was no jugular venous distention or peripheral or periorbital edema. The neurological examination was normal. A chest radiograph revealed cardiomegaly and bibasilar consolidations consistent with pulmonary edema (Figure 1). Table 1 lists pertinent laboratory studies, all of which were within normal limits, including cardiac enzymes, liver function tests, and uric acid. The electrocardiogram (ECG) demonstrated sinus tachycardia and left atrial enlargement with lateral T wave inversions (Figure 2). In consultation with the cardiology service, the patient was admitted to the ED observation unit, placed on a cardiac monitor with supplemental oxygen, and given intravenous furosemide.

Table 1. Relevant Laboratory Studies

Laboratory Test	Patient #1	Patient #2	Patient #3
Hematocrit (36–48%)	44.4	33.7	38.7
Platelets (K/μL)	371	291	203
Creatinine (0.7–1.3 mg/dL)	0.7	0.5	1.1
AST (9–30 U/L)	17	68	314
ALT (7–52 U/L)	12	47	188
Alk Phos (36–118 U/L)	110	174	88
Uric Acid (1.8–6.7)	5.4	*	*
CK (27–218 U/L)	100	213	147
CK-MB (0–5 ng/mL)	ND	1.7	4
Troponin-I (< 0.1 ng/mL)	0.04	0.16	< assay
BNP (< 100 pg/mL)	†	894	*
Urine protein (0–15 mg/dL)	19	NP	*

* Test not performed.
† Test not available to be performed at time of patient presentation.
Normal values are included in parentheses.
ND = not done (laboratory does not run if total CK is low normal); NP = test not performed because urine dip was negative for protein.

The following day, an echocardiogram revealed an ejection fraction (EF) of 40–45%. The patient was then admitted to the cardiology service and diuresed with increasing doses of furosemide, yet the urine output remained low. A right heart catheterization indicated she was intravascularly volume depleted. She was gently hydrated and started on digoxin and captopril to improve the cardiac output. Given her predisposition to clot formation in the setting of a low EF, she received enoxaparin, and was gradually shifted to warfarin. She was discharged from the hospital 1 week later on captopril, digoxin, warfarin, and metoprolol. On 6-month follow-up, the echocardiogram showed a recovered EF (now

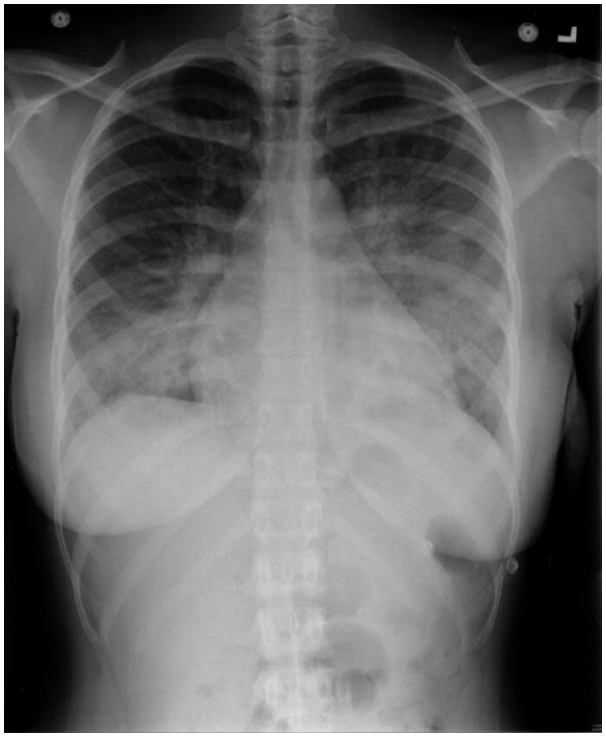

Figure 1. Chest X-ray study from Case 1 revealing cardiomegaly and pulmonary edema.

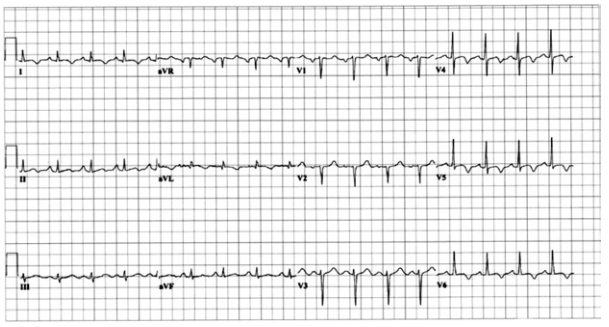

Figure 2. Electrocardiogram from Case 1 demonstrating sinus tachycardia, left atrial enlargement, and lateral T-wave inversions.