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A Case of Peripartum Cardiomyopathy

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Abstract **Case Report**

Peripartum cardiomyopathy is characterized by left ventricular systolic dysfunction during the last month of pregnancy through five months after delivery. Our case involves a 22-year-old female who was diagnosed with peripartum cardiomyopathy a few days after giving birth. She received medication management and had an external defibrillator placed. She was advised to have close cardiology follow up as an outpatient.

Keywords: peripartum cardiomyopathy.

INTRODUCTION

Peripartum cardiomyopathy can be seen in 1:1,000 to 1:4,000 cases in the United States [1]. The condition is characterized by left ventricular systolic dysfunction in female individuals during the last month of pregnancy and up to five months postpartum [1]. There is a higher incidence of the condition in female individuals who are of African heritage, aged thirty or greater, have hypertension, are multiparous, or receive tocolytic medications [2]. While a definitive understanding of peripartum cardiomyopathy has not been determined, there are theories as to the etiology [2]. Some possible causes include genetics, hormonal changes, activation of cytokines, pathologic responses to changes in hemodynamics during the pregnancy, and pathologic immune reactions to the pregnancy [2]. We describe a 22-year-old Caucasian female who developed peripartum cardiomyopathy.

CASE PRESENTATION

A 22-year-old G2P0010 Caucasian female with medical history notable for type 1 diabetes mellitus, gestational hypertension, gestational thrombocytopenia, and vape dependence presented to the hospital for induction of labor at 37.1 weeks gestation in the setting of pre-eclampsia without severe features. Her blood pressure was 152/99 mmHg, heart rate was 110 beats per minute, and temperature was 97.6 F. Her labs at presentation were notable for hemoglobin of 10.4 g/dL (reference: 12.4 – 15.2 g/dL), platelet count of 143,000 cells/µL (reference: 150,000 – 450,000 cells/µL), and urine protein/creatinine ratio of 3.10 (reference: < 0.15). She received induction of labor with oxytocin but elected for caesarean section after feeling fatigue while waiting for delivery. She underwent successful caesarean section on the day after presentation.

Two days after delivery, the patient remained tachycardic, with heart rate fluctuating between 120 and 140 beats per minute. Her electrocardiogram (ECG) was notable for sinus tachycardia. She had an echocardiogram, which revealed left ventricular ejection fraction (LVEF) of 25%, mild-moderately dilated right atrium, moderate pleural effusion in the left and right lateral regions, moderate mitral valve regurgitation, moderate tricuspid regurgitation, and moderate pulmonic valve regurgitation. This was a change from her echo from five months prior, which showed a LVEF of 55%. The cardiology team evaluated the patient and started her on spironolactone 25 mg daily, carvedilol 3.125 mg twice daily, and enalapril 5 mg daily. A sodium-glucose cotransporter 2 inhibitor was not started out of concerns that it could increase her risk of euglycemic diabetic ketoacidosis. The patient was fitted with an external defibrillator and was discharged with close outpatient follow up. She was going to bottle feed, rather than breast feed, her child. Two days later, the patient was attempting to lay flat in her bed, when she experienced significant shortness of breath, prompting her to come to our hospital for workup. During the time between

discharge and presentation to the hospital, the patient was complying with her newly started medications.

On presentation to our facility, her blood pressure was 111/96 mmHg, heart rate was 126 beats per minute, respiratory rate was 20 breaths per minute, and temperature was 98.0 ˚F. Her physical examination revealed decreased breath sounds at the bases and 2+ pitting edema in the lower extremities. Her ECG showed sinus tachycardia. Her laboratory results were notable for hemoglobin of 8.3 g/dL, platelet count of 296,000 cells/µL, B-type natriuretic peptide level of 9,497 pg/mL (reference: 0 – 125 pg/mL), and D-dimer level of 4.13 µg/mL (reference: 0.00 – 0.49 µg/mL). Due to elevated D-dimer, she underwent computed tomography pulmonary angiography which did not reveal pulmonary emboli. However, it did show bilateral pleural effusions, greater on the right side than the left side (Figure 1). The patient was evaluated by the cardiology team. She was started on intravenous furosemide dosed at 40 mg three times daily. Her spironolactone and carvedilol were continued. Her enalapril was discontinued so that she could be started on sacubitril-valsartan later in the hospital course. She underwent a repeat echocardiogram, which showed LVEF of 20%, grade 3 diastolic dysfunction, mildly reduced RV systolic function, mildly dilated right atrium, mildly dilated left atrium, large left lateral pleural effusion, moderate tricuspid regurgitation, and mild pulmonic valve regurgitation. Her carvedilol was subsequently changed to metoprolol succinate 100 mg daily. At the time of discharge, she was asymptomatic and euvolemic. Her medication regimen at discharge was sacubitril-valsartan 12-13 mg twice daily, metoprolol succinate 100 mg daily, spironolactone 25 mg daily, and furosemide 20 mg daily. She was advised to continue using her wearable defibrillator and have close outpatient follow up with cardiology. At the time of discharge, her laboratory results were notable for hemoglobin of 11.1 g/dL and platelet count of 422,000 cells/µL.

