

# Cardiomyopathy discovered during pregnancy: Insights from speckle tracking echocardiography in a cohort of pregnant patients

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## Original Article

### Abstract

**BACKGROUND:** Heart failure (HF) is considered the leading cause of cardiac-related morbidity and mortality during pregnancy. Peripartum cardiomyopathy (PPCM) presents diagnostic challenges, often mirroring dilated cardiomyopathy (DCM). The aim of the study is to evaluate echocardiographic features, including global and segmental longitudinal strain values, in pregnant women with a history of newly diagnosed left ventricular systolic dysfunction (LVSD) in the third trimester of pregnancy.

**METHODS:** This cross-sectional study, conducted in two referral cardio-obstetric clinics in Isfahan, Iran, enrolled pregnant women with newly diagnosed LV systolic dysfunction in the third trimester of pregnancy. A multidisciplinary pregnancy heart team assessed the patients. Reevaluation of patients and advanced echocardiographic investigation, including speckle tracking echocardiography (STE), were performed at least six months after delivery.

**RESULTS:** The study included 26 pregnant women. Baseline characteristics revealed varying NYHA functional classes and etiologies, including DCM or non-dilated LV cardiomyopathy and PPCM. Undiagnosed DCM with exacerbation during pregnancy or non-dilated LV cardiomyopathy were the most probable causes for LV systolic dysfunction (65.4%). In five cases, peripartum cardiomyopathy was more relevant. The mean global longitudinal strain (GLS) was -16.94% and -13.95% in PPCM and DCM, respectively. Significantly different regional longitudinal strain numbers among different LV segments in PPCM were observed ( $P=.042$ ), whereas the segmental strain in DCM patients did not differ.

**CONCLUSION:** When LVSD is discovered late in pregnancy, it is not easy for the authors to differentiate between peripartum cardiomyopathy and other cardiomyopathies. Advanced echocardiographic techniques, particularly GLS analysis, may be valuable in differentiating between these conditions.

**Keywords:** Cardiomyopathy; Global Longitudinal Strain; Echocardiography; Pregnancy; Dilated Cardiomyopathy; Peripartum Period

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### Introduction

Cardiac diseases present a significant burden during pregnancy, affecting 1 to 4% of pregnancies, with heart failure (HF) emerging as a leading cause of morbidity and mortality<sup>1</sup>. The cardiovascular system plays a

pivotal role in regulating physiological mechanisms during pregnancy, responding to increased demands and hypermetabolic states. These adaptations encompass alterations in choriodecidual space blood flow, enhanced perfusion to kidneys and skin,

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increased blood volume due to hyperaldosteronism, elevated heart rate, augmented cardiac output, reduced systemic vascular resistance, and expanded ventricular mass, volume, and end-diastolic volume<sup>2,3</sup>. However, when these adaptations occur in pregnant individuals with prior cardiomyopathy, pre-eclampsia, amniotic fluid embolism, or peripartum cardiomyopathy (PPCM), the likelihood of developing HF significantly increases<sup>4</sup>.

PPCM, despite sharing similar symptoms with dilated cardiomyopathy (DCM), poses challenges in differentiation, especially when the initial clinical presentation occurs late in pregnancy or early postpartum. The absence of a definitive molecular marker makes PPCM a diagnosis of exclusion based on clinical and echocardiographic features, often indistinguishable from heart failure caused by other etiologies. Previous beliefs associating PPCM with a type of dilated cardiomyopathy triggered by pregnancy and shared oxidative stress were challenged by later research revealing crucial differences. Current classifications of DCM exclude cases where loading conditions, coronary artery disease, and congenital heart diseases lead to systolic dysfunction, despite potential phenotypic similarities. Notably, the elevated levels of prolactin (PRL) during late pregnancy, coupled with heightened oxidative stress, likely contribute to the rapid development of PPCM<sup>5</sup>.

