A PROSPECTIVE COMPARATIVE STUDY OF SIGNIFICANCE OF TWO-DIMENSIONAL ECHOCARDIOGRAPHY IN HYPERTENSIVE DISORDERS OF PREGNANCY(HDP) AND NORMAL PREGNANCY AND ITS EFFECT ON FETOMATERNAL OUTCOME

A Dissertation submitted by

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Dissertation submitted to

BLDE (Deemed to be University) Vijayapura, Karnataka

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY In

OBSTETRICS AND GYNAECOLOGY

Under the guidance of

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IN OBSTETRICS AND GYNAECOLOGY

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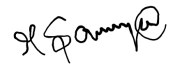

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ABBREVATIONS

ABBREVATION	EXPANSION
HDP	Hypertensive Disorders Of Pregnancy
ICU	Intensive Care Unit
ECHO	Echocardiogram
PE	Pre-Eclampsia
APLA	Antiphospholipid Antibodies
IL	Interleukin
TGF-β	Transforming Growth Factor Beta
FAS	Fetal Alcohol Syndrome
HIF-1	Hypoxia-Inducible Factors
CVS	Chorionic Villus Sampling
MG	Microgram
LDH	Lactate Dehydrogenase
MRI	Magnetic Resonance Imaging
PIH	Pregnancy Induced Hypertension
DBP	Diastolic Blood Pressure
SBP	Systolic Blood Pressure
EVT	Extrauterine Vein Invasion
RAS	Renin-Angiotensin System
VEGF	Vascular Endothelial Growth Factor
PAPP-A	Pregnancy-Associated Plasma Protein
PP	Placental Protein
ACE-I,	Angiotensin-Converting Enzyme
INR	International Normalized Ratio
APTT	Activated Partial Thromboplastin Time
DIC	Disseminated Intravascular Coagulation
NICE	National Institute Of Health And Clinical Excellence

BUN	Blood Urea Nitrogen
ALT	Alanine Transaminase
AST	Aspartate Transaminase
TEE	Transesophageal Echocardiography
2D	Two-Dimensional
3D	Three-Dimensional
LVEF	Left Ventricular Ejection Fraction
SV	Stroke Volume
ACOG	American College Of Obstetricians And Gynecologists
PVR	Peripheral Vascular Resistance
FHR	Fetal Heart Rate
RDS	Respiratory Distress Syndrome
FGR	Fetal Growth Restriction
ATN	Acute Tubular Necrosis
AFP	Alpha Fetoprotein
PLGF	Placental Growth Factor
sFlt-1	Soluble Fms-Like Tyrosine Kinase
CLASP	Collaborative Low Dose Aspirin
TTE	Two Transthoracic Echocardiographic
GDM	Gestational Diabetes Mellitus
CCF	Congestive Cardiac Failure
NICU	Neonatal Intensive Care Unit
TAPSE	Tricuspid Annular Plane Systolic Excursion
TRV	Tricuspid Regurgitation Jet Velocity
LSCS	Lower Segment Cesarean Section
PPH	Postpartum Haemorrhage
IUD	Intrauterine Device
CO	Cardiac Output
HR	Heart Rate

MAP	Mean Arterial Pressure
LV ESV	Left Ventricular End Systolic Volume
LV EDV	Left Ventricular End Diastolic Volume
CS	Caesarean Section
SMM	Severe Maternal Morbidity
NB	Newborns
NT-pro BNP	N-terminal pro B-type natriuretic peptide

ABSTRACT

Background: This study was conducted to study the echocardiographic changes in pregnant women with HDPs compared to normal pregnant women and also study the correlation between Echocardiographic abnormalities and fetal and maternal outcomes of pregnant women with HDPs and compare this with normal pregnant women.

Methods: This prospective observational study was conducted during August 2022-2024, in 142 women between age group 18 -35yrs with HDPs (Group A) compared with normal singleton pregnancy (Group B) admitted in Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapura, Karnataka, India.

