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Research Article

EFFICACY OF ECHOCARDIOGRAPHY IN PREDICTING GESTATIONAL HYPERTENSION OR PREECLAMPSIA

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A B S T R A C T

Background: Gestational hypertension or preeclampsia is difficult to predict till date. Currently no test has been validated by any regulatory body as predictor of gestational hypertension. Though a few markers have been identified but still many are yet to be revealed. Our study was done to find out the utility of maternal echocardiography in predicting gestational hypertension or preeclampsia

Materials and Methods: The study was done in the department of Obstetrics and gynaecology in collaboration with the department of cardiology at IPGME&R, Kolkata from June 2013 to May 2014 (one year period) Total 450 women were included in the study. Unfortunately 85 patients were lost during follow up. They were divided in two groups i.e. Study group (who developed gestational hypertension or preeclampsia) and control group (uncomplicated). Study group comprised of 75 patients and control group was having 290 patients. Echocardiography was done for all. Parameters of echocardiography were compared in between both groups.

Results: Preclinical phase of preeclampsia or gestational hypertension is associated with increase in the Left Ventricular mass, Left ventricular End Diastolic Volume, Stroke Volume, Cardiac Output, Left Atrial Diameter on echocardiography in the study group.

Conclusion: Maternal Echocardiography can be an effective tool in predicting preeclampsia.

INTRODUCTION

Preeclampsia or gestational hypertension is a multisystem disorder of unknown aetiology. Though different hypothesises in different time have been postulated but the exact cause is still under investigation. Incidence varies regionally and like 3% in USA[1] and up to 12 % in Bangladesh[2]. World Health Organisation has depicted that Asian region is accountable for 10% maternal mortality due to preeclampsia[3]. Microanalysis to find out the causes of unexplained intrauterine growth restriction indicates preeclampsia in about 25% cases[4]. Moreover it also accounts for 15% of preterm birth in developed countries worldwide[4]. Preeclampsia often complicated in various ways like development of Haemolysis, Elevated Liver Enzymes, Low Platelets (HELLP); Eclampsia. Early prediction and routine antenatal care can reduce the mortality rate up to 90%[5]. Prophylactic use of folic acid throughout the entire pregnancy is beneficial in high risk population[6]. Low dose aspirin (75mg/d) administration in pregnant women with risk of developing preeclampsia is effective to reduce incidence, IUGR if it is started before 16 weeks of gestational age[7]. Prediction of preeclampsia is very difficult task. Bilateral uterine artery notching in doppler study and pulsatility index (PI) more than 95th percentile in between 23 – 25 weeks of gestational age is assumed to be a risk of developing preeclampsia, gestational hypertension, and fetal growth restriction[8]. Nitric oxide synthase gene polymorphism (G894T, T-786C) is also associated with higher incidence of preeclampsia but with high false positive rate and high cost for analysis[9]. Elevated level of serum homocysteine, endothelin is also observed in pregnancies complicated by preeclampsia[10] but the laboratory standard plays a vital role in estimating these markers. In this background our study was done to find out the role of echocardiography in predicting gestational hypertension or preeclampsia in pregnant women in early second trimester.

MATERIALS AND METHOD

The study was done at IPGME&R, Kolkata at Obstetrics department in collaboration with Cardiology involving total 450 patients after taking proper informed consent in accordance with current ethical standard. The ethical permission was taken from the institutional ethical committee. It was a prospective cohort study. Inclusion criteria of the pregnant women were in between 12-16 weeks of gestational age, singleton pregnancy, age less than 40 years, BMI less than

