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Echocardiographic Assessment of Cardiovascular Hemodynamics in Pre-eclampsia and Normal Pregnancy

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Abstract

Background: To assess maternal cardiovascular function using echocardiography in normal and preeclamptic women in the third trimester of pregnancy. **Methods**: 100 subjects of which 50 with preeclampsia, not on medication and 50 normotensive women with >34 weeks gestation with singleton pregnancy were recruited. baseline characterstics were studied with systolic and diastolic parameters on echocardiography. **Results:** The following parameters were significantly higher in preeclamptic subjects as compared to normotensive controls- mean cardiac output (7.69±0.33 vs 6.39±0.31l/min); mean LV mass (144.62 ±9.12 vs 116.92±5.15g); total vascular resistance (1224.44±73.59 vs 1073.6±54.85dyne/sec/cm5.). mean Ewave (0.99±0.12 vs 0.65±0.80 m/sec); mean A wave(0.64±0.12 vs 0.51±0.007m/sec);meanIVRT (96.59±4.76 vs 82.71±3.38ms) Student 't' test was used as a test of significance. **Conclusions:** Women with preeclampsia have significant systolic and diastolic dysfunction compared to normotensive controls. Blood pressure monitoring alone is insufficient to identify risk of cardiovascular complications effectively in these subjects.

Keywords:- Echocardiography, Maternal, Diastolic, Systolic, Normal, Preeclampsia

INTRODUCTION

Pregnancy is a physiological process. To supply adequate nutrition to the growing fetus, maternal physiological adjustments of different organ systems occur in pregnancy. The adjustments are circulatory, metabolic and hormonal.

Pre-eclampsia is a multisystem disease unique to human pregnancy characterised by hypertension and organ system derangement. The disease is complicating 5-8% of the pregnancies and remains in the top three causes of maternal morbidity and mortality globally. It is the leading cause of fetal growth restriction, intrauterine fetal demise and preterm birth. These morbidity and mortality implications are most marked in poor, underprivileged, remote and rural communities and indigenous populations (WHO 2004; WHO 2007a; WHO 2007b).[1] It is estimated that approximately 50,000 women die each year of preeclampsia worldwide and approximately 300,000 babies die predominantly due to premature birth in

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women with preeclampsia. The hemodynamic studies in preeclampsia are confusing and difficult to interpret because of variations in the severity of the disease, mix of treated and untreated patients, invasive and noninvasive methods of evaluation and small sample size.[2] Echocardiography, is a non-invasive, precise device and is validated in pregnancy. It is an ideal device for measuring the cardiac function in women with preeclampsia.[3]

MATERIAL AND METHODS

The study was conducted on antenatal patients admitted in department of Obstetrics and Gynaecology at Bebe Nanki Mother and Child Care Centre attached to Govt. Medical College, Amritsar. For this study a total of 100 women were taken, divided into two groups of 50 normotensive as control group (Group I) and 50women with preeclampsia, untreated >34 weeks gestation and singleton pregnancy (Group II) were evaluated for baseline characteristics with systolic and diastolic parameters on echocardiography.

Patients were evaluated by detailed history, clinical examination and relevant investigations.

1. A quiet, temperature-controlled room and the women were allowed 15min of rest before measurements. Blood pressure was measured in the right arm with sphygmomanometer followed by echocardiography. Philips iE33 XMATRIX with a versatile X5-1 transducer machine was used to perform echocardiography and the following parameters were studied: Systolic parameters □ left ventricle end systolic volume (LV ESV).
2. Stroke volume (SV).
3. Cardiac output (CO).
4. Aortic root diameter (ARD).
5. Left ventricular outflow tract (LVOT).
6. Left ventricular mass (LVM).
7. Diastolic parameters – Ewave, A-wave, E/A ratio, isovolumetric relaxation time (IVRT).
8. E deceleration time (DtE), E wave velocity time integral (EVTI).
9. A wave velocity time integral (A VTI).