Results: The findings in women with Hypertensive disorders of Pregnancy demonstrated higher prevalence of Abnormal 2D ECHO findings as compared to normotensive pregnant women. The mean percentage of Ejection Fraction in Group A was significantly lesser (61.52 ± 5.13) as compared to Group B (65.59 ± 4.49); the mean E Wave (0.82 ± 0.45), mean A Wave (0.59 ± 0.35) in women of Group A were significantly higher than that in Group B. The mean Septal e' in Group A was lesser (0.119 ± 0.163) as compared to Group B (0.156 ± 0.133) ($p<0.001$.) This study also showed significantly higher poor feto-maternal outcomes such as Respiratory Fetal Distress, higher neonatal ICU admissions, Fetal growth retardation and low birth weight and Maternal Outcome like higher need for ICU admission in patients with abnormal echocardiographic findings among HDPs as compared to normotensive women without abnormal 2D ECHO findings.

Conclusions: The results of our study will encourage early detection and surveillance of structural and functional cardiovascular abnormalities and management of high risk pregnant women who have high blood pressure.

LIST OF CONTENTS

SI.No	PARTICULARS	PAGE NO.
1.	Introduction	22
2.	Aims and objectives	94
3.	Materials and Methodology	95
4.	Review of literature	103
5.	Results	110
6.	Discussion	140
7.	Summary	149
8.	Conclusion	151
9.	Recommendation	152
9	Limitations	153
10.	Bibliography	154
11	Annexure Consent Case Proforma Master chart Ethics CommitteeCertificate	**166** 175 177 183 187

LIST OF FIGURES

FIG NO	PARTICULARS	PAGE NO.
Figure no 1	Placental implantation	26
Figure no 2	Immunological Factors	28
Figure no 3	Areas of ischemia and infarction, periportal haemorrhage	31
Figure no 4	Abdominal CT showing a large subcapsular hematoma	31
Figure no 5	Diagram displaying the endotheliosis of the glomerular capillaries.	32
Figure no 6	The preeclamptic maternal response to the trophoblast affecting the maternal systemic circulation	35
Figure no 7	HDP Possible mechanism of action	38
Figure no 8	Schematic diagram of the two-stage theory of preeclampsia	41
Figure 9	Eclampsia and its effect	47
Figure10	Hypertensive disorders of pregnancy	47
Figure11	Echocardiographic Changes in Women across different subtypes of Hypertensive Disorder of Pregnancy.	55
Figure12	An algorithm for treating severe preeclampsia at less than 34weeks	66

Figure13	Visual disorders	79
Figure14	Systemic flowchart of methodology	133
Figure15	Picture of 2Dechocardiography machine	135
Figure16	Picture of Echocardiogram room	136
Figure17	GE Vivid T8 Ultrasound Machine	137
Figure18	Age wise distribution between HDPs group and Normotensive group	139
Figure19	Distribution of obstetric scores between HDPs group and Normotensive group	139
Figure20	Mean gestational age distribution between HDPs group and Normotensive group	140
Figure21	Distribution of HDPs	141
Figure no 22	Maternal status at different trimesters between HDPs and normotensive group	143
Figure no 23	Past medical history between HDPs and normotensive group	145
Figure no 24	General physical examination between HDPs and normotensive group	146
Figure no 25	Mean Heart Rate (bpm) between HDPs and normotensive group	148
Figure no 26	Mean SBP, DBP and MAP values between HDPs and normotensive group	149