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35kg/m^2. Patients with history of preeclampsia, diabetes mellitus, chronic hypertension, autoimmune diseases, SLE, family history of hypertension, multiple pregnancies were excluded from the study. The patients were selected by simple random technique fulfilling the inclusion criteria. After enrolment in the study all patients were interviewed as per a preformed proforma. Apart from the routine antenatal investigations they were underwent 2D & M mode Echocardiographic examination. The echocardiographic parameters were heart rate (beats/min), ejection fraction (%), stroke volume (ml/min), cardiac output (L/min), left atrial diameter (mm), left ventricular outflow tract diameter (mm), aortic root diameter (mm), left ventricular end diastolic volume (ml), left ventricular end systolic volume (ml) and left ventricular mass (gm). Cardiac output was calculated as (stroke volume × heart rate)/1000 ml. Mean arterial pressure was calculated by {(2×diastolic) + systolic}/3. Stroke volume was calculated as aortic valve area multiplied by aortic valve outflow in every minute. Estimation of left ventricular mass was done by using Devereux[11] formula.

LV mass (LVM) = 0.832 × [(LVDd + IVSd + PWd)^3 − LVDd^3] + 0.6

LVDd= Left ventricular end diastolic diameter, IVSd= Interventricular septum diameter, PWd= Posterior wall diameter.

All patients were followed up throughout the pregnancy in every four weeks. During follow up 85 patients were lost. Rest 365 patients remained in the study. Cases were defined as development of (i) Gestational hypertension i.e. blood pressure more than 140/90 mmHg on two separate occasion after 20 weeks of pregnancy (ii) Preeclampsia – gestational hypertension with significant proteinuria (iii) Proteinuria – excretion of protein more than 300 mg in 24 hours or dipstick test 1+ (iv) HELLP syndrome – haemolysis, elevated liver enzymes, low platelet count. Among the 365 patients 75 such patients we had found in our study that developed any of above mentioned conditions. They were subsequently defined as cases. The others 290 patients were control who remained uncomplicated throughout the pregnancy till delivery.

Statistical Analysis: Numerical variables were compared between both groups by Student's unpaired t-test. For paired comparisons the paired t-test was used. The Chi-square test or Fischer's exact test was used for intergroup comparison of the categorical variables. The analyses in this study were both sided and p<0.05 was considered significant statistically. The Software used for analysis were Statistica version 6 [Tulsa, Oklahoma: Stat Soft Inc.] and Graph Pad Prism version 5 [San Diego, California: Graph Pad Software Inc.].

RESULTS

Baseline demographic data as shown in the Table 1 mean age was 32.5 years in the study group and 33.7 years in the control group (p = 0.557). The mean height was 153.8 cm in study group and 152.7 cm in the control group (p= 0.259). BMI (Body mass index) (kg/m^2) 25.4 and 24.6 in the both group respectively (p= 0.546). The both groups contained comparable numbers of vegetarians i.e. 23 (30.7%) in study group and 91(31.4%) in control group (p= 0.457) and non-vegetarians i.e. 52(69.3%) and 199(68.6%) in both groups respectively (p= 0.559). Nulliparous were 46 (61.3 %) in the study group and 126 (43.4%) in the control group (p < 0.05) which was significantly higher in the study group. On the other hand multiparous were 164(56.6%) in the control and 29 (28.7%) in study group (p<0.05) which was significantly lower in the study group.

