Abstract

We present a case of a 25 year-old female who developed acute hypoxic respiratory failure secondary to postpartum heart failure with preserved ejection fraction. Despite essentially normal tissue Doppler velocities and E/e' ratio by existing guidelines for the adult population, available literature suggests that our patient's markers of diastolic function were significantly abnormal relative to what is expected in the third trimester of pregnancy. Advanced echo-Doppler analysis and her clinical presentation support the presence of underlying diastolic dysfunction. This case illustrates the challenges associated with the diagnosis of diastolic dysfunction during pregnancy and the need for pregnancy specific guidelines.

Introduction

We present a case of postpartum heart failure with preserved ejection fraction (pHFpEF) due to diastolic dysfunction. While peripartum cardiomyopathy, characterized by profound systolic dysfunction, is a well-described entity, there are few examples of acute peripartum heart failure secondary to diastolic dysfunction. Furthermore, there is little consensus on how to assess diastolic function in pregnancy. Reported risk factors for diastolic dysfunction in pregnancy include grand multiparity, preeclampsia and gestational hypertension.[1, 2]

This case features a patient with seemingly 'normal' diastolic parameters by current guidelines, who nevertheless, had clear clinical manifestations of elevated filling pressures. It illustrates (1) the challenges associated with the diagnosis of diastolic dysfunction during pregnancy; (2) the value of Doppler echocardiographic techniques and strain imaging in detecting subclinical cardiomyopathy; and (3) the need for specific guidelines for the assessment of diastolic function in pregnancy.

Clinical presentation

A 25-year-old G6P2134 woman without prior cardiac history was admitted in active labor at 32 weeks gestation of an otherwise uncomplicated twin pregnancy. She had an unremarkable medical history. Prior to delivery, she was intermittently hypertensive with a systolic blood pressure as high as 155 mm Hg (range: 110 to 155 mm Hg). She was started on intravenous normal saline at a rate of 125 mL/hour per unit protocol. After two days of labor, she had an uncomplicated vaginal delivery of both twins. On postpartum day 1, the patient developed new onset chest pain and dyspnea. Repeat examination was notable for a blood pressure of 130/77 mmHg, heart rate of 68 bpm, respiratory rate of 24 rpm and SpO_2 of 92% on room air.

Auscultation of the chest revealed bibasilar rales. The patient was placed on supplemental oxygen by nasal cannula.

Diagnostic testing

Laboratory work-up for preeclampsia was unremarkable and inconsistent with this diagnosis. A 12-lead electrocardiogram showed sinus rhythm with 1st degree heart block. The chest radiograph revealed mild pulmonary edema (Figure 1). Transthoracic echocardiogram showed a dilated left atrium (LA) and ventricle (LV) with left atrial end systolic volume index (LAVi) 45 mL/m^2 (Figure 2), ejection fraction (EF) 60% and global longitudinal strain (GLS) -15.8% (Figure 3). The E/A ratio was 2.1 (Figure 4). The septal e' was 7 cm/s and lateral e' 12.2 cm/s with an E/e' ratio of 10.3. Estimated pulmonary artery systolic pressure was mildly elevated at 38 mmHg. There was systolic-to-diastolic flow reversal of pulmonary venous inflow. Doppler data, overall, were suggestive of elevated LA pressure.

Case Resolution

The leading differential diagnosis was that the patient had a subclinical cardiomyopathy primarily manifesting as diastolic dysfunction. Her symptoms resolved with diuresis and she was discharged on post-partum day 2 without further intervention.

Discussion:

We present the case of a woman who developed postpartum HFpEF: clear-cut pulmonary edema and a normal EF on contemporaneous echocardiography. We hypothesize that this patient had subclinical diastolic dysfunction which, in combination with the fluid shifts present in the peripartum period, produced a syndrome of postpartum HFpEF. In this context, we interpret her E/A ratio > 2 and diastolic dominant pulmonary venous inflow as indicating restrictive filling

and elevated LA pressure, respectively.[3, 4] Her abnormally low GLS and unexpectedly low tissue Doppler, both markers of underlying myocardial dysfunction, suggest diminished restoring forces and further corroborate the presence of diastolic dysfunction.

In Table I we review the available literature concerning diastolic function in pregnancy that led us to the above conclusion. As shown in the Table I, our patient's GLS was notably lower than what is expected in the third trimester, consistent with subclinical myocardial dysfunction.[5-8] Additionally, our patient's septal and lateral e' (7.0 cm/s and 12.2 cm/s, respectively) were both at least 2 standard deviations below the reported means in available literature.[3, 8-11] Likewise, both septal and lateral E/e' ratios (14.1 and 8.1, respectively) were greater than 2 standard deviations higher than the reported means and were consistent with the clinically evident elevated LA pressure.[3, 9-11]

We believe that this case illustrates some pitfalls of extrapolating the current ASE guidelines for the diagnosis of diastolic dysfunction in pregnant women. First of all, increased LAVi, a cardinal feature of DD, may be related to the physiologic volume expansion associated with pregnancy. Secondly, tissue Doppler velocities that are within the normal range in the non-pregnant state may be lower than 'normal' for pregnant women, reflecting the fact that tissue Doppler e' may be preload-sensitive.[12] Similarly, the ASE threshold of E/e' ratio > 14 as a criteria to diagnose diastolic dysfunction may lack sensitivity in pregnancy, again due to physiologic increases in e' velocities. In any event, this case illustrates the need for pregnancy specific guidelines in the assessment of diastolic function in pregnancy.