Figure no 27	Mean Ejection Fraction (%) between HDPs and normotensive group	149
Figure no 28	Mean Echocardiographic parameters between HDPs and normotensive group	150
Figure no 29	Mean LV Mass between HDPs and normotensive group	150
Figure no 30	Mean Diastolic parameters between HDPs and normotensive group- part 1	152
Figure no 31	Mean Diastolic parameters between HDPs and normotensive group- part 2	152
Figure no 32	Indication for different types of delivery between HDPs and normotensive group- part 1	155
Figure no 33	Indication for different types of delivery between HDPs and normotensive group- part 2	155
Figure no 34	Maternal complication between HDPs and normotensive group	157
Figure no 35	Maternal outcomes between HDPs and normotensive group	158
Figure no 36	Fetal outcomes between HDPs and normotensive group- part 1	160
Figure no 37	Fetal outcomes between HDPs and normotensive group- part 2	160
Figure no 38	Mean Baby's Birth Weight (in Kgs) between HDPs and normotensive group	161
Figure no 39	Abnormal 2D ECHO findings between HDPs and normotensive group	162
Figure no 40	Maternal outcomes based on Abnormal 2D ECHO findings between HDPs and normotensive group	164

Figure no 41	Distribution of APGAR Scores as Fetal Outcome between HDPs and normotensive group- part 1	165
Figure no 42	Fetal Outcome between HDPs and normotensive group- part 2	166
Figure no 43	Fetal Outcomes based on abnormal 2D ECHO findings patients between HDPs and normotensive group- part 3	166
Figure no 44	Mean Baby's Birth Weight (in Kgs) between HDPs and normotensive groups with Abnormal 2D ECHO findings	167

LIST OF TABLES

Table No	DESCRIPTION	PAGE NO.
Table 1	Mississippi classification of HELLP syndrome	71
Table 2	Baseline Characteristics between Hypertensive & Normotensive Pregnant Women	110
Table 3	Distribution of Hypertensive Pregnant Women	112
Table 4	Comparison of Maternal Status at different Trimesters between 2 groups using Chi Square Test	113
Table 5	Comparison of Past Medical History between 2 groups using Chi Square Test	116
Table 6	Comparison of General Physical Examination between 2 groups using Chi Square Test	117

Table 7	Comparison of mean clinical and Echocardiographic parameters between 2 groups using Independent Student t Test	118
Table 8	Comparison of mean Diastolic parameters between 2 groups using Mann Whitney Test	123
Table 9	Comparison of Mode of Delivery between 2 groups using Chi Square Test	125
Table 10	Comparison of Indications for different types of Delivery between 2 groups using Chi Square Test	125
Table 11	Comparison of Maternal Complications between 2 groups using Chi Square Test	128
Table 12	Comparison of Maternal Outcomes between 2 groups using Chi Square Test	129
Table 13	Comparison of Fetal Outcomes between 2 groups using Chi Square Test	131
Table 14	Comparison of mean Baby's Birth Weight (in Kgs) between 2 groups using Independent Student t Test	133
Table 15	Comparison of Abnormal 2D ECHO findings between 2 groups using Chi Square Test	134
Table 16	Comparison of Maternal outcomes based on Abnormal 2D ECHO finding patients between 2 groups using Chi Square Test	135
Table 17	Comparison of Fetal outcomes among Abnormal 2D ECHO finding patients between 2 groups using Chi Square Test	136
Table 18	Comparison of mean Baby's Birth Weight (in Kgs) between 2 groups with Abnormal 2D ECHO findings using Independent Student t Test	139

INTRODUCTION

The most common medical condition that develops during pregnancy is hypertension, which complicates two to three percent of pregnancies. hypertensive disorders of pregnancy as (HDP) are a significant factor in maternal morbidity and mortality.[1] The frequency of hypertension among hospitalized deliveries from 1998 to 2006 increased from 67.2 to 81.4 cases per 1000 deliveries.[2] Maternal hypertension is more common in women older than 40 years, pre-pregnancy obesity, excess weight gain during pregnancy, and women suffering from gestational diabetes.[3]

The World Health Organization (WHO) states that hemorrhage, HDP, and infection constitute the "lethal trifecta" of pregnancy. These conditions take the lives of at least one woman every seven minutes and greatly contribute to maternal mortality and morbidity.[4] Between 1990 and 2019, the number of people affected by HDP increased globally from 16.30 million to 18.08 million, suggesting a 10.9% increase over the past 20 years.[5]