Inclusion Criteria:

- Pregnancy >34 weeks Singleton pregnancy

Exclusion criteria:

- Gestational age <34 weeks or unsure of dates.
- Chronic hypertension (excluded by history and presentation). Heart Disease.
- Medical Disorder, Diabetes mellitus and Renal Disease. Moderate or severe anaemia.
- Twin pregnancy, Alcohol and Tobacco use.

RESULTS

[Table 1] shows the baseline features of the study population. The mean systolic blood pressure of the subjects with preeclampsia was (152.7±11.63mmHg) which was higher as compared to (112.48±6.37mmHg) in normotensive. Similarly the mean diastolic blood pressure of the subjects with preeclampsia was (99.6±7.32mmHg) which was higher as compared to (72.12±2.7mmHg) in normotensive patients. The mean arterial pressure of the subjects with preeclampsia was (117.58±7.77mmHg) as compared to (85.69±2.59mmHg) in normotensive. This observation was statistically significant at $p<0.001$.

[Table 2] shows comparison of systolic parameters between two study groups. The mean LV ESV in the preeclamptic group was higher (71.36±4.03ml) as compared to (42.05±2.86ml) in normotensive. The mean LV

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EDV in the preeclampsia group was higher (174.41±6.23ml) as compared to (125.88±4.68ml) in normotensive. Cardiac output in the preeclampsia group was (7.68±0.33L/min) as compared to (6.39±0.31L/min) in normotensive group. The observation was statistically significant at P < 0.0001. Mean TVR in preeclampsia group was higher at (1224.45±73.59 dyne/ sec/ cm5) as compared to (1073.60±54.85 dyne/ sec/ cm5) in normotensive group. Mean LVM in preeclampsia group was (144.62+9.118g) as compared to (116.920+ 5.146g) in normotensive group and similarly the aortic root diameter was (3.158+ 0.253cm) as compared to (2.2174+ 0.211cm) which was statistically significant.

[Table 3] shows comparison of diastolic parameters between normotensive and preeclampsia subjects. Mean E wave in preeclampsia group was (0.990±0.124 m/sec) as compared to (0.6463±0.802m/sec) in normotensive group. A wave in preeclampsia group was (0.637±0.112m/sec) as compared to (0.507±0.074m/sec) in normotensive group. E deceleration time in preeclampsia group was (173.26±22.73m/sec) as compared to (124.2±4.99m/sec) in normotensive group. IVRT in preeclampsia group was (96.5 ±4.76ms) as compared to (82.71±3.38ms) in normotensive group. E VTI in preeclampsia group was (13.90±2.13ms) as compared to (11.97±1.08ms) in normotensive study group. A VTI in preeclampsia group was (6.414±0.486cm) as compared to (2.644±0.38cm).

Table 1: Comparative Study of Age, Blood Pressure and Mean Arterial Pressure of Normotensive And Preeclampsia.

	No of patients	**Mean**	**Std. Deviation**
Age(yrs)			
• Normotensive	50	22.68	2.41
• Pre-eclampsia	50	23.86	3.83^{NS}
• Total	100	23.27	3.24
Systolic Blood Pressure(mm Hg)			
• Normotensive	50	112.48	6.37
• Pre-eclampsia	50	152.72	11.63^{S}
• Total	100	132.60	22.27
Diastolic Blood Pressure(mm Hg)			
• Normotensive	50	72.12	2.72
• Pre-eclampsia	50	99.60	7.33^{S}
• Total	100	85.86	14.86
Mean Arterial Pressure(mm Hg)			
• Normotensive	50	85.69	2.59
• Pre-eclampsia	50	117.58	7.78^{S}
• Total	100	101.64	17.03

S=Significant ($p<0.001$); NS = Non Significant ($p>0.05$)

