ORIGINAL ARTICLE

Maternal Cardiovascular Hemodynamics in Preeclampsia: Echocardiographic Assessment and Evaluation of Subclinical Left Ventricular Dysfunction

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ABSTRACT

Background: Preeclampsia is a disorder of pregnancy having an impact on multiple organ systems. Cardiac manifestations in preeclampsia are varied which can be easily studied by echocardiography. With the advancement in the field of strain imaging, we can also analyze and predict the subclinical left ventricular dysfunction in these patients which may help us to assess high-risk groups.

Objective: To compare maternal cardiovascular hemodynamics and function with emphasis on diastolic parameters and strain imaging using echocardiography in normal pregnant women with those having preeclampsia in the third trimester of pregnancy.

Methods: Thirty preeclamptic women and 30 women with a normal singleton pregnancy of 34 weeks gestation were compared for systolic and diastolic parameters on echocardiography. Strain imaging was done to find the global and circumferential strain.

Results: Blood pressure and total vascular resistance were significantly more in preeclamptic subjects than control. Women with preeclampsia had more diastolic dysfunction than those having a normal pregnancy. The global longitudinal strain was less in the preeclamptic group (−15.63 ± 1.69) than control (−20.86 ± 1.52). Similarly, the circumferential strain was lower in the preeclamptic group (−16.93 ± 1.11) than control (−21.76 ± 1.25).

Conclusion: Preeclamptic women have a significant difference in systolic and diastolic dysfunction as compared to pregnant females having normal blood pressure. Early assessment of these parameters may help to identify the high-risk groups. Knowledge of these changes may help us to understand better the pathophysiological basis of disease which may further help in managing these patients.

Keywords: Echocardiography, Left ventricular dysfunction, Preeclampsia.

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BACKGROUND

Preeclampsia is a disorder of pregnancy characterized by new-onset hypertension, i.e., systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg developing after 20 weeks of gestation with the involvement of at least one organ system which completely resolves within 12 weeks postpartum.[1-4] It remains one of the major causes of maternal morbidity and mortality and affects 5–8% of pregnant patients.[5] The recent development in understanding the pathophysiology of preeclampsia especially the endothelial dysfunction has invoked interest in the pathological basis of its multiorgan involvement. Cardiovascular manifestations due to preeclampsia are varied which may be either secondary to hypertension, endothelial dysfunction, subclinical left ventricular dysfunction or any other unidentified pathological mechanism.[6-11] In this observational study, we assessed the various cardiovascular changes in patients of preeclampsia with special reference to subclinical left ventricular dysfunction when compared with that of normal pregnancy.

SUBJECTS AND METHODS

This study was carried out in LPS Institute of Cardiology in collaboration with the Department of Obstetrics and Gynecology, GSVM, Kanpur. Women with 34 weeks gestational age having singleton pregnancy, not meeting the exclusion criteria, were enrolled in the study.
The exclusion criteria were:
- Gestational age less than 34 weeks or not sure of dates
- Known cardiac disease (e.g., structural heart disease coronary heart disease, cardiomyopathies, etc.)
- Chronic or gestational hypertension
- Renal impairment,
- Twin pregnancy and
- Diabetes mellitus

- Obesity
- Moderate to severe anemia.

Preeclampsia diagnosis was on the basis on the National High Blood Pressure Education Program Working Group definition, also endorsed by the American Congress of Obstetricians and Gynecologists (ACOG). Preeclampsia was defined as ≥140 mm Hg systolic or ≥90 mm Hg diastolic with proteinuria (>300 mg/24 hours) in a previously normotensive woman after 20 weeks of gestation.[2] Total of 30 preeclamptic patients was taken as cases which were echocardiographically assessed for various systolic and diastolic parameters and compared with that of the same number of females with normal pregnancy which served as control. Blood pressure was measured using a standard auscultatory method with the help of mercury sphygmomanometer. Blood pressure was measured in the sitting position in the right arm at the level of the heart.

