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Peripartum Cardiomyopathy Complicated with Possible Pulmonary Embolism: A Case Report

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ABSTRACT

Peripartum cardiomyopathy associated with pulmonary embolism is a rare life threatening complication of pregnancy. The early symptoms of peripartum cardiomyopathy in heart failure and pulmonary embolism mimic those of the normal physiologic changes of pregnancy, making a diagnostic dilemma. Hence, it is important to understand the principles of assessment of these conditions which mimic physiological symptoms of pregnancy. Here, we share our experience of a 22 year old, primigravida at 38 weeks of gestation with no comorbidities presented in obstetric emergency with two days history of shortness of breath and productive cough who survived from critical event following the start of unfractionated heparin infusion and other supportive treatment.

Key words: Critical event, Peripartum cardiomyopathy, Pulmonary embolism, Unfractionated heparin.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) or pregnancy associated cardiomyopathy is defined as development of systolic heart failure (SHF) that occurs towards the last month of pregnancy and up to five months after delivery, with left ventricular ejection fraction (LVEF) less than 45% in the absence of other identifiable causes of HF (1). Patients with PPCM and LVEF < 35% are at risk for development of thromboembolism, which affects about 6.6% to 6.8% of women (2) due to hypercoagulable state of pregnancy and stasis of blood due to severe left ventricle (LV) stasis.

The incidence of PPCM is increasing may be due to improved diagnosis and recognition; and also due to increasing maternal age, preeclampsia, multiparity, multiple gestations and maternal cardiovascular risk. As the early signs and symptoms of HF in PPCM and pulmonary embolism (PE) may mimic the physiological changes that occur in pregnancy, there is a delay in diagnosis and management of the patient making the mortality rate high. Therefore, it is crucial to differentiate between physiological versus pathological signs and symptoms.

The aim of this case report is to familiarize a rare life threatening disease so that early diagnosis and prompt management is initiated to help prevent mortality.

CASE REPORT

A 22 year old, primigravida at 38 weeks of gestation was referred to the Obstetric emergency (ER) with suspected rheumatic heart disease in failure with complaint of shortness of breath and productive cough since two days.

On presentation, she was dyspneic with respiratory rate of 60/min, heart rate of 200/min, blood pressure of 110/80 mm of Hg and peripheral capillary oxygen saturation (SpO_2) of 40 – 50 % on high flow oxygen supplementation via simple face mask and bilateral crepitations were heard on chest auscultation with no added heart sound. Due to low SpO_2 and fall in Glasgow Coma Scale (GCS), she was intubated in ER.

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Immediately after intubation, she developed bradycardia followed by asystole. Cardiopulmonary resuscitation (CPR) was commenced immediately and was successful to achieve return of spontaneous circulation (ROSC) with SpO_2 of 90 %, heart rate (HR) of 180/min and blood pressure (BP) of 110/70 mm of Hg. Blood mixed frothy secretion was present in endotracheal tube which was suctioned and impression was made as pulmonary edema due to heart failure with pneumonia. Patient was then shifted in maternal intensive care unit (MICU) for further management on diuretics, antibiotics and ventilatory support.

In MICU, again her oxygen saturation started to fall to 30% with decrease in urine output inspite of treatment with diuretics, morphine, few bolus doses of nitroglycerine and ventilatory support. As patient was in critical condition, we made the assumption of pulmonary embolism based on 1) no clinical improvement with the medication we initiated; 2) the unremarkable finding we found on chest auscultation; 3) the ABG report which showed lactic acidosis with severe hypoxemia and low normal partial pressure of arterial carbon dioxide (Table 1); and 4) the vital signs that showed tachycardia, tachypnea, hypoxia with normal blood pressure. Based on high clinical suspicion of sign and symptoms closer to pulmonary embolism, we started intravenous unfractionated heparin (10,000 units followed by infusion of 15 units/kg/hr), diuretic and other supportive treatment. After couple of hours her saturation started to increase up to 70%. On 2nd day of admission (DOA), patient went into labour and dead fetus was delivered with the help of forceps. On 4th DOA, tracheal extubated was done and oxygen saturation was maintained on facemask.

On 5th day, echocardiography was done and showed reduction of systolic function with LVEF of 15-20%, mild mitral regurgitation (MR) and mild tricuspid regurgitation (TR). Intravenous infusion of unfractionated heparin (15 units/kg/hr) was changed to low molecular weight heparin subcutaneously before shifting to ward.

With the chest X-ray (Figure 1), electrocardiography (Figure 2), echocardiography and improvement of patient condition with anticoagulant and diuretic, we made the impression of peripartum cardiomyopathy with possible pulmonary embolism. Patient was shifted to the ward on 5th day and was stable throughout the admission in the ward and was discharged on eighth day of admission.