Despite the teratogenic potential of certain medications used for standard HF treatment, effective management becomes challenging in pregnant subjects affected. However, with appropriate medication and, more importantly, routine follow-up, a favorable prognosis is attainable, as a majority of PPCM patients fully recover within three to six months postpartum<sup>6</sup>. When encountering a pregnant woman with systolic dysfunction and heart failure symptoms in the late weeks of pregnancy, the authors consider diverse causes with varying prognoses. Advanced echocardiographic techniques, particularly global longitudinal strain (GLS), prove invaluable by offering insightful findings on structural and functional recovery. GLS, superior to left ventricular ejection fraction (LVEF) in detecting subtle LV dysfunction, provides a broader range of interpretation and independently predicts mortality in heart failure with reduced EF<sup>7</sup>.

Hence, recognizing the dearth of data concerning

GLS in women experiencing systolic heart failure during pregnancy, the authors' objective is to impart insights from their endeavors in employing repeated echocardiography, coupled with GLS measurements. This study involves patients newly diagnosed with left ventricular systolic dysfunction during the late stages of pregnancy at the authors' center. The assessments are conducted six months post-delivery, offering a comprehensive understanding of the structural and functional dynamics in this specific population.

## Methods

### *Study Protocol and Design*

This cross-sectional study was conducted at the Cardio-Obstetric clinics in Alzahra and Chamran hospitals, Isfahan, Iran, between 2022 and 2023. These clinics are tertiary referral centers for pregnant patients with cardiovascular diseases from both governmental and private pregnancy clinics in Isfahan province. The study adhered to the regional healthcare policy of Isfahan University of Medical Sciences.

Initial patient evaluations were conducted by the pregnancy heart team, comprising a cardiologist experienced in managing heart diseases during pregnancy, an echocardiography specialist, and an obstetrician-gynecologist with substantial experience in managing pregnancies complicated by heart diseases. Pregnant women classified as WHO class 2-3, 3, or 4 (8) with cardiovascular disease were referred to the Cardio-Obstetric clinics. Heart failure diagnosis adhered to the guidelines for managing cardiovascular diseases during pregnancy set by the Task Force for the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology (ESC)<sup>8</sup>.

All patients with systolic heart failure were referred to the clinic considering WHO class, and received available treatments. Also, an approach to de novo heart failure including myocarditis, ischemic heart disease, infiltrative disease, autoimmune disease, history of chemotherapy, substance abuse, tachycardia-mediated cardiomyopathy, endocrine-related cardiomyopathy based on updated regional and global guidelines was performed.

Patients were referred to the heart failure clinic for ongoing care. They were monitored every other week in the third trimester until delivery. If the heart

failure was severe or challenging to control, or if signs of fetal distress were detected, delivery was recommended. In instances where the heart failure was mild and manageable, and fetal assessment indicated no concerns, the decision was made to proceed with the pregnancy under close supervision within the hospital. After delivery, according to patient history and clinical presentation, workups including coronary evaluation, rhythm Holter monitoring, and CMR (it was available for only ten patients) were performed in the postpartum period. All patients were also receiving guideline-directed medical treatment (GDMT) considering their breastfeeding status and symptoms, with dose adjustments made during frequent visits under the supervision of the cardiology and obstetrics team.

In this study, 36 patients with newly diagnosed systolic heart failure in the third trimester of pregnancy that were referred to the authors' clinic underwent an echocardiographic study at the time of presentation. Left ventricular ejection fraction (LVEF) by the Simpson method, left ventricular end-diastolic diameter and left ventricular volume, LA volume, LV diastolic function, valvular function, and estimation of systolic PAP were evaluated for all of them. Data regarding the pregnancy status, including gestational age, history of previous pregnancies, and presence of pregnancy-induced hypertension or pre-eclampsia, were recorded in medical files.

After obtaining approval from the institutional ethical committee, eligible patients meeting the inclusion and exclusion criteria underwent echocardiography and strain measurement with the speckle tracking method 6 months postpartum while they received guideline-based medical treatment, with written informed consent.