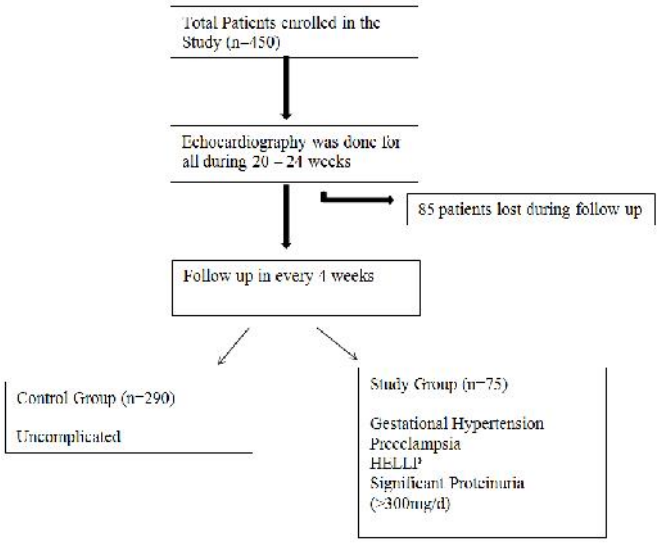

Fig 1 Study Protocol

Baseline haemoglobin and haematocrit values were 10.2g/dl & 35.2% in study group and 9.9 g/dl & 34.3% in control group respectively (p= 0.231, p= 0.043). All were normotensive at the time of recruitment i.e. blood pressure were 122.5 (110-128)/73.4 (66- 82)mm Hg and 121.8 (112-126)/72.5 (62 – 80) mm Hg in both groups respectively (p= 0.346, p=0.452). There were also no significant differences in the mean arterial pressure i.e. 89.8 (80 – 96) mm Hg and 88.9 (79 – 95) mmHg in both groups respectively (p=0.675).

Table 1 Baseline demographic characteristics (n=365)

Characteristic	Study Group (n=75)	Control Group (n=290)	p value
Age (years)	32.5 (20- 36)	33.7 (20 – 37)	0.557
Height (cm)	153.8(143 – 166)	152.7 (142 – 160)	0.259
Body Mass Index (BMI) (kg/m²)	25.4 (21.4 – 25.2)	24.6 (22.8 – 28.5)	0.546
Diet			
Vegetarian	23 (30.7%)	91 (31.4%)	0.457
Non vegetarian	52 (69.3%)	199 (68.6 %)	0.559
Parity			
Nullipara	46 (61.3 %)	126 (43.4%)	<0.05
Multipara	29 (28.7%)	164 (56.6%)	<0.05
Haemoglobin (g/dl)	10.2 (9.1 – 11.7)	9.9 (9.0 – 11.5)	0.231
Haematocrit (%)	35.2 (32- 39.5)	34.3 (31- 38.6)	0.043
Blood Pressure(mm Hg)			
Systolic	122.5 (110- 128)	121.8 (112-126)	0.346
Diastolic	73.4 (66- 82)	72.5 (62 – 80)	0.452
Mean Arterial Pressure (mm Hg)	89.8 (80 – 96)	88.9 (79 – 95)	0.675

Table 2 showing the echocardiographic parameters indicate no significant differences in heart rate (beats/min) 76 ± 8 and 74 ± 7, ejection fraction (%) 70.8 ± 5.2 and 71.6 ± 4.8, left ventricular end systolic volume (ml) 26.8 ± 4 and 27 ± 3.2, left ventricular outflow tract diameter (mm) 19.6 ± 0.7 and 19.2 ± 0.5, aortic root diameter (mm) 28.5 ± 0.6 and 27.9 ± 0.5 in both study and control groups respectively and no significant differences were found. Left ventricular mass (gm.) 166.7 ± 5.6 and 138 ± 7.2, left ventricular end diastolic volume (ml) 110 ± 5.5 and 86.7 ± 4.5, stroke volume (ml/min) 78 ± 10 and 66 ± 9, cardiac output (L/min) 6.8 ± 1.3 and 5.1 ± 0.8, left atrial diameter (mm) 37.8 ± 3.2 and 32.5 ± 2.9 in both groups

respectively and all these parameters were observed significantly higher in the study group ($p < 0.05$).

