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What's going wrong with this postpartum woman?☆

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a left ventricular systolic dysfunction failure emerges during the antepartum or puerperal period, and can result in maternal death. Reported incidences are increasing and differing globally. Echocardiography is the cornerstone for the diagnosis. The immediate goals in acute management are the stabilization of the hemodynamic state, providing symptomatic relief, and ensuring fetal wellbeing. Emergency physicians should be aware of PPCM at the differential diagnosis of dyspnea in pregnancy related emergencies and play role in early diagnosis.

1. Introduction

Peripartum cardiomyopathy (PPCM) is characterized by heart failure as a result of left ventricular systolic dysfunction that emerges during the antepartum or puerperal period, and can result in maternal death [1-3]. Varying with demographical, definitional, and diagnostic confusional determinants, reported incidences are increasing associated with diagnostic awareness and rising high-risk pregnancies differing globally as 1:968 births in the US and ever so less well documented outside of the US 1:1000 births in Africa and Asia with as high as 1:100 births in Northern Nigeria and 1:300 births in Haiti, likely due to the maternal factors including nourishment and comorbidities affecting PPCM [4-7]. Herein we report on a postpartum patient who presented to the emergency department (ED) with dyspnea, which should make a physician consider the possibility of PPCM.

2. Case

A 35-year-old Turkish white woman was admitted to our ED with an acute onset of dyspnea and squeezing chest pain that had started a couple of hours before presentation. She described having a similar occurrence of dyspnea but with less intensity 1 week earlier. Our patient had visited a pulmonologist and had been diagnosed with pneumonia and was prescribed amoxicillin clavulanate and salbutamol. Her chest X-ray showed no pathological changes during this visit. The woman was G1P1A0 and had a history of gestational hypertension and diabetes mellitus with an uncomplicated ceasarean section of a healthy infant 3 weeks earlier.

On admission, the patient's blood pressure was 180/110 mm Hg, with an oxygen saturation of 78 on pulse oxymetry on room air, a heart rate of 140 beats/min, a body temperature of 36.7 °C, and a respiratory rate of 22 breaths/min. Physical examination showed bilateral lower extremity edema existing for 2 weeks, bilateral zone 3 pulmonary rales, and rhythmic and tachycardic heart sounds on auscultation. She complained of orthopnea and palpitations. The electrocardiogram (ECG) was nonspecific with sinus tachycardia. The chest radiograph showed no specific findings (Fig. 1). Bedside echocardiography was performed by a cardiologist and left ventricular ejection fraction (EF) was found to be 20%. Diffuse left ventricular hypokinesia and left atrial enlargement were also the existing sonographic pathologies. Her laboratory findings were as follows; hemoglobin level: 9.8 g/dL (12–16 g/dL), hematocrit level: 31.8% (37%–47%), glucose level: 146 mg/dL, potassium level: 4.19 mEq/L and brain natriuretic peptide (BNP) value: 410 pg/mL (0–100 pg/mL). Other markers including troponin, d-dimer, TSH, renal function tests, and liver enzymes were within normal levels. Arterial blood gas analysis on oxygen supply showed a PaO2 of 99 mm Hg (83–108 mm Hg), a pH of 7.46 (7.38–7.46), a PaCO2 of 27.3 mm Hg (35–48 mm Hg), and HCO3 of 19,0 mmol/L (22.2–28.3 mmol/L).

In the ED, an 8 L/min of oxygen with nasal cannula, intravenous (IV) nitroglycerine at a rate 20 μg/min, and an IV bolus of 40 mg furosemide were initiated. The patient was admitted to the coronary intensive care unit (CICU) with the diagnosis of PPCMP and was given an additional anticoagulant and metoprolol therapy. The patient was discharged after 3 days with an EF of 35% and was prescribed an oral treatment of

☆ Evaluation of postpartum dyspnea in emergency department.