**Inclusion Criteria:** Patients eligible for inclusion met the following criteria at presentation:

1. New diagnosis of HF secondary to left ventricular systolic dysfunction with LVEF <50% in the third trimester of pregnancy or postpartum.
2. eighteen years of age and more.
3. Time between evaluation echocardiography exam and achieving guideline-directed medical therapy for at least 6 months.
4. Available sufficient quality of echocardiographic images for offline analysis.

**Exclusion Criteria:** Patients were excluded if, at presentation, they met any of the following criteria:

1. Pre-existing known cardiac or thyroid disease or any substance abuse.
2. Concomitant therapy for other systemic illness.
3. History of myocardial infarction and/or significant coronary artery disease (stenosis >50%, ruled out by previous coronary artery angiography or computed tomography).
4. Primary valvular disease.
5. Hypertensive or congenital heart disease.
6. Known hypertrophic or restrictive cardiomyopathy.
7. Chemotherapy, chest irradiation.

### *Echocardiography*

The left ventricular ejection fraction was calculated using Simpson's method of disks and verified by visual inspection by echocardiographers. Septal and LV posterior wall thickness, LV diastolic diameters and volumes, and left atrial (LA) volume were measured from 2D images. All patients underwent conventional echocardiography with the same tool and setting as in the baseline study. All measurements were taken from the first echocardiographic study performed at presentation and studies closest to 6 months of follow-up. Furthermore, in echocardiography 6<sup>th</sup> to 9<sup>th</sup> months post-partum for each study, the regional longitudinal systolic strain in the 2-3 and 4 chamber views was obtained with a Philips EPIC 7 S5-1 probe and offline QLAB software. The automatic tracking in systole was adjusted manually, and GLS was calculated by the software as an average of the 18 segments. Strain measurements were performed by two different echocardiographers and mean values were obtained, blinded to the clinical characteristics of the patients.

### *Statistical Analysis*

Continuous and categorized variables were summarized as Mean  $\pm$  SD and frequency (percentage), respectively. Taking into account the results of the Shapiro-Wilk test along with the skewness and kurtosis indices of the data, it was assumed that all variables followed a normal distribution. Therefore, parametric tests were selected for data analysis. Differences between-group were assessed using independent sample T-test and ANOVA. Post-hoc analyses were performed using the Tukey HSD test. To examine the correlation between LVEF and GLS, Pearson linear correlation

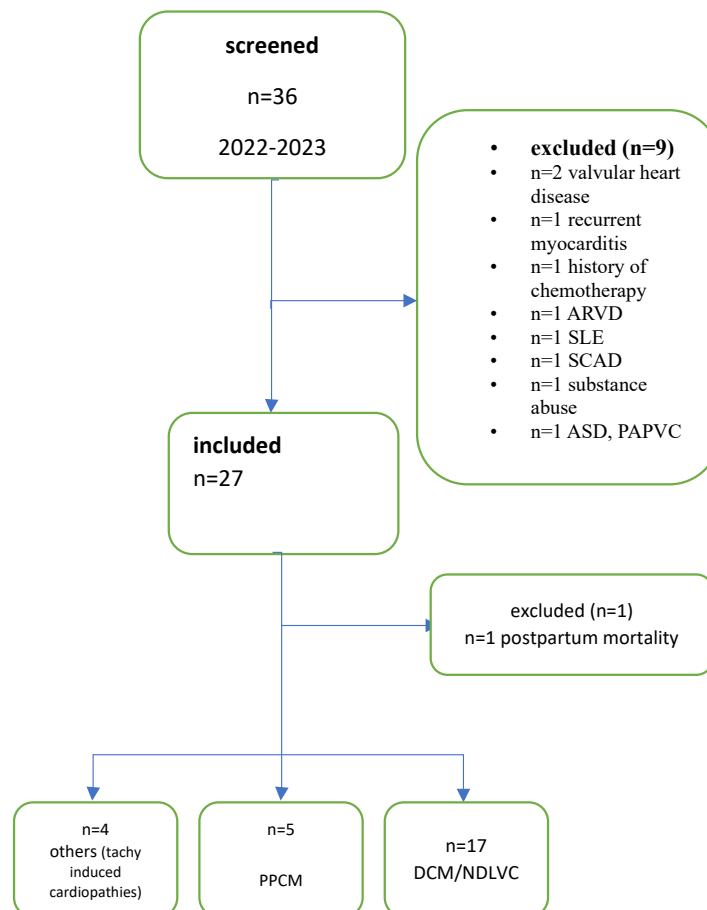
analysis was employed. The comparison of data from baseline and follow-up surveys was performed using a paired sample T-test for variables. All analyses were conducted using the Statistical Package for Social Sciences (SPSS Inc. PASW statistics for Windows, Chicago) Statistics version 24.