Table 3 Echocardiographic Parameters in both groups (n=365)

Parameters	Study group (n=75)	Control group (n=290)	p value
Heart rate (beats/min)	76 ± 8	74 ± 7	NS
Ejection fraction (%)	70.8 ± 5.2	71.6 ± 4.8	NS
Stroke Volume (ml/min)	78 ± 10	66 ± 9	< 0.05
Cardiac Output (L/min)	6.8 ± 1.3	5.1 ± 0.8	< 0.05
Left Atrial Diameter (mm)	37.8 ± 3.2	32.5 ± 2.9	< 0.05
Left Ventricular Outflow tract diameter (mm)	19.6 ± 0.7	19.2 ± 0.5	NS
Aortic root diameter (mm)	28.5 ± 0.6	27.9 ± 0.5	NS
Left ventricular End Diastolic Volume (ml)	110 ± 5.5	86.7 ± 4.5	< 0.05
Left Ventricular End Systolic Volume (ml)	26.8 ± 4	27 ± 3.2	NS
Left Ventricular mass (gm)	166.7 ± 5.6	138 ± 7.2	< 0.05

DISCUSSION

Hypertensive disorders of pregnancy are the leading cause of death in the developed countries and also one of the important causes worldwide. Though the global incidence is about 2%[12] but it is quiet higher in India. Eclampsia is one of the dreaded complications of preeclampsia which contributes increased maternal and neonatal mortality as well as morbidity. Preterm labour, fetal growth restriction may also occur due to preeclampsia. These are currently identified as greater obstetrical syndrome[13]. Effective treatment and use of magnesium sulphate has reduced the mortality rate due to eclampsia and severe preeclampsia. Early prediction can reduce the complication and helps to continue the pregnancy more which is beneficial in reducing neonatal morbidity and mortality[14]. Use of low dose aspirin (75 mg /day) throughout pregnancy if started before 16 weeks in high risk population is effective in reducing the risk of preeclampsia by 21%[15]. But currently early prediction of preeclampsia is under investigation. Uterine artery Doppler study is an effective tool in this regard but in between 23 – 25 weeks of gestational age it can predict, not before that[8]. Serum markers like angiotensin, nitric oxide, endothelin, lectin, adiponectin, soluble fms like tyrosine kinase (sFlt -1) etc. are non-specific. Nulliparity is one of the risk factors of pregnancy induced hypertension[16] which was observed in our study.

Increased inflammatory markers, immunological maladaptation, some genetic polymorphism in preeclampsia may cause damage to the different end organs like liver, kidney, brain, heart[17]. Impairment of left ventricular properties along with vascular reactivity occurs before development of clinical preeclampsia. It causes the changes in the hemodynamic indices[18]. Asymmetric hypertrophy of left ventricle and basal septum is characteristic of preeclampsia[19]. Preeclampsia is also associated with left ventricular diastolic dysfunction in 30% cases[20]. This diastolic impairment is associated with increased afterload. On the other hand activation of renin angiotensin system to maintain the glomerular filtration rate stimulates heart rate as well as cardiac outpur[21]. Our study also found high cardiac output in the study group. Left ventricular end diastolic volume was also higher in the study group due to the associated diastolic dysfunction[20].

Increased left ventricular mass in the risk group before development of preeclampsia occurs due to cardiac remodelling. Previously the cardiac burden of preeclampsia was underestimated in different studies. Due to the physiological changes during pregnancy ventricular hypertrophy and increase in cardiac diameter, thereby increase in cardiac mass[22]. This ventricular hypertrophy is a physiological response which normalizes within three to four weeks in postpartum period[22]. Subclinical period of preeclampsia is associated with increase in the left ventricular mass more compared with normal pregnancy[22]. Increase in the cardiac load is compensated by cardiac myocyte growth. Thereby it changes the architecture of the collagen tissue and increases the mass of connective tissue and ground substance which mainly harbouring collagen. It is responsible for the cardiac stiffness due to such remodelling and ultimately compromises the mechanical efficacy of the heart [23].

CONCLUSION

Preclinical phase of preeclampsia or gestational hypertension is associated with increase in the Left Ventricular mass, Left ventricular End Diastolic Volume, Stroke Volume, Cardiac Output, Left Atrial Diameter on echocardiography. It can be an effective tool for predicting pregnancy induced hypertension. More RCTs with involving more patients are required to establish the hypothesis.

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