



International Journal of Critical Care and Emergency Medicine

CASE REPORT

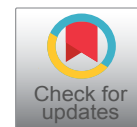
A Young Woman with Dyspnea Following Delivery- Cardiomyopathy, Pulmonary Embolism or Both?

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Abstract

Introduction: Postpartum cardiomyopathy is an important cause of heart failure in young women with an increasing rate in recent years. It may occur from the last month of pregnancy and up to five months after delivery. The etiology remains unclear and the disease has a high level of morbidity and mortality. Complications include CHF, arrhythmias, cardiogenic shock and thromboembolism. Treatment is similar to the treatment for CHF. Bromocriptine has shown encouraging preliminary results but is not standard therapy.

Case presentation: We present a 40 y/o healthy female that was referred to the ED due to shortness of breath. Upon admission she was stable respiratory and hemodynamically, she had tachycardia without fever. Her physical exam was normal except to rapid heart sounds, her abdomen had mild diffuse tenderness upon palpation and there was minimal edema on both ankles. ECG showed sinus tachycardia with an LBBB pattern. Chest X-ray and lab results were normal. Chest CT revealed a pulmonary embolism in the right lower lobe.

Echocardiogram revealed a mildly dilated left ventricle with mild to moderate systolic dysfunction. The left atrium was mildly dilated with higher pressure in the left atrium than right. Mitral valve leaflets were mildly thickened with moderate to severe regurgitation, tricuspid valve had mild regurgitation with moderately elevated pulmonary hypertension. BNP levels were high.

Discussion: In this case we report a healthy postpartum woman that presented to our ED with a combination of a LBBB pattern on her ECG, PE on spiral CT and suspected PPCM on echocardiogram.

Keywords

PPCM- Peripartum cardiomyopathy, PE- Pulmonary embolism, CHF- Congestive heart failure

Case Description

A 40 y/o healthy female was referred to the ED due to shortness of breath and a mild dry cough from the night before her admission. She denied having chest pain, palpitations or fever. She reported that 4 days prior to her admission she gave birth in a planned home vaginal birth. Her medical history was significant for an early spontaneous abortion with a negative hypercoagulability workup.

Upon admission her blood pressure was 131/87, her pulse was 120 and regular, oral temperature was 36.4 C and oxygen saturation was 97% on room air. Her physical exam showed no tachypnea or dyspnea, lungs were clear without rales or wheezing, heart sounds were rapid, regular and without murmurs or additional sounds, her abdomen was lax with mild diffuse tenderness upon palpation and there was minimal edema on both ankles. ECG showed sinus tachycardia with a left axis deviation and a wide QRS. Chest X-ray was normal- without effusion, infiltration or congestion. Lab results including SMAC, CBC were within normal limits and blood gases were also normal with a PH of 7.404, PCO₂ of 37 and a HCO₃ of 22. D dimer was not obtained due to the recent delivery. Because of a high suspicion for pulmonary embolism a spiral chest CT was ordered and revealed a segmental and subsegmental pulmonary embolism in the right lower lobe with a mild pleural effusion bilaterally. Treatment with a low molecular weight Heparin was started after the diagnosis. An echocardiogram showed a mildly dilated left ventricle (LVED 65 MM)



Citation: Gershinsky Y, Leibowitz D, Salameh S (2018) A Young Woman with Dyspnea Following Delivery- Cardiomyopathy, Pulmonary Embolism or Both?. Int J Crit Care Emerg Med 4:056. doi.org/10.23937/2474-3674/1510056

Accepted: November 19, 2018; **Published:** November 21, 2018

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with mild to moderate systolic dysfunction, right ventricle and atrium were normal, the left atrium was mildly dilated (LA 2D of 46 MM) with bulging of the inter-atrial septum towards the right suggesting left atrium pressures higher than right atrial pressures. The mitral valve leaflets were mildly thickened with moderate to severe regurgitation, the tricuspid valve had mild regurgitation with a peak systolic TR gradient of 44 mmHg suggesting moderately elevated pulmonary hypertension. An US duplex of the feet was performed with no evidence of DVT. High sensitive Troponin T was negative (0.027 ng/ml with normal range up to 0.3) while Pro BNP was elevated (732 PG/ML with normal range up to 115). The patient was then admitted to the internal ward and a short work up for hypercoagulable state was performed including anti-cardiolipin IgM and IgG, anti B2 glycoprotein IgG and IgM and Lupus anticoagulant with negative results.