## Results

### Baseline Characteristics

Among 36 pregnant women recently diagnosed with left ventricular (LV) systolic dysfunction in the late stages of pregnancy at the Cardio-Obstetric clinic, nine patients with left ventricular ejection fraction (LVEF)  $\leq 50\%$  were excluded based on past medical history, baseline lab data, and alternative diagnoses. Also, one case of maternal mortality due to cardiac arrhythmias occurred. The exclusion criteria led to the inclusion of 26 pregnant women in the study

(Figure 1). Table 1 provides an overview of the baseline general clinical characteristics of the study population. The mean age of the participants was  $31.4 \pm 5.8$  years, with 18 patients being primigravida. At the initial visit, eight patients were classified as New York Heart Association (NYHA) functional class 2, twelve as class 3, and six as class 4. Three patients exhibited mild LV dysfunction, 10 had moderate dysfunction, and 13 had severe systolic dysfunction at baseline. Additionally, five patients were diagnosed with pre-eclampsia. Eight patients underwent vaginal delivery, while the remaining 18 patients were scheduled for cesarean section. Based on echocardiography, CMR, and Holter monitoring, clinical conditions, and symptoms, 26 patients were eligible, and nine patients were excluded after additional diagnostic evaluations. One patient with bicuspid aortic valve and moderate to severe aortic regurgitation, one patient with a



**Figure 1.** The design of the study and the chart outlining the criteria used to screen and include patients with PPCM and DCM. SCAD: Spontaneous Coronary Artery Dissection, ARVD: Arrhythmogenic Right Ventricular Dysplasia, SLE: Systemic Lupus erythematosus, ASD: Arterial Septal Defect, PAPVC: Partial Anomalous Pulmonary Venous connection NDLVC= non-dilated left ventricular cardiomyopathy

history of chemotherapy and radiation for Hodgkin lymphoma four years before pregnancy, one patient with a history of addiction and substance abuse, one patient with severe mitral regurgitation and moderate mitral stenosis due to rheumatic heart disease, one patient with a history of myocarditis with LVEF = 50% before pregnancy and recurrence three days after delivery with ventricular arrhythmias and severe impaired systolic function, one case of systemic lupus erythematosus in the setting of approach to de novo heart failure during pregnancy, one case of partial anomalous pulmonary venous connection and atrial septal defect in echocardiography one month after delivery that was confirmed by TEE. One patient was diagnosed with arrhythmogenic right ventricular cardiomyopathy (ARVC) confirmed by CMR report three months after delivery, one with spontaneous coronary artery dissection (SCAD) based on coronary CT angiography and IVUS who was presented with chest pain and pulmonary edema at the second day postpartum, and one case of maternal mortality due

to cardiac arrhythmias and sudden cardiac death 21 days after delivery. Eventually, 26 patients underwent echocardiography with strain measurement at least six months while receiving GDMT (Figure 1).

#### *The etiology of HF*

Based on echocardiography, laboratory data, and cardiac magnetic resonance imaging (CMR), the authors classified peripartum cardiomyopathy (PPCM) as the etiology of heart failure (HF) in five patients who had documented normal cardiac function according to echocardiography studies before pregnancy. In 17 patients, the etiology of HF was considered to be dilated cardiomyopathy (DCM) or non-dilated left ventricle (LV) cardiomyopathy, which had not been previously diagnosed. The remaining four patients were considered to have tachycardia-induced cardiomyopathy, although it is uncertain whether it resulted from PPCM or if their arrhythmia led to a reduction in ejection fraction (Figure 1).