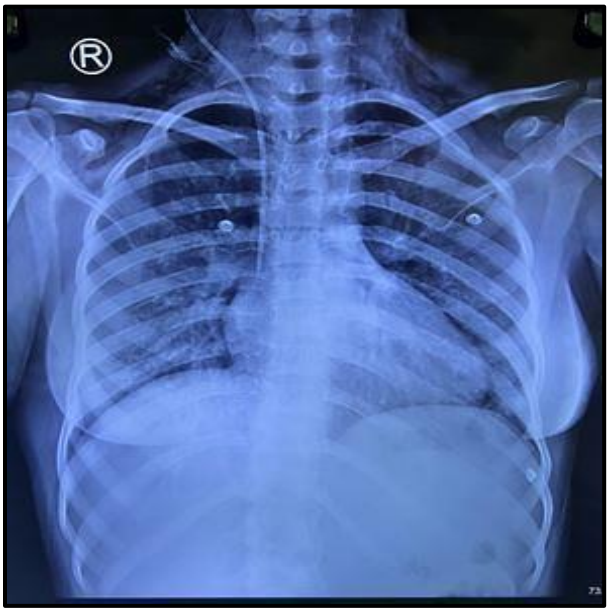

Figure 1: Chest X-ray showing an increased cardiothoracic ratio.

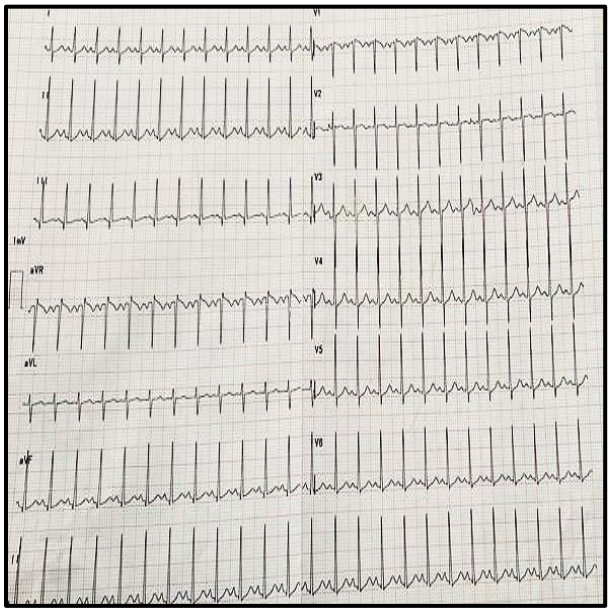

Figure 2: Electrocardiography showing sinus tachycardia.

Patient was reviewed by a cardiologist and discharged on oral torsemide 10 mg, spironolactone 12.5 mg, Losartan 25 mg and Metoprolol XL 25 mg with advised to follow up in two weeks in cardiology outpatient department (OPD). The ABG and blood investigation reports done during hospital stay are listed in Table 1 and Table 2 respectively. Unfortunately, the computed tomography pulmonary angiogram (CTPA) was not done due to technical problem in the scan machine and couldn't confirm our diagnosis as PE.

Table 1: Arterial blood gas

Date & Time	**12/12 9:00am**	**12/12 5:30 pm**	**12/13; 7:25 am**	**12/14 8 am**	**12/15 4:15pm**
pH	7.19	7.23	7.38	7.4	7.49
PaO2 (mmHg)	37.8	53.4	82.1	71.8	93.5
PaCO2 (mmHg)	33.3	43.4	33.4	21.9	27.9
SaO2 (%)	43.7	78.5	95.6	92.3	97.9
HCO3 (mmol/L)	12.6	17	20.4	22.1	23.9
Lactate (mmol/L)	7.1	2.7	1.5	1.2	1.7

Table 2: Laboratory analysis

Parameters	**Day 1**	**Day 2**	**Day 3**
Hemoglobin (gm/dl)	10.9	7.5	8.0
PCV (%)	41.2	23.3	24.3
Total Count (cell/mm^3)	15,300	11000	9100
Platelets(cells/mm^3)	4,44,000	196000	163000
Blood sugar (mg/dl)	118	95	80
Urea/Creatinine (mg/dl)	28/0.8	68/2.38	103/1.8
Na^+/K^+ (mmol/L)	131/4.5	130/4.4	138/4.0
Total protein/Albumin (gm/dl)	6.1/3.3	5.2/2.8	N/A*
Total/direct bilirubin (mg/dl)	3.23/1.65	2.5/1.36	N/A
SGPT/SGOT (U/L)	350/623	244/479	N/A
ALP/GGT (U/L)	462/41	345	N/A
LDH (U/L)	1033	N/A	N/A
D-Dimer (mg/dl)	5.17	N/A	N/A
PT/INR (secs)	18/1.22	18/1.22	15/1.0
aPTT (secs)	N/A	N/A	28 secs

*Not applicable = N/A

DISCUSSION

Peripartum cardiomyopathy is a rare life-threatening complication of pregnancy with a high mortality rate of around 50% (3–5). It may be associated with severe and lasting complications including severe HF, pulmonary edema, cardiogenic shock, cardiopulmonary arrest secondary to HF or arrhythmias, thromboembolic complications, and death. The hypercoagulable state of pregnancy and stasis of blood in patients with PPCM and LVEF <35%, are at risk for development of thromboembolism affecting 6.6% to 6.8% of women (2). The symptoms of the normal physiologic changes of pregnancy mimic to those of heart failure and PE (Table 3) (6).