Transthoracic echocardiogram was performed by single echocardiographer using a Philips IE33 system (Philips Medical Systems, Andover, MA, USA) and an S5-1 1.5/3.6 MHz transducer who was blinded to the patient's diagnosis.

Current guidelines of the American Society of Echocardiography were used to obtain Left ventricular (LV) measurements. Modified biplane Simpson's method was used to calculate LV volumes and LVEF.

LV stroke volume was calculated as the product of left ventricular (LV) outflow area and LV outflow tract VTI (velocity time integral) which was assessed by means of pulsed-wave Doppler positioned at the LV outflow tract. To assess diastolic parameters apical four chambers were used along with tissue Doppler imaging.

To obtain global longitudinal strain (GLS) three apical views were acquired at a frame rate of 40–80 MHz for three consecutive cardiac cycles and was stored digi-tally as raw data which then underwent post-processing analysis. QLAB 9 cardiac motion quantification (CMQ); Philips Medical Systems software was used for post-processing analysis. Three sampling points were placed manually at the septal, lateral mitral annulus and the apical endocardium for each of the apical views (two-chamber (2CH), 3CH, and 4CH). A region of interest was then generated by the software to cover the entire thick-ness along the LV myocardium which was then adjusted manually to provide optimal tracking. Avoiding prema-ture beats longitudinal 2D speckle-tracking strain values were obtained from one representative cycle. Segmental longitudinal strain values were based on the American Society of Echocardiography's 17-segment LV model. Average of regional strain (Figs 1 to 4) was calculated and was represented in a single bull's-eye summary. LV circumferential strain was assessed in parasternal short axis view.

SPSS software 19 statistical packages for Windows (Chicago. Inc) was used to analyze all statistical data. Continuous variables were expressed as mean ± standard deviation (Gaussian distribution) or range, and qualita-tive data were expressed as a percentage. Continuous variables between the two groups were compared using the unpaired t-test.

RESULTS

The baseline features such as the age of the patient, gesta-tional age, parity, BMI and race were compared between cases and control and they had statistically insignificant difference (Table 1).

Differences in systolic BP, diastolic BP mean arterial pressure (MAP) and TVR were higher in preeclamptic patients and had statistically significant difference with respect to control however left ventricular end-systolic
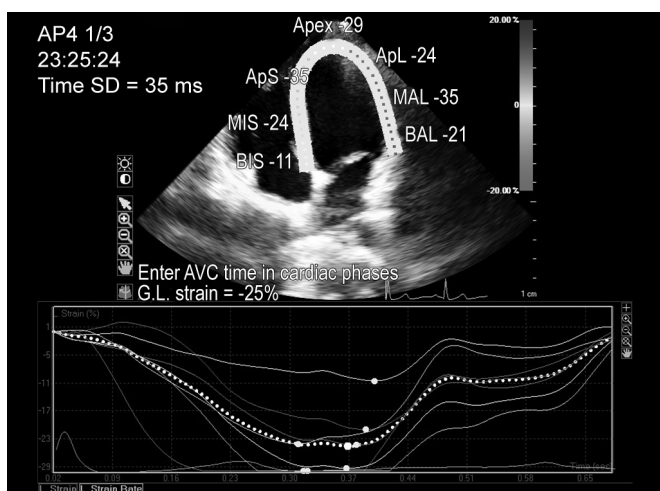

Fig. 1: 2D speckle tracking image showing longitudnal strain in normal pregnancy
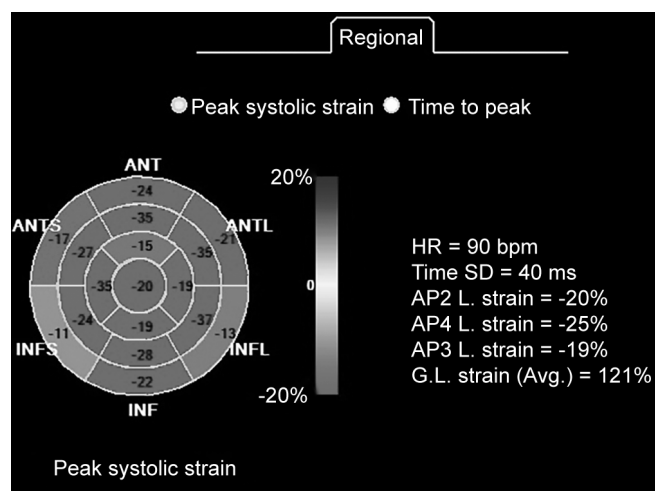