**Table 1.** Baseline general clinical characteristics of the study population

Age, year Mean±SD		31.4±5.8
Gestational age at diagnosis Mean±SD		33.1±1.17
Gestational age of delivery Mean±SD		36.3±1.16
Etiology n (%)	DCM/ NDLCV	17(65.4)
	PPCM	5(19.2)
	Other	4(15.4)
EF n (%)	mild	3(11.5)
	moderate	10(38.5)
	severe	13(50.0)
NYHA n (%)	2	8(30.8)
	3	12(46.2)
	4	6(23.1)
prim gravida n (%)		18(69.2)
Preeclampsia N (%)		5(19.2)
	PPCM	3(60)
	DCM/ NDLCV	2(11.7)
Type of delivery N (%)	NVD	8(30.7)
	C-section	18(69.3)

LV EF: left ventricular ejection fraction; EDV: end- diastolic volume; LV: left ventricle; LA: left atrium; NDLCV: non-dilated left ventricular cardiomyopathy

**Table 2.** Echocardiography findings from baseline and follow-up visit

	Baseline (Mean±SD)	After 6 to 9 months of delivery (Mean±SD)	P-value*
LV EF, %	32.54±7.60	35.62±9.76	0.147
EDV index, ml/m <sup>2</sup>	65.27±9.96	60.02±9.53	<0.001
LV diastolic diameter, cm	5.01±0.80	4.61±1.15	0.028
LA volume index, ml/m <sup>2</sup>	31.73±4.22	29.31±3.63	<0.001

LV EF: left ventricular ejection fraction; EDV: end- diastolic volume; LV: left ventricle; LA: left atrium

\* Paired T test



**LVEF**

The baseline LVEF was moderately decreased (Table 2), with a mean LVEF of 32.5% at baseline and 35.6% at follow-up 6 to 9 months after delivery (P = 0.147), indicating statistical non-significance. LVEF did not improve in the total population 6 to 9 months after delivery. However, patients with PPCM exhibited complete or partial recovery (more than a 10% increase but less than 55%). Despite this, diastolic LV volume and diameter significantly decreased, and indices improved after delivery (P = 0.028). One patient recovered to LVEF >50% but had an abnormal global longitudinal strain (GLS) of -15.3%.

**Global Longitudinal Strain (GLS)**

Table 3 presents findings from global and regional longitudinal strain obtained from apical two, three,

and four-chamber views using the speckle tracking method. In follow-up echocardiography, the mean GLS was -14.73% ( $\pm 2.40\%$ ). Global longitudinal strain and regional strain of apical 4 chamber segments were significantly higher in PPCM patients compared to DCM patients (mean GLS -16.94% in PPCM and -13.95% in DCM, P = 0.011; mean apical four-chamber segments -18.02% in PPCM and -14.02% in DCM, P = 0.009) (Table 3). Patients with PPCM exhibited significantly different regional longitudinal strain numbers among different views (P = 0.042), whereas segmental strain in DCM patients did not differ across echocardiography views (Table 4). As shown in Table 5, further pairwise comparison revealed significantly different strain numbers in the segmental strain of the apical 3 chamber view compared to segments obtained from the apical 2 and 4 chamber views in patients with PPCM (P = 0.037).

**Table 3.** Findings from global and regional longitudinal strain obtained from apical 2 chamber view, apical 3 chamber view, and apical 4 chamber compared between PPCM and DCM/NDLVC patients.