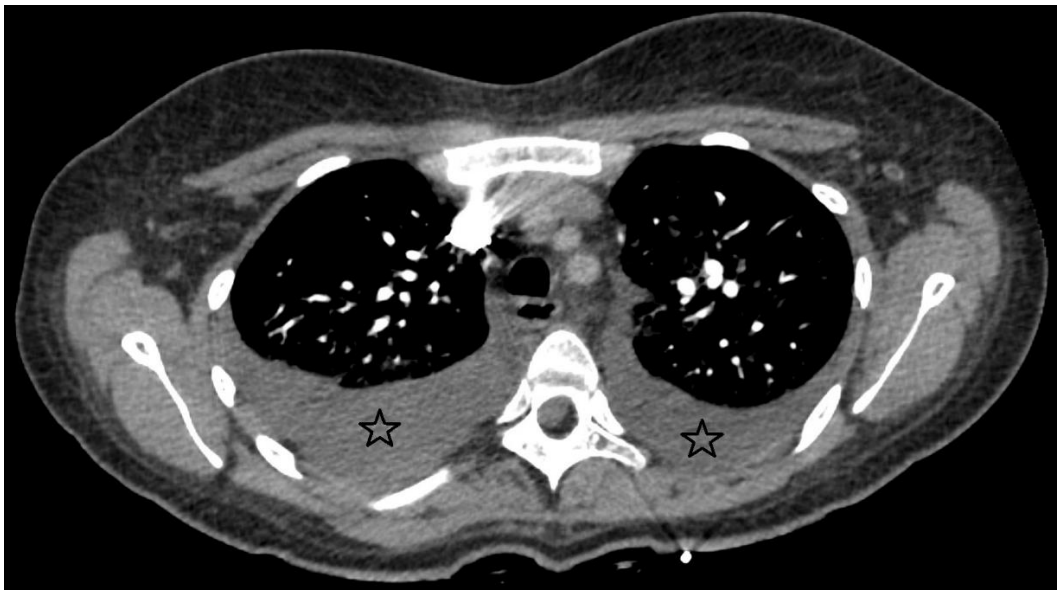

Figure 1: Computed Tomography of the Chest

Legend: The stars indicate the pleural effusions.

DISCUSSION

Management of peripartum cardiomyopathy is derived from the guideline-directed medical therapy (GDMT) used in cases of heart failure with reduced ejection fraction [3]. After delivery, usage of beta-blockers, aldosterone receptor antagonists, angiotensin-converting-enzyme inhibitors or angiotensin II receptor blockers, and sodium-glucose cotransporter 2 inhibitors are commonly used [3]. Agents such as sacubitril-valsartan can also be utilized [3]. To allow for reduction in afterload after delivery, hydralazine and isosorbide dinitrate can be used together [3]. Pursuing volume management with diuretics and nitrates can be pursued after delivery [3].

During pregnancy, beta blockers can be used [3]. The use of aldosterone receptor antagonists, angiotensin-converting-enzyme inhibitors, and angiotensin II receptor blockers is contraindicated during pregnancy [3]. Data are limited for the use of sodium-glucose cotransporter 2 inhibitors and agents such as sacubitril-valsartan, even after delivery [3].

Sometimes, more advanced interventions are required, including left ventricular assist devices, extracorporeal membrane oxygenation, and intra-aortic balloon pumps [3]. Defibrillators, especially wearable ones, can be provided for patients with peripartum cardiomyopathy due to the increased risk of ventricular arrhythmias [3].

Approximately 15% of cases of peripartum cardiomyopathy have a genetic component [4]. The most commonly involved gene is *TTN* [4]. Other genes include *VCL*, *BAG3*, *FLNC*, and *DSP* [4].

In many individuals, the LVEF will increase to more than 50% by six months from the time of diagnosis [3]. Some individuals will need longer than six months for recovery, while others may not reach normal LVEF at all [3]. The mortality rate associated with the condition ranges from 7 to 15% worldwide [5].

CONCLUSION

Management of peripartum cardiomyopathy is similar to that of heart failure with reduce ejection fraction. Many individuals will need medication management, although some will need more advanced interventions. Defibrillators can have an important role as the condition is associated with increased risk of arrhythmias. Most, but not all, individuals will have an improvement in their LVEF within 6 months.

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