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Use of Point-of-Care Ultrasound in the Diagnosis of Postpartum Cardiomyopathy

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Use of Point-of-Care Ultrasound in the diagnosis of Postpartum Cardiomyopathy (PPCM)

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Abstract:

Postpartum cardiomyopathy (PPCM), also known as peripartum cardiomyopathy is a rare form of heart failure (HF) that occurs in the late stages of pregnancy or in the early postpartum period. For it to be classified as PPCM, it must occur in the absence of another identifiable cause for HF and have left ventricular (LV) systolic dysfunction with an LV ejection fraction (LVEF) less than 45% . Here we present the case of a 46-year-old female G3P2 presented 5 days postpartum cesarean section delivery in acute respiratory distress. In which point of care ultrasound was used for assessment of the lungs to visualize B-lines and significant reduction in ejection fracture of the heart, allowed us to make the decision to pursue management of heart failure and have successful outcomes for this patient.

Case Presentation:

46-year-old female ,G3P2 ,presented 5 days postpartum cesarean section delivery in acute respiratory distress. Patient was accompanied by husband who stated symptoms began earlier on the day of presentation and quickly progressed. Her husband reported associated cough with productive reddish sputum with possible hemoptysis. Upon arrival to the emergency department patient was in severe respiratory distress and decision was made to attempt noninvasive ventilation with high-flow nasal cannula (HFNC). The patient began to deteriorate rather quickly and went into severe respiratory distress where she pulled off all oxygen and started hitting her chest screaming “I can’t breathe”. Initial vitals included blood pressure (BP) 195/105, heart rate(HR) 110, Respiratory Rate (RR) 40, SPO2 86% on room air improved to 92%+ with HFNC. With her being recently postpartum the two main differential diagnosis were a pulmonary embolism (PE) and PPCM. As management differed for these diagnosis, a STAT bedside point of care ultrasound (POCUS) was performed. On lung window significant B-lines were seen bilaterally. A limited cardiac exam showed dilation of LV with significant decreased LVEF. In the absence of a D-sign nor a McConnell's sign. Decision was made to pursue BIPAP, diuretic and vasodilator. As the patient became more agitated, patient was ultimately intubated and admitted to the intensive care unit. Her past medical history was unclear as husband stated that 10 years ago patient had a cardiac event but is unsure what it was . The patient was not on any medications. The family history was negative. The social history was negative for tobacco use, alcohol use or recreational use.

Labs and imaging obtained included CBC, CMP, ABG, proBNP, Troponin, EKG, CXR, CTA Chest. Pertinent findings included an ABG indicating acute respiratory failure with hypercapnia. pH 7.11, pCO2 54.2, pO2 133.0, Bicarb 17.2. proBNP 2,515. Troponin T HS result was 16 which increased to 67. Leukocytosis with a WBC of 16.22 was seen. The EKG showed sinus tachycardia at 101 beats per minute with a left bundle branch block with a possible left atrial enlargement and a prolonged QTc at 495. [Figure 1]. CXR showed bilateral infiltrates. [Figure 2]. CTA Chest showed no large saddle PE. Infiltrates were seen bilaterallly. [Figure 3 and 4]. After PE and MI were ruled out, the differential diagnosis included multifocal pneumonia versus acute cardiomyopathy. Patient was given broad spectrum antibiotics in addition to IV diuretic. After admission, patient obtained a formal transthoracic echocardiograph (TTE), which revealed a significant decrease in EF, indicating approximately 25% with severe global hypokinesis as seen in Figure 5-7. Beta-blockers and angiotensin-converting enzyme (ACE) inhibitors were also added by cardiology.

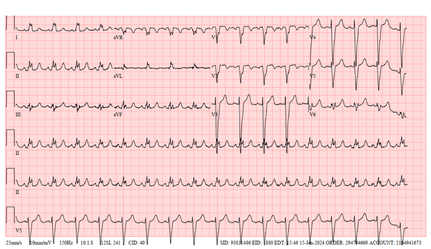


Figure 1: patients EKG showing Sinus tachycardia 101 beats per minute with a left bundle branch block with a possible left atrial enlargement and a prolonged QTc at 495.