	DCM/NDLVC (Mean $\pm$ SD)	PPCM (Mean $\pm$ SD)	P-value*
GLS	-13.96 $\pm$ 2.18	-16.94 $\pm$ 1.59	0.011
AP3	-14.12 $\pm$ 2.57	-15.70 $\pm$ 1.92	0.221
AP2	-14.12 $\pm$ 2.88	-16.50 $\pm$ 1.41	0.093
AP4	-14.03 $\pm$ 2.84	-18.02 $\pm$ 2.03	0.009

GLS: global longitudinal strain; AP2: apical 2 chamber view; AP3: apical 3 chamber view; AP4: apical 4 chamber view; DCM: dilated cardiomyopathy; PPCM: peripartum cardiomyopathy. \*Independent sample T test

**Table 4.** Comparison of findings from regional longitudinal strain obtained from apical 2 chamber view, apical 3 chamber view, and apical 4 chamber in each type of heart failure

TYPE		Mean $\pm$ SD	P-value*
DCM/NDLVC	AP3	-14.12 $\pm$ 2.57	0.957
	AP2	-14.12 $\pm$ 2.88	
	AP4	-14.03 $\pm$ 2.84	
PPCM	AP3	-15.70 $\pm$ 1.92	0.042
	AP2	-16.50 $\pm$ 1.41	
	AP4	-18.02 $\pm$ 2.03	
Other	AP3	-13.20 $\pm$ 3.08	0.116
	AP2	-17.61 $\pm$ 4.00	
	AP4	-14.63 $\pm$ 2.29	

NDLVC: non-dilated left ventricular cardiomyopathy

\*Repeated measure ANOVA

**Table 5.** Post-hoc analysis of the differences among regional longitudinal strain obtained from apical 2 chamber view, apical 3 chamber view, and apical 4 chamber in PPCM patients

TYPE	view		Mean Difference of Longitudinal Strain $\pm$ SE	P-value *
PPCM	3chamber	2chamber	.800 $\pm$ .259	.037
	2chamber	4chamber	1.520 $\pm$ .700	.096
	4chamber	3chamber	-2.320 $\pm$ .753	.037

SE: standard error

\* Turkey HSD test

However, there was no significant difference in strain numbers between the apical 2 and 4 chamber views in this patient population ( $P = 0.096$ ). Linear correlation analysis between GLS and the final LVEF indicated no significant correlation between LVEF and GLS ( $p$ -value = 0.122,  $R = -0.311$ ).

### Discussion

PPCM and DCM share common etiological factors, including genetic mutations, oxidative stress, and damage to heart vessels and muscle fibers<sup>5,9</sup>. Distinct pathways likely lead to each condition, with the hormone 16 kDa PRL playing a crucial role in PPCM but not in DCM<sup>5,8</sup>. The rapid decline in heart function observed in PPCM patients may be attributed to elevated levels of 16 kDa PRL, in conjunction with high oxidative stress. However, other factors, such as a lack of protection against oxidative stress or a fragile blood vessel system, could contribute to PPCM. Despite these commonalities, differences in disease progression and outcomes underscore the importance of considering PPCM and DCM as distinct conditions<sup>5</sup>.

Global longitudinal strain (GLS) emerges as a valuable measure for a spectrum of left ventricular heart diseases, proving more reproducible than traditional measures like LVEF and offering additional prognostic value for detecting subclinical LV dysfunction<sup>10</sup>. A study by Briasoulis *et al.* on 47 PPCM patients with reduced LVEF found that baseline GLS lacked associations with all-cause mortality, rehospitalization, or LVEF recovery, emphasizing the need for repeated measures over time<sup>11</sup>. In a prospective study of 89 women across 30 centers (Investigations of Pregnancy Associated Cardiomyopathy consortium), GLS was independently associated with adverse outcomes, including death, heart transplant, LVAD implantation, or persistently decreased LVEF <50% over one year of follow-up<sup>12</sup>.

In this study, post-delivery medical treatment aligned with GDMT protocols, adjusting for patient symptoms, LVEF, and lactation status. Echocardiography, CMR, and Holter monitoring findings led to the identification of five PPCM and 17 DCM cases, alongside one ARVD, instances of frequent PVCs, Afib, and SCAD. Unfortunately, the authors reported one case of mortality three weeks

post-delivery as sudden cardiac death. Decision-making complexities surrounding ICD implantation within six months postpartum arose due to the potential for LVEF recovery with ongoing medical therapy, especially if PPCM was a more probable diagnosis. Additional modalities, particularly CMR for myocardial fibrosis detection, became imperative.