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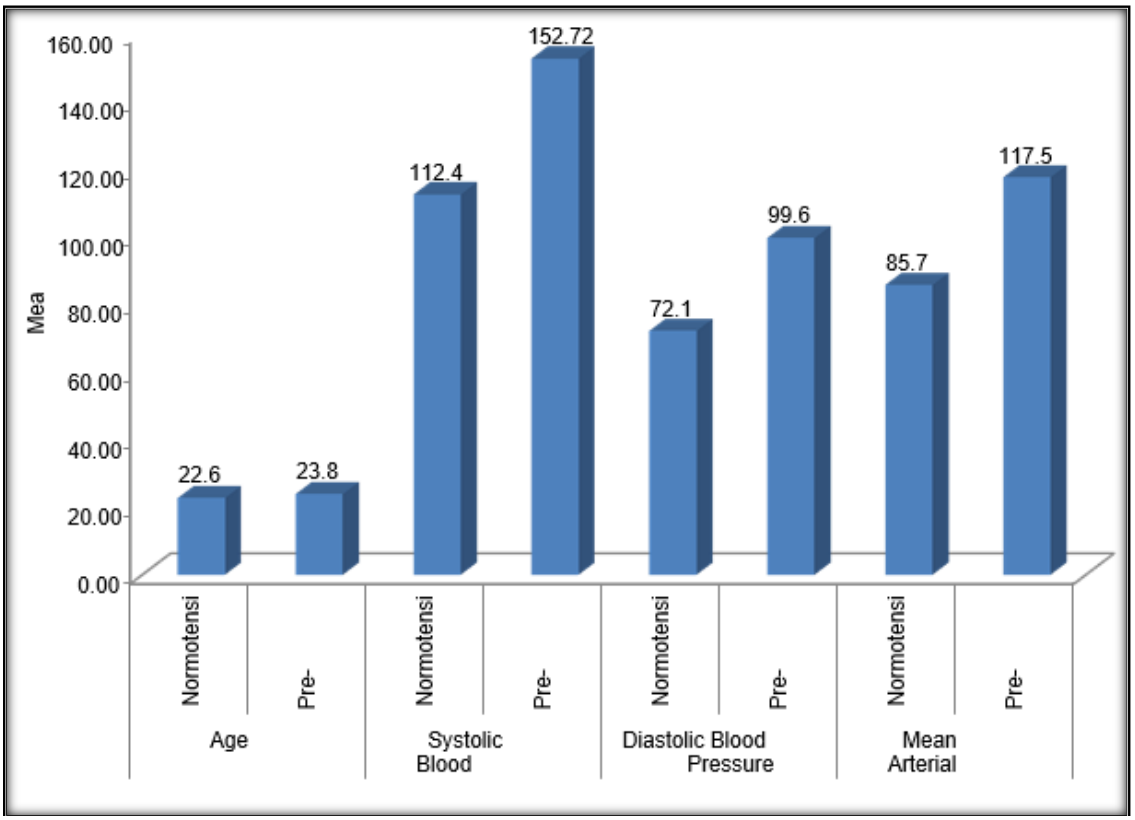

Table 2: Comparative Study of Systolic Parameters

	No of patients	Mean±SD
Left Ventricular End Systolic Volume (LVESV)(ml)		
• Normotensive	50	42.05±2.86
• Pre-eclampsia	50	71.36±4.03
• Total	100	56.70±15.13
Left Ventricular end Diastolic Volume (LVEDV)(ml)		
• Normotensive	50	125.88±4.68
• Pre-eclampsia	50	174.40±6.22
• Total	100	150.14±24.99
Stroke Volume (SV)(ml)		
• Normotensive	50	83.82±4.65
• Pre-eclampsia	50	103.09±2.91
• Total	100	93.46±10.42
Cardiac Output (CO)(L/min)		
• Normotensive	50	6.39±0.31
• Pre-eclampsia	50	7.69±0.33
• Total	100	7.04±.72
Left Ventricular Mass (LVM)(g)		