Indian population has a 6.9% incidence of HDP.[6] In hospitals in India, preeclampsia incidence ranges from 5% to 15%, while eclampsia rates are at 1.5%.7. Between 1976 and 2014, the average risk of eclampsia in India was 1.5%, with a range of 0.179 to 5%. Every year, illnesses associated to pregnancy claim the lives of more than 5.2 million women globally. An estimated 62,000–77,000 people die from HDP every year, making

up 18.1% of the worldwide maternal deaths.[7]

HDPs are prevalent, can result in serious consequences for women who are pregnant, including heart attacks and strokes, and are the primary cause of pregnancy-related mortality in the US.[8] It encompasses both chronic hypertension that starts before pregnancy and persists during pregnancy, as well as pregnancy-associated hypertension that starts during or after pregnancy.[1]

Four categories are suggested for the classification of hypertensive disorders in pregnancy by the National High Blood Pressure Education Programme Working Group on High Blood Pressure in Pregnancy. Chronic hypertension, preeclampsia- eclampsia, preeclampsia superimposed on chronic hypertension, and gestational hypertension are the four conditions that need to be addressed. (either transitory pregnancy hypertension or chronic hypertension found in the second phase of pregnancy).[9]

Risk factors[10]:

- Early age
- Nulliparous women
- Age of mother more than 35 years
- Environmental elements
- Molar pregnancy
- Multifetal pregnancy

- Uterine artery Doppler abnormality at 18-24weeks of period of gestation

High risk factors[10]:-

- Prior history of PE
- APLA syndrome
- Patients with present SLE (Systemic Lupus Erythematosus)
- Chronic kidney disease
- Pre-existing history of diabetes mellitus and/or Hypertension

Moderate risk factors:-

- Multifetal pregnancy
- Primigravida
- BMI (Body Mass Index) having more than 35 kg/m2
- History of PE in running in the family
- Interval between pregnancy is more than 10 years.
- Mother´s age more than 40 years

Etio-Pathogenesis[11]:-

The following traits make women more susceptible to developinggestational hypertensiondisorders:

- First-time exposure to chorionic villi.
- Excessive exposure to chorionic villi, as in twins or molar pregnancy
- Existing diseases linked to inflammation or endothelial activation
- Genetic predisposition to hypertension developing during pregnancy

Phenotypic Expression – Pre-eclampsia

"Two-stage disorder" theory of pre-eclampsia[12]

- stage I—the placental syndrome is caused by faulty endovascular trophoblastic remodelling that downstream causes stage II—the maternal syndrome
- Stage II can be modified by maternal conditions like chronic hypertension, renal disease, obesity, immunological or connective tissue disorders, and diabetes.

Aetiology[13]:

a. An aberrant trophoblastic invasion of the uterinevessels together with placental implantation
b. Immunoregulatory tolerance between maternal, paternal,and foetal tissues that is defective
c. Maladaptation of the mother to the inflammatory orcardiovascular changes that are typical with pregnancy
d. Genetic aspects.

Stage I—Placental Syndrome

Trophoblastic invasion abnormality[14]:

- Normal: Endovascular trophoblasts replace the muscular and vascularendothelial cells.
- Abnormality: Endovascular trophoblasts line decidual vessels, but not myometrial vessels, causing a high resistance flow and a narrow diameter vessel.
- This defective endovascular remodelling may also be brought on by soluble antiangiogenic growth factors.
- Women who have a defective placentation are more susceptible to placental abruption, gestational hypertension, preeclampsia syndrome, preterm births, and foetus growth restriction.