The authors' findings underscore significantly higher GLS and segmental strain in the apical four-chamber view for PPCM patients compared to DCM. Notably, they observed significant differences in regional strain numbers between views for PPCM patients, unlike those with DCM. This suggests the potential utility of GLS in distinguishing between PPCM and DCM patients. There was a non-significant change in LVEF on the echocardiography data for DCM patients. Despite non-significant LVEF changes in echocardiography for DCM patients, the wide range of NYHA functional classes and LV dysfunction among participants highlights the varied causes of HF during pregnancy.

The limitations of traditional measures in capturing changes in cardiac function in pregnant women with HF are evident in the authors' study. Advanced echocardiographic techniques, such as Speckle tracking echocardiography, were crucial in accurately assessing cardiac function. It showed differences in GLS between PPCM and DCM patients, as well as echocardiography in different views, with the aim of assessing cardiac function in this population accurately. This highlights the importance of advanced echocardiographic techniques in these patients.

Establishing reference ranges for GLS cut points in normal pregnancy could aid in distinguishing pathological vs. normal pregnancy symptoms<sup>13</sup>.

Studies on healthy pregnant women indicate minimal changes in GLS during pregnancy<sup>14</sup>, while investigations into 3D STE in pregnant women demonstrate modified myocardial deformation, reverting to baseline post-delivery<sup>15</sup>. Despite improvements in LVEF, EDV, and ESV, GLS remained mildly abnormal in a study of 53 pregnant women with PPCM<sup>16</sup>.

The strength of this study is the diagnostic reevaluation of patients with LV systolic dysfunction who were diagnosed during pregnancy for the first time to differentiate between different causes of systolic heart failure to some extent, as well as

planning medical treatments and prognosis. Among the limitations of this study, the following can be mentioned: GLS measurement in HF patients was not done for most of the patients at the time of diagnosis due to not suitable quality for strain analysis, and GLS measurement were examined in the final echocardiography, which was at 6 to 9 months after the diagnosis of systolic dysfunction. There is no comparison of GLS in the follow-up echo of these patients.

The small sample size is a limitation, emphasizing the need for larger-scale studies.

### Conclusion

In conclusion, this study accentuates the limitations of traditional cardiac function measures and underscores the importance of advanced imaging techniques like Speckle tracking echocardiography. The clinical implications of distinguishing between PPCM and DCM, particularly regarding patient management and outcomes, necessitate further exploration. Future research should address limitations, incorporating larger sample sizes to enhance the generalizability of findings and provide a more comprehensive understanding of systolic dysfunction and heart failure during pregnancy.

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### Conflict of Interest

Conflict of Interest Disclosure: There are no conflicting interests that might influence the impartiality of the research documented.

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### Author's Contributions

Parvin Bahrami: Conceived and designed the study;

obtained funding; managed data collection, curation, and analysis; and participated in drafting the manuscript and tables. Azam Soleimani: Contributed to the acquisition of data; critically revised the manuscript for important intellectual content. Reihaneh Zavar: Assisted in patient recruitment and data collection. Hosein Masoumi: Contributed to the analysis, and interpretation of the data; provided critical input during manuscript preparation; and drafted and revised the manuscript, tables, and figure. Farzad Adelparvar: Participated in drafting the manuscript.