After achieving an anti Xa level of 0.6 with LMWH overlapping treatment with Warfarin was started. In addition, after consulting with a cardiologist and on the basis of the echocardiographic findings she was diagnosed as suffering from left heart failure secondary to peripartum cardiomyopathy and began receiving treatment with an ACEI, Beta blockers and loop diuretics. After 5 days of hospitalization she was released home in good condition. A repeat echocardiogram after 5 weeks showed no change in LV size and function or in the severity of the MR, however the PHTN returned to normal range. Repeated ECG during this period showed NSR with a rate of 83 BPM and a persistent LBBB morphology.

In this case we report a 40 y/o healthy woman that presented to our ED with a combination of a LBBB pattern on her ECG, PE on spiral CT and suspected PPCM on echocardiogram.

Discussion

PPCM is an important cause of heart failure in young women with an incidence ranging between 0.2-3% of live births (about 1 to 1421-9861 live births) [1] depending on ethnicity with an increasing rate in recent years [2]. It may occur from the last month of pregnancy and up to five months after delivery. This condition withholds high percentage of morbidity and mortality [3] and its etiology is unclear.

PPCM has 4 diagnostic criteria- 1) Developing heart failure in the time period described above; 2) No other apparent reason for heart failure; 3) Lack of evidence of heart disease prior to the month before delivery; 4) An echocardiogram showing decreased LV systolic function with an ejection fraction of less than 45% or fractional shortening of 30% or both [4].

According to the European Society of Cardiology working group on PPCM, it is an idiopathic cardiomyopathy presenting with HF secondary to LV systolic dy-

sfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found [5].

The risk factors for PPCM are higher maternal age, African-American decent, personal history of HTN, multiparity and multi fetal pregnancies [6].

The clinical presentation of these patients includes exertion dyspnea, orthopnea and peripheral edema. As these symptoms are similar to those of normal end of pregnancy symptoms they are easily overlooked by non-experienced medical staff. The physical exam usually reveals tachypnea, tachycardia, elevated JVP, pulmonary rales and peripheral edema. ECG typically shows a sinus tachycardia with non-specific ST-T changes, LVH and LBBB might be noted. CXR usually shows pulmonary congestion with or without pulmonary effusion. Echocardiogram findings include of course LV systolic dysfunction and sometimes show ventricular dilatation, also common are mitral and tricuspidal regurgitations with elevated pulmonary pressure. Lab tests can be helpful-Troponin is elevated in some cases and has a prognostic importance [7], BNP (brain natriuretic peptide) is usually elevated [8].

The prognosis in PPCM for recovery of LV function is > 50% that occurs mostly within 2-6 months of diagnosis [9]. It is not clear if standard medical treatment for heart failure improves the rates of recovery [10].

Mortality rate is as high as 18% and the rate of heart transplantation is about 10% in the US [3]. Complications include CHF, arrhythmias, cardiogenic shock and thromboembolism. These women should be advised not to have a subsequent pregnancy because the high rates of unfavorable maternal or fetal outcomes [11].

Breast feeding in these mothers is advised as the evidence is that it is safe for the mothers and babies and even withholds a positive prognostic factor for LV recovery [12].

Treatment is based on the basic treatment for CHF. This includes ACEI/ARB (only in post-partum as they are contraindicated during pregnancy), diuretics and beta blockers in the stable patient. Intravenous vasodilators are restricted to the non-stable patient suffering from decompensated HF. Spironolactone, digoxin and anticoagulation can be added to the treatment. A drug showing encouraging although insufficient data is Bromocriptine and due to that is not widely recommended [13].

This case of PPCM we present is unique due to the diagnosis of a PE that accompanied it. Another reason it is unique is due to the non-rate dependent LBBB pattern on ECG that is not usually found in pulmonary embolism. We have found only three case reports previously documenting a combination of PE and PPCM [14-16]. In most cases these diagnoses come as differential diagnosis for a woman during late stages of pregnancy and early post-partum that presents with dyspnea to the ED [17,18].

There is uncertainty whether the PE was a complication of the PPCM or an independent diagnosis that isn't a result of the PPCM.

The concept of two different diagnoses is important as clinicians are trained to look for the one cause of the patient's clinical manifestation, usually it is based on the philosophic rule known as Occam's razor. This rule is probably right in most cases but not in all cases. If the clinician is unsatisfied with a diagnosis, he has reached because it does not explain the full clinical presentation of the patient, he should continue the workup and pursue other differential diagnosis. In our case the LBBB pattern on the ECG that isn't consistent with the diagnosis of pulmonary embolism raised the suspicion that there might be another diagnosis waiting to be revealed. This idea should be emphasized in the ED especially in patients that are in a life-threatening situation. We suggest that an echocardiogram be performed to all patients hospitalized with a new diagnosis of PE if it is possible. In cases that the differential diagnosis is unclear an echocardiogram should be mandatory as soon as possible.

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