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• Normotensive	50	116.92±5.15
• Pre-eclampsia	50	144.62±9.12
• Total	100	130.77±15.75
Aortic Root Diameter (ARD)(cm)		
• Normotensive	50	2.22±0.21
• Pre-eclampsia	50	3.16±0.25
• Total	100	2.69±0.53
Left Ventricular Outflow Tract (LVOT)(cm)		
• Normotensive	50	1.59±0.08
• Pre-eclampsia	50	1.83±0.14
• Total	100	1.71±0.17
Total Vascular Resistance (TVR) (dyne/sec/cm5)		
• Normotensive	50	1073.6±54.85
• Pre-eclampsia	50	1224.44±73.59
• Total	100	1149.02±99.58

S=Significant (p<0.0001)

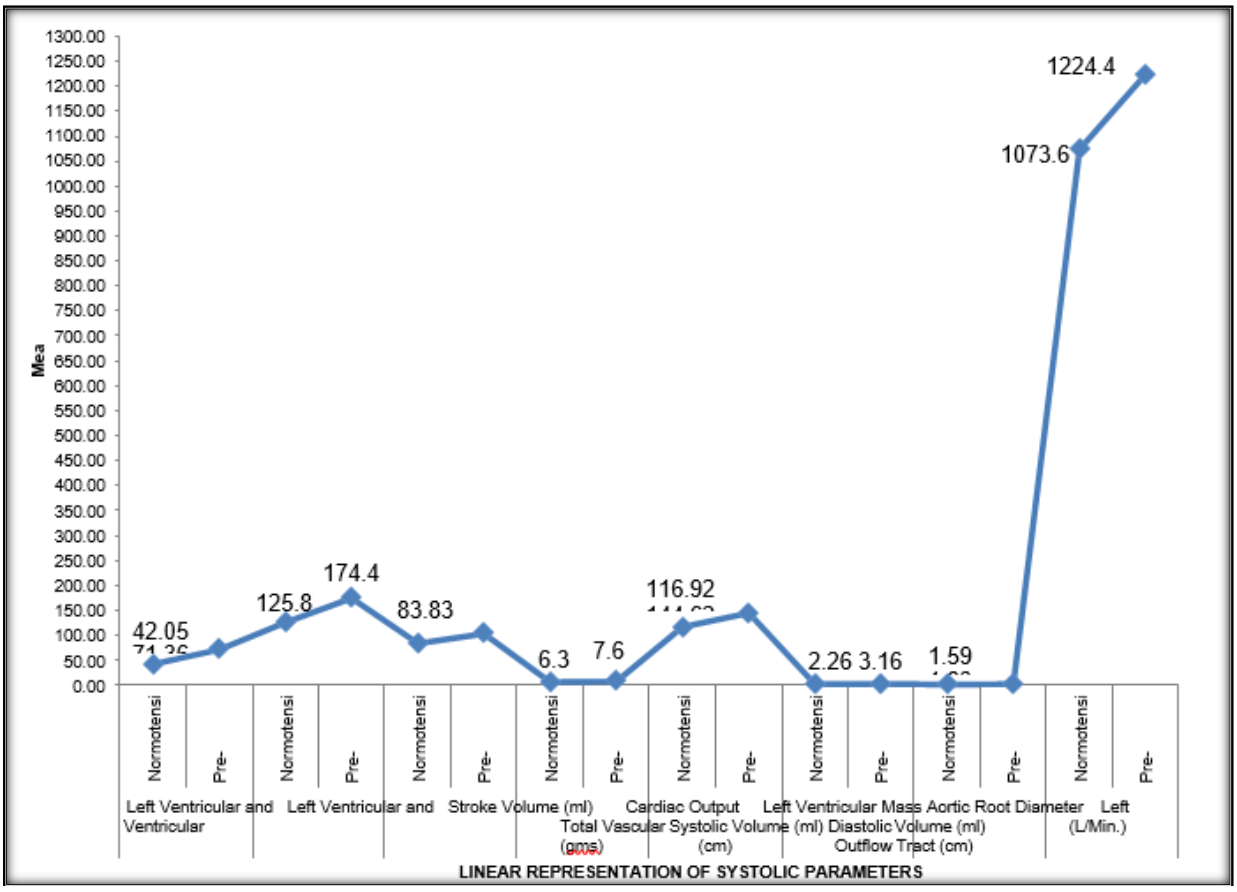

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Table 3: Comparative Study of Diastolic Parameters