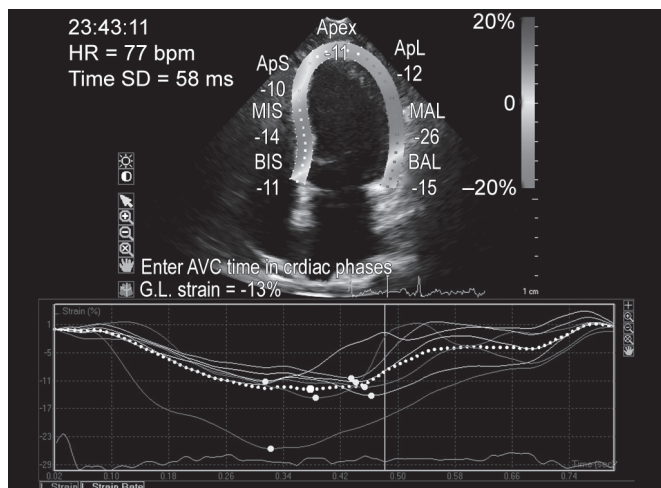**Fig. 2:** Bulls eye map showing global longitudnal strain in normal pregnancy

Fig. 3: 2D speckle tracking image showing longitudnal strain in preeclampsia
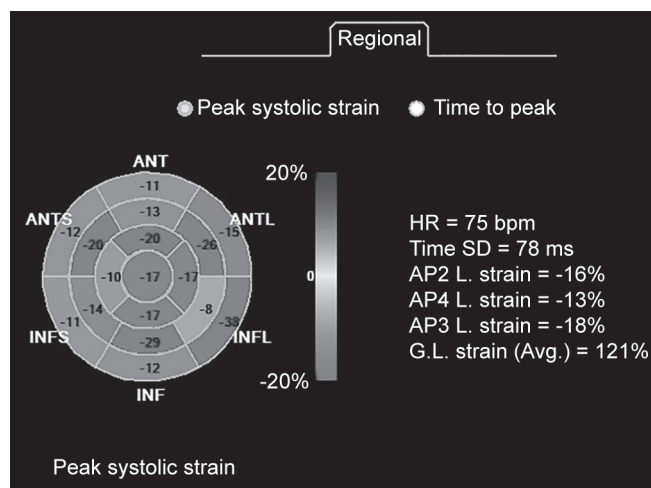

Fig. 4: Bulls eye map showing global longitudnal strain in preeclampsia

volume (LVESV), left ventricular end diastolic volume (LVEDV), stroke volume, cardiac output and ejection fraction showed statistically insignificant difference between two groups (Table 2).

Table 3 shows a comparison of diastolic parameters between preeclamptic subjects and control. Preeclamptic patients had more diastolic dysfunction as compared to control.

Patients with preeclampsia had more aortic, mitral and tricuspid valve regurgitation as compared with control. Subjects having global longitudinal strain less than 18% without obvious LV dysfunction on 2D echocardiography were said to have subclinical LV dysfunction. Subclinical left ventricular dysfunction was more in preeclamptic patients as compared to control which was assessed by LV strain imaging as a decrease in longitudinal and circumferential strain (Table 4 and Graph 1). Also, we found that systolic blood pressure had an inverse correlation with the global longitudinal strain (Graph 2).