Postpartum heart failure with preserved ejection fraction

We hypothesize that our patient's symptoms were primarily related to impaired relaxation and stiffening of the left ventricle resulting in an abnormal end diastolic pressure volume relationship

(EDPVR) (Figure 5, C), rather than afterload (e.g. preeclampsia) or preload excess (e.g. iatrogenic volume loading) in the setting of a normal EDPVR (Figure 5, B). pHFpEF is not a well-described disorder, and is considered to be a separate entity from peripartum cardiomyopathy by current guidelines (by definition the LV ejection fraction is less than 45% in peripartum cardiomyopathy).[13, 14] The timing and clinical symptoms of pHFpEF are similar to that of peripartum cardiomyopathy, but often leads to a milder presentation with lesser elevations of serum BNP.[13, 15] Moderate and severe diastolic dysfunction, in particular, are associated with an increased mortality risk in the general population, but more data is needed to properly define severity of diastolic function in pregnancy and to understand its long-term consequences.[13, 14]

Conclusion:

This case highlights the fact that EF can belie the presence of subclinical myocardial dysfunction and the importance of strain imaging in patients at increased risk for developing a cardiomyopathy. Further, our patient's tissue Doppler velocities and E/e' ratio were either normal or near normal based on current guidelines for the general population, however, the available literature suggests that these parameters were significantly different than what would be expected for a woman in her third trimester of pregnancy. This highlights the need for more normative data in peripartum and postpartum women.

Author Contributions:

MG: Case review, drafting article, approval of article
LP: Case review, drafting article, approval of article
LF: Case review, critical revision of article, approval of article
CH: Case review, critical revision of article, approval of article
GA: Case review, critical revision of article, approval of article
LK: Case review, critical revision of article, approval of article

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Tables

Table I: Comparison of diastolic function indices throughout pregnancy

Echo parameter	Reference (age-matched)	First trimester	Third trimester	Severe preeclampsia	Our patient (PP-HFpEF)
GLS	-20.3±3.0[5] -20.3±3.4[7]	-21.3±2.6[5]	-18.9±3.0[5] -19.8±2.1[6] -19.9±3.4[7] -20.1±1.5[8]	-18.0±2.6[6] -19.1±1.5[8]	-15.8
Septal e' (cm/s)	14.0±3.0[9] 13.1±1.9[10]	13.3±2.6[3]	12.5±1.7[3] 11.6±1.9[8] 12.7±2.6*[9] 12.2±1.6[10] 12.5±2.4[11]	9.6±2.4[8]	7.0
Lateral e' (cm/s)	17.8±2.9[9] 17.5±1.9[10]	18.2±3.0[3]	17.8±3.1[3] 18.3±2.8*[9] 18.7±2.4[10]		12.2
E/septal e'	6.4±1.4[9] 7.5±1.1[10]	8.1±2.2[3]	6.8±1.5[3] 6.3±1.4*[9] 7.5±1.2[10] 6.4±1.2[11]		14.1
E/lateral e'	4.9±0.8[9] 5.6±1.0[10]	5.9±1.6[3]	4.8±1.3 3 4.3±0.9*[9] 4.9±1.0[10]		8.1
E/mean e'	5.4±1.4[7] 5.6 (5.0-6.2)[$16]	5.6 (4.3-6.5)[$ 16]	5.8±1.3[6] 6.1±2.3[7] 7.4±1.6[8] 6.0 (4.3-6.4)[$16] 5.7±1.0[17]	7.3±2.2[6] 10.8±2.8[8] 8.6±1.5[17]	10.3
PASP (mmHg)	28±5[7]		24±5[6] 30±5[7] 22.5±6.1[8]	27±5[6] 31±7.9[8]	38
LAVi (ml/m²)	20±7[7] 19±3[10] 32 (30-47)[$16]	37 (32-44)[$16]	24±7[7] 24±2[10] 34 (27-41)[$16] 17±4[11]		45

GLS, global longitudinal strain; E, early diastolic mitral inflow velocity; e' early diastolic mitral annular tissue velocity; PASP, pulmonary artery systolic pressure (estimated from tricuspid regurgitant jet velocity); LAVi, left atrial end systolic volume index.

*includes patients in second and third trimester.

[$]represents interquartile range.

Figure Legends

Figure 1: Chest radiograph

Portable chest radiograph revealing of mild pulmonary edema, trace bilateral pleural effusions, and enlarged cardiac silhouette. This constellation of findings was concerning for cardiogenic pulmonary edema due to elevated left sided filling pressures.

Figure 2: Left atrial end systolic volume index

Left atrial end systolic volume index, obtained via biplane method of disks, was increased to 45 ml/m^2. Left atrial dilation is an expected finding in pregnancy, particularly in the third trimester. LA = left atrium, LV = left ventricle, RA = right atrium, RV = right ventricle.

Figure 3: Global Longitudinal Strain

Global longitudinal strain was mildly reduced at -15.8%, consistent with an underlying cardiomyopathy.

Figure 4: Pulse Wave Doppler of Mitral Valve Inflow

Mitral inflow velocities were consistent with elevated left atrial pressure with an E/A ratio of 2.1. E = mitral valve inflow in early diastole, A = mitral valve inflow during atrial contraction.

Figure 5: Comparison of End Diastolic Pressure Volume Relationships

Comparison of the end diastolic pressure volume relationship of a patient with normal diastolic function and a normal volume status (A), to one with normal diastolic function and volume overload (B), finally, to one with diastolic dysfunction (C). In the presence of diastolic dysfunction, LV end diastolic pressures increase at relatively lower end diastolic volume due to increased LV stiffness ($\Delta P/\Delta V$).