E-Wave(m/s)			No of patients	Mean±S.D.
• Normotensive			50	0.65±0.80
• Pre-eclampsia			50	0.99±0.12
• Total			100	0.82±0.20
A-Wave(m/s)				
• Normotensive			50	0.51±0.07
• Pre-eclampsia			50	0.64±0.12
• Total			100	0.57±0.12
E/A-Ratio				
• Normotensive			50	1.28±0.07
• Pre-eclampsia			50	1.57±0.18
• Total			100	1.42±0.20
E Deceleration time(ms)				
• Normotensive			50	124.20±4.99
• Pre-eclampsia			50	173.26±22.72
• Total			100	148.73±29.59
Isovolumetric Relaxation Time (IVRT)(ms)				
• Normotensive			50	82.71±3.38
• Pre-eclampsia			50	96.59±4.76
• Total			100	89.65±8.10
E-Wave Velocity (E-VTI)(ms)	Integral	Time		
• Normotensive			50	11.97±1.08
• Pre-eclampsia			50	13.90±2.13
• Total			100	12.94±1.94
A-Wave Velocity (A-VTI)(ms)	Integral	Time		
• Normotensive			50	2.64±0.38
• Pre-eclampsia			50	6.41±0.49
• Total			100	4.52±1.94

S=Significant ($p<0.0001$)

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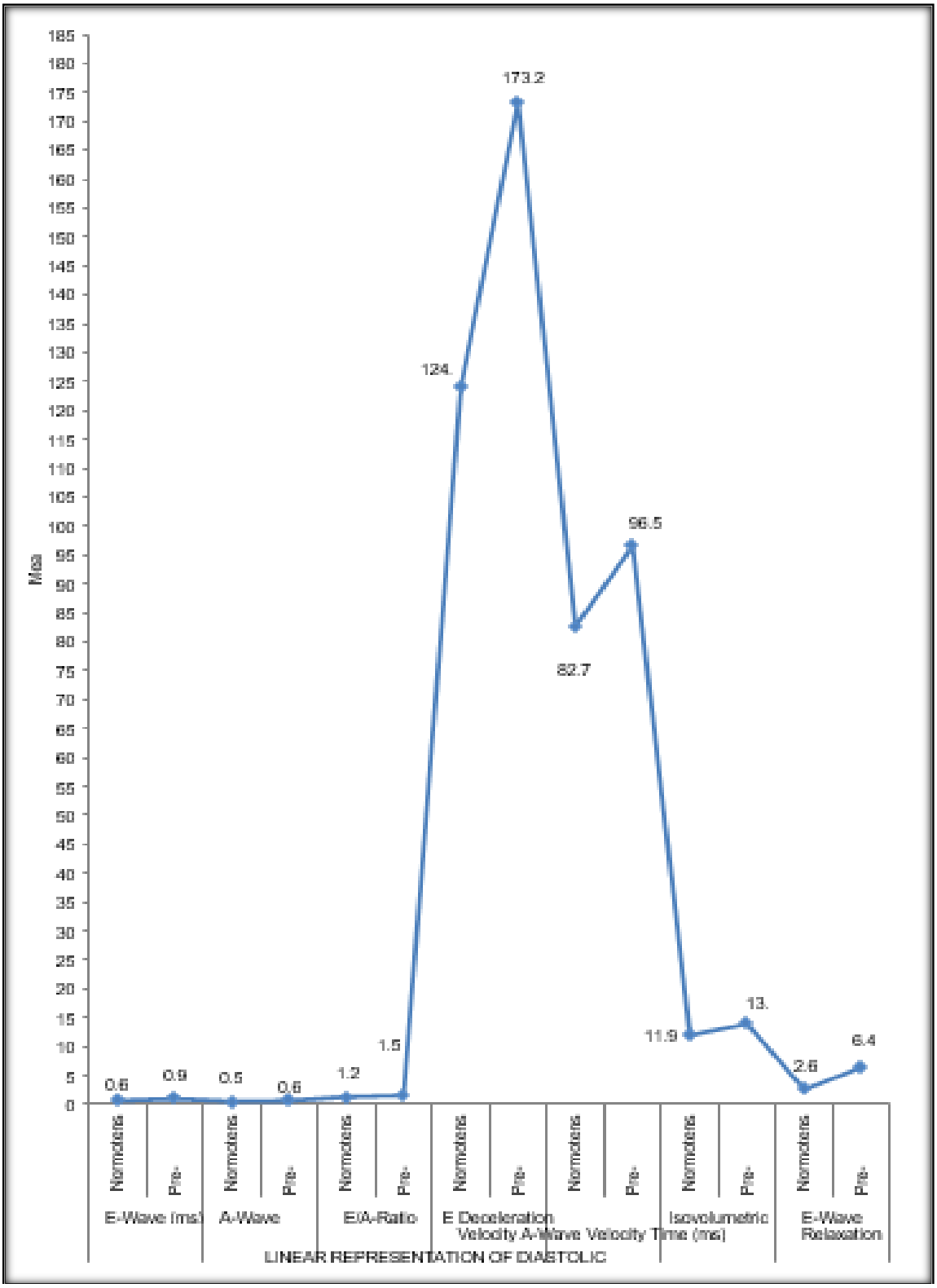