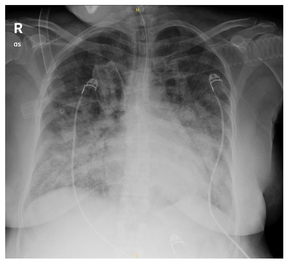


Figure 2: CXR demonstrating bilateral infiltrates

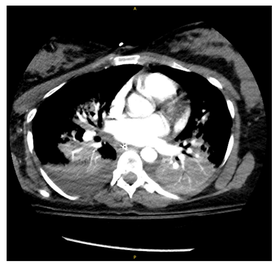


Figure 3: CT PE study showing no large saddle PE

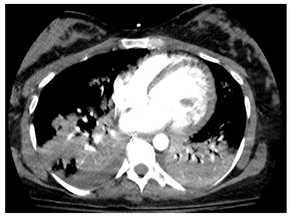


Figure 4: CT PE study showing infiltrative changes bilaterally

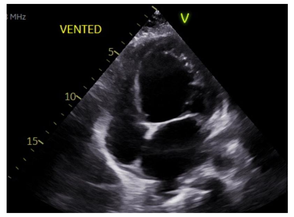
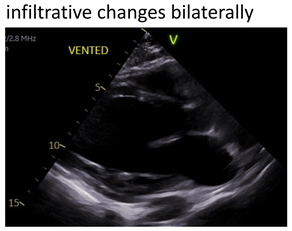


Figure 5: Transthoracic Echocardiography



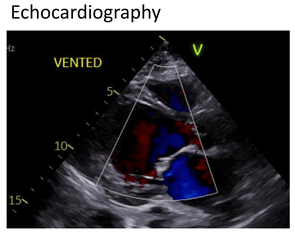


Figure 6 and 7: Transthoracic Echocardiography
*RA= Right Atrium, LA= Left Atrium, RV= Right Atrium, LV= Left Atrium, PA= Pulmonary Trunk, R PA= Right Pulmonary Artery

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Discussion:

PPCM is a rare cause of HF. For it to be classified as PPCM, it must occur in the absence of another identifiable cause for HF and have left ventricular (LV) systolic dysfunction with an LV ejection fraction (LVEF) less than 45% [1].
Risk factors that increase incidence of the disease include, age greater than 30, African descent, pregnancy with multiple gestation, maternal cocaine use, parity >4, long term oral tocolytic therapy, prior or concurrent preeclampsia, eclampsia or postpartum hypertension [2].

Patient presentation of PPCM will include dyspnea, cough, orthopnea, pedal edema and hemoptysis. Initial diagnosis may be difficulty as many of these symptoms are nonspecific and occur in normal pregnancy. Physical exam findings include elevated jugular venous pressure, displaced apical impulse, a third heart sound and a murmur of mitral regurgitation.

Diagnostic tools include electrocardiogram (ECG), Echocardiograph (TTE), Chest Radiograph (X-ray), Plasma brain natriuretic peptide (BNP). Differential diagnosis include myocardial infarction (MI), Pulmonary Embolism (PE), undetected congenital heart disease, or valvular disease [2].

In women with PPCM the mainstay of treatment is similar to treatment for acute on chronic HF with reduced EF. However, clinicians should be cognizant of adverse effects of medications on the fetus or breastfeeding neonate. Management includes oxygen therapy, betablockers, nitrites, diuretics, digoxin. If PPCM is diagnosed during pregnancy, OBGYN team should be consulted for planning of delivery if possible [3]. ACE inhibitors and aldosterone antagonists such as spironolactone are contraindicated during pregnancy but can be started in the postpartum period. Lastly if needed, inotropes are safe in both antepartum and postpartum period [4].

PPCM overall has a promising long term prognosis, with nearly 60 percent of patients having recovery of left ventricular function. Patients who wish to have future pregnancies should be educated about recurrence of PPCM [5].

Conclusions:

Postpartum cardiomyopathy is a rare cause of heart failure with reduced ejection fracture. In an acute setting it can be difficult to distinguish PPCM from other diagnosis. For example, pulmonary embolism, pneumonia, amniotic fluid embolism, pulmonary edema due to preeclampsia/eclampsia, and myocardial infarction.

In addition to clinical symptoms, portable chest x-ray and labs. A bedside point-of-care ultrasound assessment can be one of the most useless diagnostic tools. It can help navigate management in an acute emergent setting. Especially when choosing between anticoagulating for a possible large saddle pulmonary emboli versus treatment with diuretics, vasodilators and non-invasive ventilation for cardiomyopathy. Allowing a rapid assessment of the lungs to visualize B-lines and significant reduction in ejection fracture of the heart, allowed us to make the decision to pursue management of heart failure and have successful outcomes for this patient. The patient was ultimate extubated and discharged home to her newborn approximately 3-4 days after presenting to the emergency department.