DISCUSSION

Preeclamptic patients have varied hemodynamic presentation which depends on the severity of the disease, presence of comorbidities, use of medications, gestational age and left ventricular dysfunction.[12] Pathogenesis of preeclampsia is divided into two stages. The first stage is characterized by decrease placental perfusion due to inadequate development of maternal spiral arteries. This results in the release of vascular toxins and other anti-angiogenic factors which causes generalized endothelial dysfunction.[13] This widespread endothelial dysfunction results in hypertension, proteinuria, and edema. In our study, we found high total vascular resistance as compared to control which was similar to other studies by Valensise et al. and Poppas et al.[14,15] High TVR causes an increase in afterload which causes LV hypertrophy

Table 1: Baseline characteristics of cases (preeclamptic patients) and control (normal pregnancy)

Baseline characteristics	Cases	Control	p value
Age (years)	27.4 ± 3.8	27.1±4.3	NS
Parity	1.9 ± 1.0	1.8 ± 1.0	NS
Gestational age (weeks)	35 ± 2.8	35 ± 1.1	NS
BMI (kg/m^2)	24.6 ± 3.17	23.4 ± 4.14	NS
Smoking	Nil	Nil	Nil
Race/ethnicity			
Asian	30	30	Nil
White			
Black			
Others			

Table 2: Clinical and echocardiographic parameters of cases and control

	Cases	Control	p value
Heart rate (beats/min)	89 ± 4	90 ± 4	0.34
Systolic BP (mm of Hg)	158.53 ± 10.89	118.33 ± 7.06	<0.001
Diastolic BP (mm of Hg)	100.93 ± 6.59	71.33 ± 7.09	<0.001
Mean arterial pressure (mm of Hg)	120.14 ± 6.23	98.26 ± 5.34	<0.001
LVESV (mL)	34.67 ± 10.12	29.27 ± 7.25	0.17
LVEDV (mL)	105.23 ± 19.74	103.64 ± 11.46	0.62
Stroke volume (mL)	71.3 ± 6.19	74.2 ± 4.22	0.34
Cardiac output (mL/min)	6319 ± 1110	6660±1213	0.10
TVR (dynes/cm^5)	1568.74 ± 156.2	1123.67 ± 86.38	0.02
EF (%)	55.83 ± 2.74	57.38 ± 2.33	0.45

Table 3: Diastolic parameters between cases and control

| Doppler parameters	Cases	Control	p value
E wave	1.03 ± 0.17	0.66 ± 0.13	0.0001
A wave	0.77 ± 0.29	0.51 ± 0.17	0.0001
E/A Ratio	1.497 ± 0.492	1.35 ± 0.224	0.3
Septal e̍ (cm/seconds)	9.97 ± 1.75	11.29±1.43	0.01
Lateral e' (cm/seconds)	11.93 ± 2.41	13.75±2.09	0.01
IVRT (milliseconds)	95.91 ± 8.73	82.47 ± 6.7	0.03
DT (milliseconds)	190.27 ± 36.73	124 ± 17.67	0.02

Table 4: Global longitudinal and circumferential strain along with valvular regurgitation in cases and control

|	Cases	Control	p value
Global longitudinal strain (%)	−15.63 ± 1.69	−20.86 ± 1.52	<0.001
Circumferential strain (%)	-16.93 ± 1.11	−21.76 ± 1.25	<0.001
Mitral regurgitation			<0.01
Mild	9	2	
Moderate	2	0	
Severe	0	0	
Tricuspid regurgitation			
Mild	7	2	
Moderate	1	0	
Severe	0	0	
Aortic regurgitation			
Mild	7	0	
Moderate	0	0	
Severe	0	0	

as well as an increase in filling pressure leading to diastolic dysfunction. Our study was consistent with various other studies of preeclamptic patients in which diastolic dysfunction has been reported in 30–50% of patients.[16] In our study we found preeclamptic patients having lower cardiac output as compared to control. LV dysfunction either overt or subclinical is seen as associated with preeclampsia.[17-21] Severe LV dysfunction in the form of peripartum cardiomyopathy is more common in preeclamptics.[22] The pathophysiological basis of LV dysfunction in PPCM has been related to various anti-angiogenic factors. A soluble version of the vascular endothelial growth factor receptor 1 (sVEGFR1), the most important of such factors are elevated in preeclampsia.[22,23] Though in our study we did not find any preeclamptic patient with overt LV dysfunction, the subclinical LV dysfunction might be responsible for low cardiac output.