Table 3: Differential diagnosis of dyspnea in pregnancy

Diagnosis	Signs and Symptoms	Diagnostic tests
Benign dyspnea of pregnancy	Gradual onset mild dyspnea, S3 may be present, normal SpO_2 and jugular venous distention (JVD)	Chest X-ray (CXR) and echocardiography would be normal
Peripartum cardiomyopathy	Symptoms of right heart failure common in 1-6 weeks of postpartum period, JVD, pulmonary rales, peripheral edema; laterally displaced apical impulse	CXR suggestive of pulmonary edema and possible cardiomegaly; LVEF <45 % and global hypokinesia on echocardiography
Severe pre-eclampsia or eclampsia	Edema usually in antepartum, associated headache; hypertension, JVD, pulmonary rales and peripheral edema, S4 may be present	Proteinuria, CXR shows pulmonary edema; echocardiography shows preserved or mildly decreased LVEF, left ventricle hypertrophy (LVH) present if chronic hypertension
Cardiac dysfunction (secondary to ischemia or stress cardiomyopathy)	Acute HF symptoms preceded by chest pain, JVD, pulmonary rales, murmur of ischemic mitral regurgitation (MR) is present	ECG shows ischemic changes; raised cardiac specific enzymes; CXR reveals pulmonary edema; segmental wall motion abnormalities with or without ischemic MR on echocardiography; coronary angiography may show stenosis/occlusion/dissection

Cardiac dysfunction secondary to arrhythmia	Heart failure symptoms preceded by palpitations, tachycardia, pulmonary rales, peripheral edema	ECG shows tachyarrhythmia; CXR shows pulmonary edema; echocardiography shows diffuse hypokinesia
Previously undiagnosed valvular disease (eg, rheumatic valve disease)	Features of right heart failure usually present in late second trimester; JVD, pulmonary rales, peripheral edema, murmur of valvular stenosis or regurgitation (or both) present	ECG show chamber enlargement/or hypertrophy; CXR shows pulmonary edema with enlargement of left atrium (LA) and pulmonary artery (PA) in mitral stenosis; echocardiography shows valve lesion(s)
Pulmonary embolism	Sudden onset dyspnea associated with pleuritic chest pain during peripartum or up to 8 weeks postpartum, may be accompanied by features suggestive of deep vein thrombosis (DVT), tachycardia, associated right ventricle (RV) heave, accentuation of P2; pulmonary exam often unremarkable	CT pulmonary angiography shows pulmonary embolism; D-Dimer levels are non-specific; ABG shows respiratory alkalosis/hypoxemia/may be non-specific; Echo cardiography to evaluate the size of RV
Amniotic fluid embolism syndrome	Acute onset cardio-respiratory collapse during or immediately after labor, usually associated with bleeding from disseminated intravascular coagulopathy (DIC); tachypnea, hypotension, and crackles are present	Raised D-dimer, low fibrinogen, and thrombocytopenia
Asthma	History of cough, wheezing; use of accessory muscles; on chest examination wheezes or decreased air movement (or both)	Positive provocation test or response to bronchodilator challenge in pulmonary function test

Pneumonia	Acute onset fever with chills and rigors, breathlessness, productive cough (blood stained sputum), tachypnea, decrease in $SpO_{2,}$ bronchial breathing, crepitations and decreased air entry on auscultation	Increased total leucocyte count); CXR shows consolidation and interstitial infiltrates, echocardiography is normal

The risk factors for PPCM include primiparity, multiparity, advanced maternal age, multifetal pregnancy, pre-eclampsia and gestational hypertension, asthma, autoimmune disease, substance abuse, obesity, thyroid disorder, prolong use of tocolysis and African American race (6, 7). The one risk factor we could identify in our patient was primiparity.

The worldwide variation in the incidence of PPCM ranges from 1 in 102 live births to 1 in 20000 live births due to different ethnicities (6). With the higher maternal mortality rate of PPCM complicated with PE, the management of such patient requires a multidisciplinary setting with familiarization of the various pharmacotherapies available for better prognostic outcome. Because of unavailability of bed side chest x-ray and echocardiography in MICU, we had difficulty in making the diagnosis and proper management.

The crucial decision that we made based on high clinical suspicion of PE to start therapeutic unfractionated heparin at correct time made her to survive as she had already started to develop kidney injury. Early use of anticoagulant should be strongly considered due to the high maternal mortality for an untreated PPCM with PE.

Women diagnosed with PPCM should be counseled about the risks of subsequent pregnancy and should be closely monitored throughout the pregnancy and until six months postpartum with frequent clinical examinations and serial echocardiograms.

CONCLUSION

The advancement in imaging technology makes the clinician easier to make a primary diagnosis of PPCM. As echocardiogram is a noninvasive bed side procedure, should be consider especially when life threatening cardiopulmonary pathologies are suspected to institute proper treatment and to avoid potentially fatal errors whenever feasible. However, in poor resource settings and in do or die situation where further investigations are not possible, a confident clinical diagnosis must be made in view of high clinical suspicion based on history, clinical presentations and reports that are readily available.

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CONFLICT OF INTEREST

None

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