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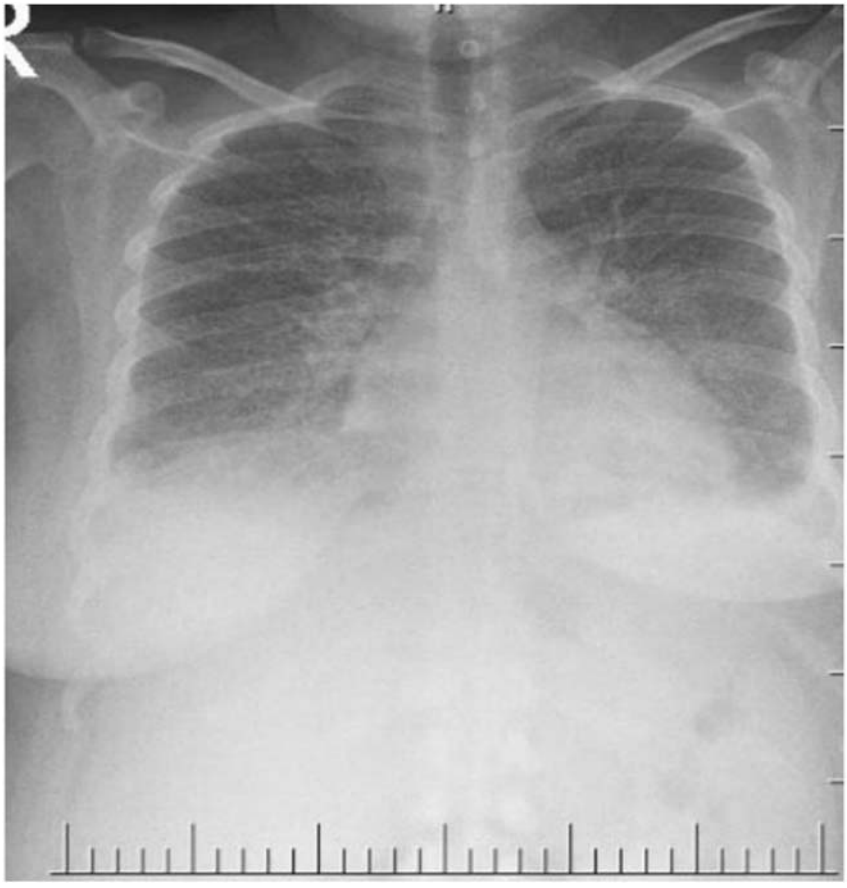

Fig. 1. The PA chest radiograph of patient on ED admisson.

metoprolol 100 mg/day, ivabradine 10 mg/day, perindopril 5 mg/day, spirinolactone 25 mg/day, and furosemide 40 mg/day. Twenty days after discharge, hypokinesia still remained, but the patient's EF was 55%.

3. Discussion

Cardiovascular disorders such as PPCM complicate 4% of all pregnancies, and can occur in women without a previously known medical problem and, without an unknown causation [1,2,8,9]. Development of HF in the last month of pregnancy or within 5 months after delivery, LV systolic dysfunction (LV EF < 45% by echocardiography), no identifiable cause of heart failure, and no recognized heart disease before the last month of pregnancy are the four required diagnostic criteria of PPCM [10]. Also the elimination of other causes of dyspnea and chest pain, such as acute myocardial infarction (AMI), pulmonary tromboembolism (PTE), pneumonia, thoracic aortic dissection, using several diagnostic tools including ECG, chest radiography, echocardiography, and brain natriuretic peptide (BNP) levels are necessary [9,11]. In our patient; because of the absence of ST-T segment abnormalities; negative troponin and d-dimer values; the absence of fever, cough, sputum; pathological pulmonary sounds on auscultation; and the character of the pain, we eliminated the differential diagnoses of AMI, PTE, pneumonia, and aortic dissection.

Symptoms of PPCM are mostly physical in character, occur as a result of systolic left ventricular dysfunction. Symptoms include dyspnea, orthopnea, paroxysmal nocturnal dyspnea, edema, fatigue, cough, feeling of impending death, panic, anxiety, chest pain, nausea/vomiting, bodily pain, decreased urine output, and fear. PPCM mostly emerges in the first week of the postpartum period but can present during the second or third trimesters of gestation or be delayed until the first 4 months of the puerperal period [7,11,12].

Echocardiography is the cornerstone for the diagnosis of PPCM. The main echocardiographic sign of PPCM is reduced left ventricular systolic function as indicated by a left ventricular ejection fraction of <45% and left ventricular hypokinesis. Global cardiac dilatation affecting all of the heart chambers is not necessarily present but is usually found. Mitral and tricuspid regurgitation and pulmonary hypertension are the other possible findings [9,13,14]. ECG is usually required for differential diagnosis and has nonspecific diagnostic findings such as Q-wave abnormalities, ST segment changes, T-wave inversion, conduction abnormalities (bundle branch blocks or 2°or 3°degree AV blocks), or other rhythm disturbances (premature atrial or ventricular beats, atrial fibrillation or flutter, or sinus tachycardia) [4,15]. Plain films may show pleural effusion and pulmonary congestion as a result of heart failure. The diagnosis of the disorder usually does not necessitate any other tests, but it may be facilitated by cardiac magnetic resonance imaging, cardiac biopsy, and heart catheterization. Additionally, most women with the condition have elevated BNP levels [9,16]. Our patient's echocardiogram showed a reduced EF (20%) and her BNP level was increased, thus suggesting a diagnosis of PPCM. There is no single diagnostic test for PPCM; suspicion of PPCM with the exclusion of alternative etiologies by the combination of medical history, physical examination, and appropriate diagnostic tests is the pathway to clinical diagnosis [13,17].

Since patients may complain of symptoms of term pregnancy that are almost identical to those of PPCM, a high index of suspicion should be maintained. Furthermore, as a timely and rapid diagnosis of PPCM may save lives and prevent morbidity, a 3-step bedside diagnostic screening tool can be used to distinguish the symptoms of term pregnancy from those of PPCM [18]. The first step of this tool uses signs and symptoms as the initial parameters (orthopnea, dyspnea, cough, edema, excess weight gain, and palpitations), where a score equal to or >4 increases the likelihood of PPCM and compels the physician to

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