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DISCUSSION

In our study, it was found that the mean LV ESV in normotensive women was (42.05±2.86ml) as compared to pre-eclamptic women (71.36±4.03ml). The elevated end systolic volume suggests that the elevated end systolic pressure is generated by increased afterload. Our results were comparable with a study carried out by Solanki et al.[4] who assessed LVESV in pre-eclamptics and normotensives and found that mean LVESV was higher in the PIH group as compared to the controls (36.04±13.32 vs. 27.2±3.5 ml).

In our study it was found that the mean LV EDV in normotensive women was (125.88±4.651ml) as compared to (174.40 ±6.225 ml) in preeclampsia, which was comparable with the study done by Solanki et al,[4] who studied the same parameter in pre- eclamptics and normotensives and found a significant difference in LV EDV between the two groups (108.23±27.95 vs. 107.73±5.66 ml, 100±16 vs. 94±7 ml). Ghossein Doha et al,[5] also concluded that the mean LV EDV was higher in pre-eclamptic group, i.e. (100±16 vs. 94±7 ml).

In our study it was found that mean stroke volume in normotensive women was (83.82±4.65ml) as compared to (103.090±2.917ml) in preeclampsia. Our results were comparable to Dennis et al,[6] study who observed an increased stroke volume in untreated pre- eclamptic group, i.e. (58.9±12.8 vs. 56.9±7.2 ml). Solanki et al,[4] did a similar study and found that stroke volume in pre-eclampsia and normotensives was 73.3±14.19 versus 70.8±3.22 ml, respectively.

In our study it was found that the mean cardiac output in normotensive women was (6.39±0.309L/min) as compared to (7.686±0.326L/min) as compared to preeclampsia women. Our results were comparable to the study done by Dennis et al,[6] who found CO to be higher in the PIH group as compared to the controls (4789±14 vs. 4109±59 ml/min). As per study by Solanki et al,[4] CO in pre-eclamptics was 6600.85±4.56 ml/ min as compared to 5600.1±1.77 ml/min which was significantly higher.

In our study it was found that aortic root diameter in normotensive women was (2.217±0.211cm) as compared to (3.157±0.253cm) in preeclamptic women. Our results were comparable to the study done by Solanki et al,[4] in which women with pre-eclampsia had higher aortic root diameter as compared to normotensive control (2.48 vs. 2.02 cm). Naidoo et al also reported a higher mean ARD in pre-eclamptics as compared to the controls (24 vs. 23 mm).