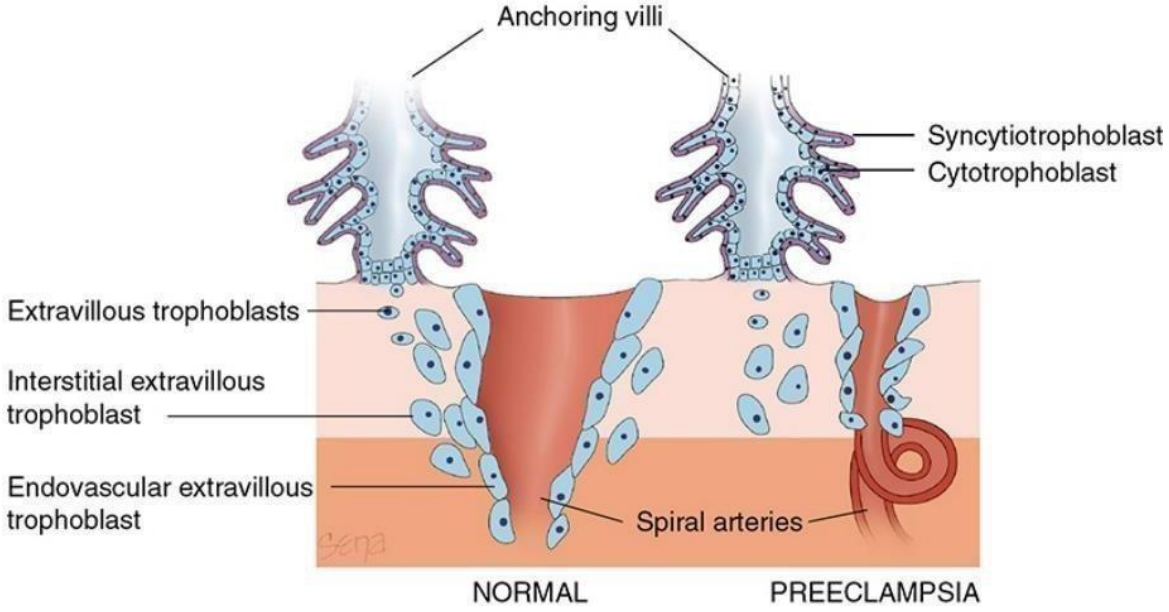


Figure no 1 : Placental implantation

Immunological Factors

- Acute graft rejection.
- Tolerance dysregulation seen when the paternal antigenic load is increased.
- Preeclampsia affects pregnant women with trisomy 13 foetuses with a 30- to 40%frequency.
- There is a rise in serum antiangiogenic factor, which mightharm the placenta and isencoded by the soluble tyrosine kinase 1 gene on chromosome 13. Therefore, preeclampsiais a serious concern for women carrying Trisomy 13 foetuses[12N].
- Women may be "immunised" against preeclampsia if they have previously beenexposed to paternal antigens.
- Preeclampsia risk is higher in multiparas who become pregnant with anew partner[16].
- Preeclampsia-prone women have lower levels of the immunosuppressive gene human leukocyte antigen G (HLAG).
- Th2 (anti-inflammatory cytokines) and Th1 (pro- inflammatory cytokines) lymphocyte activity is altered. Observed in women who develop preeclampsia in the first trimesteror the second trimester.
- CD4 cells produce Th1 cells when IL 12 is present.
- Th1 cells are generated from CD4 cells when IL 12 is present. In the rejection of allografts, Th1 cells play a key role.
- In the presence of “IL4 and IL10”, CD4 cells produce Th2 cells. The allergic reactioninvolves th2 cells. Soluble HLA- G is produced by extra villous trophoblast,

which induces regulatory type Tg1 cells. Maternal tolerance is promoted by IL10 produced from Tg1 cells.