Advances in the field of strain imaging by echocardiography as also helped to evaluate subclinical left ventricular systolic and diastolic functions. In diastolic dysfunction attenuation of early longitudinal muscle relaxation can be observed a decrease in longitudinal strain while in the setting of a normal ejection fraction

changes observed in radial and circumferential strain likely represent subclinical dysfunction. The subclinical LV dysfunction may also develop due to subendocardial microvascular ischemia and fibrosis due to increased end-systolic wall stress from an increased afterload.[17] In our study, we found a decrease in global longitudinal strain in preeclamptics as compared to control. The normal value of global longitudinal strain as assessed by echocardiographic techniques is usually greater than 18%.[21] Persons having global longitudinal strain less than 18% without obvious LV dysfunction on 2D echocardiography were said to have subclinical LV dysfunction. Our result was similar to the study done by Sahul et al. in which the values for longitudinal and
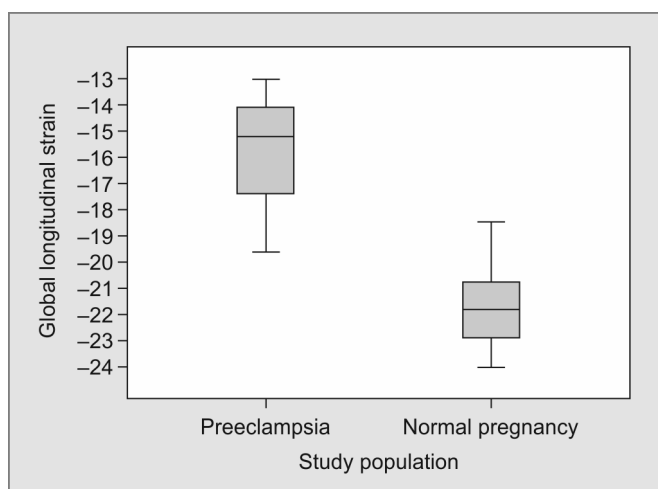
Graph 1: Box plot showing global longitudinal strain in normal pregnancy and preeclampsia
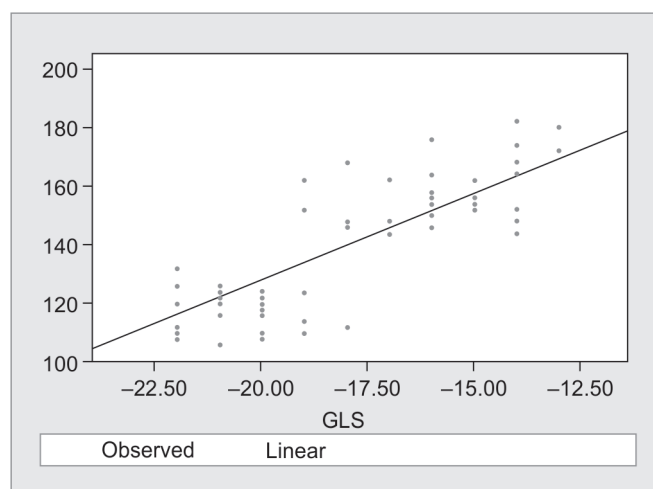
Graph 2: Dot plot showing inverse relation of blood pressure to global longitudinal strain

circumferential strain were lower in the preeclamptic group as compared to control.[24] Valvular regurgitations were more common in preeclampsia than a normal pregnancy. This may be due to subclinical LV dysfunction, altered hemodynamics or in a few cases due to change in ventricular geometry.

CONCLUSION

With this study, we can infer that pregnancies complicated with preeclampsia have various structural, hemodynamic and functional changes in the cardiovascular system which we can very easily detect by echocardiography. Knowledge of these changes may help us to understand better the pathophysiological basis of disease which may further help in managing these patients.

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