In our study it was found that TVR in normotensive women was (1073.60±54.85 dyne/sec/cm5) as compared to (1224.446±73.589 dyne/sec/cm5) in preeclamptic women. High TVR in pre-eclampsia suggests elevated after load which is linked with reduced emptying of left ventricle. Solanki et al,[4] did a study and observed that mean TVR in the PIH group was 1396.85±156.2 dynes/ sec/cm - 5 as compared to the 1204.5±71.18 dynes/sec/ cm - 5. Dennis et al6 in his study found a significant difference in mean TVR in pre-eclamptics and controls. Mean TVR was 2015.7±624.7 and 1612.5±315.4 dynes/sec/ cm - 5 in the PIH group and normotensives, respectively. Similarly Neha et al,[7] found a significant difference in mean TVR in

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preeclampsia and normotensive which was (1389±57.04 vs 1286±45.01dyne/sec/cm5).

In our study among the diastolic parameters it was found that mean E wave in normotensive women was (0.646±0.802m/sec) as compared to (0.990±0.124m/sec) in preeclamptic women, which indicates that the pressure gradient across the mitral valve during early passive filling was higher. Our results were comparable to the study byb Shivananjiah C et al,[8] in which mean E wave in preeclamptic women was (0.98±0.14m/sec) as compared to (0.66±0.09m/sec) in normotensive women.

The mean A wave velocity in our study was (0.507 ±0.0738m/sec) in normotensive women as compared to (0.637±0.118m/sec) in preeclamptic women which reveals the significance of atrial systole. This was comparable to the study by Shivananjiah C et al8 in which the mean A wave velocity of preeclamptic women was (0.70±0.12m/sec) in comparison to the normotensive women (0.56±0.03m/sec). Our results were also comparable to the study by Solanki et al,[4] in which mean A wave velocity in normotensive women was (0.500±0.13ms) as compared to (0.775±0.278ms) in preeclamptic women.

The prolonged IVRT in preeclamptic patients was (96.59±4.76ms) as compared to (82.71±3.3ms) in normotensive women as left ventricle pressure takes greater time to fall below the atrial pressure compared to normotensive women. This was also comparable to the study by Solanki et al,[4] in which mean IVRT in normotensive women was (83.3±5.9ms) as compared to (96.13±9.13ms) in preeclamptic women.

Although our results are comparable to those given by different studies, still we feel there were few limitations in this study like it was not possible to follow up subjects in the postpartum period to examine whether the altered cardiovascular hemodynamic state reverts to normal after pregnancy. Similarly the study focused on left ventricular systolic and diastolic function and left ventricular structure, and as such did not specifically examine right heart function. The measurement of inferior vena caval size is difficult in pregnancy due to the gravid uterus and the inability of the women to lie supine for longer periods because of aortocaval compression.[9,10,11,12]

In normal pregnancy an increased preload and a decreased afterload favors an improved emptying of the left ventricle during systole and a reduction of the end systolic pressure. In preeclamptic women, the elevated after load is linked with a reduced emptying of the left ventricle and elevated end systolic pressure. This study demonstrate that hypertension in women with preeclampsia is predominantly caused by an increased cardiac output, with increased systemic vascular resistance. The cardiac output increase is due to an increase in end diastolic volumes subsequently leading to increase in stroke volume. There are physiological changes in left ventricular structure and function during normal pregnancy but exaggerated physiological changes are seen in preeclampsia subjects. This study suggests that women with preeclampsia had a uniform pattern of high resistance and high cardiac output.[13,14,15]

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CONCLUSIONS

Preeclampsia is associated with significantly higher prevalence of asymptomatic abnormal global left ventricular (LV) abnormal function/geometry and myocardial injury. It is also associated with significantly higher risk of subsequent heart failure, ischemic and hypertensive heart diseases, and related mortality compared with uneventful pregnancy in later life. Maternal echocardiography if introduced into routine management protocol could help to identify women who are at high risk to develop complications.

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