## CLINICAL VIGNETTE

# Dyspnea in a Postpartum Woman

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### *Case Report*

A 43-year-old Caucasian female with no prior cardiac history presented to cardiology for evaluation of dyspnea. She was 3 weeks post-partum following her first pregnancy, which had resulted in birth of twin boys by cesarean section. Since her delivery, she complained of worsening lower extremity edema and dyspnea. Her pregnancy was significant for 40 lbs weight gain, attributed to her twin gestation. Two days prior to her appointment, she was evaluated at an urgent care clinic for worsening dyspnea, and was noted to be in heart failure. A chest film revealed cardiomegaly with increased pulmonary vasculature. She was started on a low dose diuretic, with some improvement in dyspnea but persistent fatigue. On examination, her blood pressure was 150/70 mm Hg and pulse was tachycardic at 110 beats per minute. Her neck veins were elevated at 12 cms, and her apical impulse was displaced, in the sixth intercostal space at the anterior axillary line. Cardiac auscultation revealed the presence of a third heart sound and a grade 2/6 mid systolic murmur at the apex. She had crackles at both lungs bases. Abdomen was soft and non-tender and lower extremities were cool with 3 plus pitting edema to the knees.

ECG showed sinus tachycardia with T wave flattening in lateral leads. An echocardiogram performed on the day of evaluation revealed moderately increased left ventricular size with severely reduced ejection fraction of 25-30%. Moderate mitral and tricuspid valve regurgitations were seen. In addition, there was mild pulmonary hypertension. The patient was advised to increase her diuretic dosage, and low dose beta-blocker was initiated. At 2 weeks follow-up, the patient noted continued improvement with reduction of her edema and dyspnea. Her beta-blocker dose was increased. At 4 weeks, her echocardiogram did not show any significant improvement except reduction of her pulmonary artery pressures. At 3 months, she stopped nursing and was started on an angiotensin converting enzyme inhibitor. At 4 months follow-up, her ejection fraction had significantly improved to 45-50% with reduction of mitral and tricuspid valve regurgitations. Her diuretics were discontinued and she continued other agents. At 6 months follow-up, left ventricular function had normalized and she noted an improvement in her exercise capacity and endurance. She was advised to not conceive any further children.

### *Epidemiology*

Peripartum cardiomyopathy (PPCM) has been correlated with poverty and is very common in areas where both gravidity and parity are high, e.g. West Africa, Haiti. Notably, other risk factors such as age and African origin have been found with relative inconsistency. For example, 24-37% of cases occur in primigravid females, although heightened age has been shown to be just as relevant[1,2]. Furthermore, Elkayam et al. showed that PPCM is not limited to only African-American females[3]; however, Gentry et al. showed a 16-fold greater incidence in African -American females in comparison to non-African-American females.[4]

### *Presentation*

Patients usually present during the puerperium through five months postpartum. A typical presentation with PPCM is a patient with cardiac asthma, paroxysmal nocturnal dyspnea, chest pain, cough, lower-extremity edema, raised jugular venous pressure, murmurs consistent with mitral and tricuspid valve regurgitation, and pulmonary bruits.

### *Diagnosis*

The diagnosis of PPCM is challenging since many women in the last month of normal

pregnancy exhibit dyspnea, fatigue, and pedal edema. These symptoms are similar to those of early congestive heart failure. Because there is no current routine assessment of cardiac function in normal late pregnancy, it is important to keep a high index of suspicion for heart failure during normal to late delivery. There is also a correlation among PPCM patients and the presence of mural thrombi and cardioembolic stroke[5]. Family members should be screened as the disease maybe hereditary. PPCM is confirmed by standard echocardiographic assess-ment of left ventricular (LV) systolic dys-function, including depressed LV shortening fraction (<30%) or LV ejection fraction (<45%).[6]

### *Treatment*

Patients with PPCM require immediate attention. Oxygen should be administered until an arterial oxygen saturation of ≥95% is achieved. For conditions resulting in a fluid overload state, intravenous diuretics should be used with an initial bolus of furosemide 20-40 mg. In the presence of a thrombus, unfractionated heparin is recommended during pregnancy, and either warfarin or heparin postpartum. Inotropes may need to be considered in patients with low cardiac output state when there are signs of hypoperfusion.

Bromocriptine, a dopamine 2D agonist which blocks prolactin, has shown to be beneficial to patients with PPCM in experimental trials. Because of increased risk of thromboembolic stroke and myocardial infarction with this drug, anticoagulation therapy is highly recommended to those taking bromocriptine[6].

### *Prognosis*

Because of the similarity between the symptoms of PPCM and symptoms in normal late pregnancy, it is likely that the condition is under diagnosed. As a result, patients may not receive proper treatment for cardiomyopathy resulting in morbidity and mortality. Globally, affluent nations have a very low mortality rate for PPCM in comparison to less wealthy nations. Furthermore, mortality is higher in patients with late diagnoses. Risk is significantly higher in patients with persistent LV dysfunction before subsequent pregnancy[6]. Whitehead et al. reported that mortality increased with maternal age, in parity of >4, and women of African descent were 6.4 times more likely to die compared with whites[7]. Overall, LV systolic function tends to return to normal in the United States, Haiti, and Turkey with reported ranges of 23-41% of cases[8]. Continued studies in PPCM will likely decrease rates of mortality in both first- and third-world nations significantly.

### *Conclusion*

PPCM is a type of dilated cardiomyopathy of unknown origin, which occurs in previously healthy women in the final months of pregnancy and up to 5 months after delivery. Although the incidence is low, (less than 0.1% of pregnancies), morbidity and mortality rates vary and have been reported as high as 5% to 32%. The outcome of PPCM is also variable as some patients experience resolution of symptoms and normalization of cardiac function. On the other hand, the disease may progress to severe cardiac failure and even sudden cardiac death. In general, future pregnancies are discouraged in survivors of PPCM due to the increased risk of recurrence of heart failure and death in subsequent pregnancies.

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