### References

1. Elkayam U, Goland S, Pieper PG, Silversides C. High-risk cardiac disease in pregnancy: part I. *J Am Coll Cardiol.* 2016;68(4):396-410. <https://doi.org/10.1016/j.jacc.2016.05.048>
2. Dorn GW. The fuzzy logic of physiological cardiac hypertrophy. *Hypertension.* 2007;49(5):962-70. <https://doi.org/10.1161/hypertensionaha.106.079426>
3. Carlin A, Alfirevic Z. Physiological changes of pregnancy and monitoring. *Best Pract Res Clin Obstet Gynaecol.* 2008;22(5):801-23. <https://doi.org/10.1016/j.bpobgyn.2008.06.005>
4. Anthony J, Sliwa K. Decompensated Heart Failure in Pregnancy. *Card Fail Rev.* 2016 May;2(1):20-6. <https://doi.org/10.15420/cfr.2015.24.2>
5. Bollen IA, Van Deel ED, Kuster DW, Van Der Velden J. Peripartum cardiomyopathy and dilated cardiomyopathy: different at heart. *Front Physiol.* 2015 Jan 15;5:531. <https://doi.org/10.3389/fphys.2014.00531>
6. Rodriguez Ziccardi M, Siddique MS. Peripartum Cardiomyopathy. [Updated 2023 Jul 17]. In: *StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.* <https://www.ncbi.nlm.nih.gov/books/NBK482185/>
7. Potter E, Marwick TH. Assessment of Left Ventricular Function by Echocardiography: The Case for Routinely Adding Global Longitudinal Strain to Ejection Fraction. *JACC Cardiovasc Imaging.* 2018 Feb;11(2 Pt 1):260-74. <https://doi.org/10.1016/j.jcmg.2017.11.017>
8. Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J, Blomström-Lundqvist C, Cifková R, De Bonis M, et al. 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy. *Eur Heart J.* 2018 Sep 7;39(34):3165-241. <https://doi.org/10.1093/eurheartj/ehy340>
9. Morales A, Painter T, Li R, Siegfried JD, Li D, Norton



- N, Hershberger RE. Rare variant mutations in pregnancy-associated or peripartum cardiomyopathy. *Circulation*. 2010 May 25;121(20):2176-82. <https://doi.org/10.1161/circulationaha.109.931220>
10. Hilfiker-Kleiner D, Kaminski K, Podewski E, Bonda T, Schaefer A, Sliwa K, et al. A cathepsin D-cleaved 16 kDa form of prolactin mediates postpartum cardiomyopathy. *Cell*. 2007 Feb 9;128(3):589-600. <https://doi.org/10.1016/j.cell.2006.12.036>
  11. Briasoulis A, Mocanu M, Marinescu K, Qaqi O, Palla M, Telila T, et al. Longitudinal systolic strain profiles and outcomes in peripartum cardiomyopathy. *Echocardiography*. 2016 Sep;33(9):1354-60. <https://doi.org/10.1111/echo.13277>
  12. Davis EM, Ewald G, Givertz MM, Rajagopalan N, Cooper Jr LT, Briller J, et al. Maternal Obesity Affects Cardiac Remodeling and Recovery in Women with Peripartum Cardiomyopathy. *Am J Perinatol*. 2019 Apr;36(5):476-83. <https://doi.org/10.1055/s-0038-1669439>
  13. Ajmi H, Abid D, Milouchi S, Louati D, Sghaier A, Choura D, et al. Interest of speckle tracking in the detection of cardiac involvement in pregnant women with hypertensive disorder. *Pregnancy Hypertens*. 2018 Jan;11:136-41. <https://doi.org/10.1016/j.preghy.2017.10.008>
  14. Ando T, Kaur R, Holmes AA, Brusati A, Fujikura K, Taub CC. Physiological adaptation of the left ventricle during the second and third trimesters of a healthy pregnancy: a speckle tracking echocardiography study. *Am J Cardiovasc Dis*. 2015 Aug 1;5(2):119-26. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4539098/>
  15. Cong J, Fan T, Yang X, Squires JW, Cheng G, Zhang L, et al. Structural and functional changes in maternal left ventricle during pregnancy: a three-dimensional speckle-tracking echocardiography study. *Cardiovasc Ultrasound*. 2015 Jan 27;13:6. <https://doi.org/10.1186/1476-7120-13-6>
  16. Bortnick AE, Lama von Buchwald C, Hasani A, Liu C, Berkowitz JL, Vega S, et al. Persistence of abnormal global longitudinal strain in women with peripartum cardiomyopathy. *Echocardiography*. 2021;38(6):885-91. <https://doi.org/10.1111/echo.15071>

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