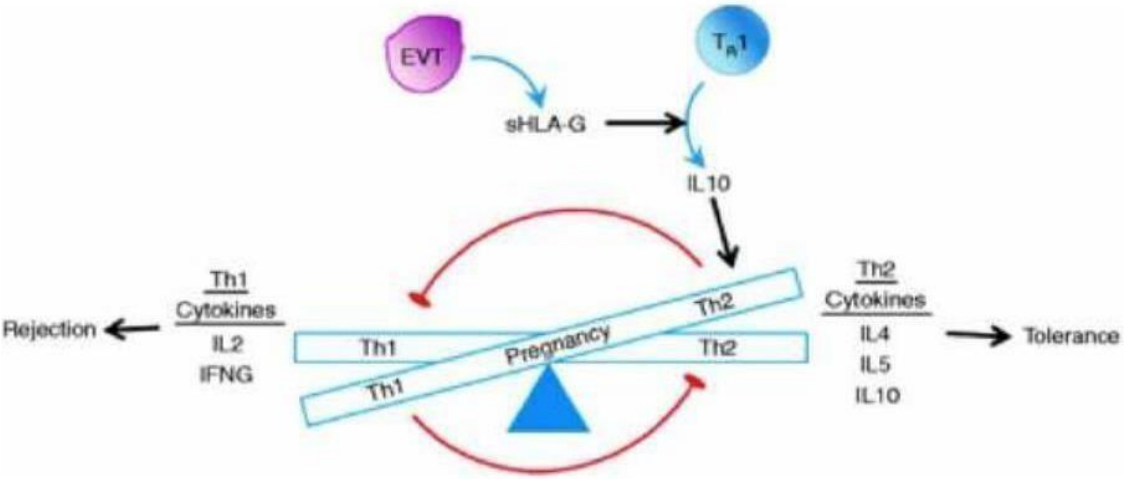


FIGURE NO 2 : Immunological Factors

Genetic Factors

- Preeclampsia is a multifactorial, polygenic disorder
- There is increase in risk of preeclampsia in daughters of preeclampsia mothers; sisters of preeclampsia women; and twins[14N].
- Ethno racial factors like in African American women.
- The genetic propensity to preeclampsia is caused by thecombination of maternal andpaternal inherited genes.
- Candidate genes include FAS polymorphism, theprotein HIF-1 α (Hypoxia induced factor), IL-1 β,Lymophotoxin -α, TGF-β.

Stage II—Maternal Syndrome

- Vasoconstriction: Hypertension is caused by an increase in vasoconstriction and flow resistance in response to endothelial activation.
- Increased responsiveness to vasopressor: Normally pregnant women start to become resistant to the vasopressors. Individuals who are susceptible to preeclampsia have highersensitivity to vasopressors.
- Prostaglandins: In individuals prone to preeclampsia, prostacyclin (PGI2) levels decline and thromboxane A2 levels rise, this enhances susceptibility to vasopressor infusions.
- Nitric oxide: Nitric oxide synthesis suppression causes the heart rate to decrease andthe mean arterial pressure to rise, mimicking the features of preeclampsia.
- Endothelin :

❖ Endothelin-1 levels are higher in preeclampsia patientsthan they are in normalpregnant women.

❖ Before the appearance of clinical features, levels ofsFlt-1 and sEng start torise.

❖ Treatment with magnesium sulphate reduces ET-1levelS

PATHOPHYSIOLOGY

CVS[18]:-

- Cardiac output is lowered and peripheral resistance is elevated

- Normal or hyper dynamic ventricular function.

- An alveolar endothelial leak that causes pulmonary oedema and a decrease in oncoticpressure because of low serum album.

Blood Volume[19]:-

- Gestational hypertension, the blood volume is at a normal level;
- In preeclampsia, the blood volume decreases

Disorders of Coagulation[20,21]:-

- Reduced platelet levels
- There are increased LDH (lactate Dehydrogenase) levels.
- The peripheral blood may show schistocytosis, spherocytosis, orreticulocytosis.
- Increased liver enzyme levels.
- Fibronectin - Elevated.

Liver [22]:-

- Right epigastric discomfort and pain are caused by liver haemorrhage andinfarction.
- Hepatic hematoma formation may be possible. This coulddevelop into a sub capsularhematoma and grow further[23].

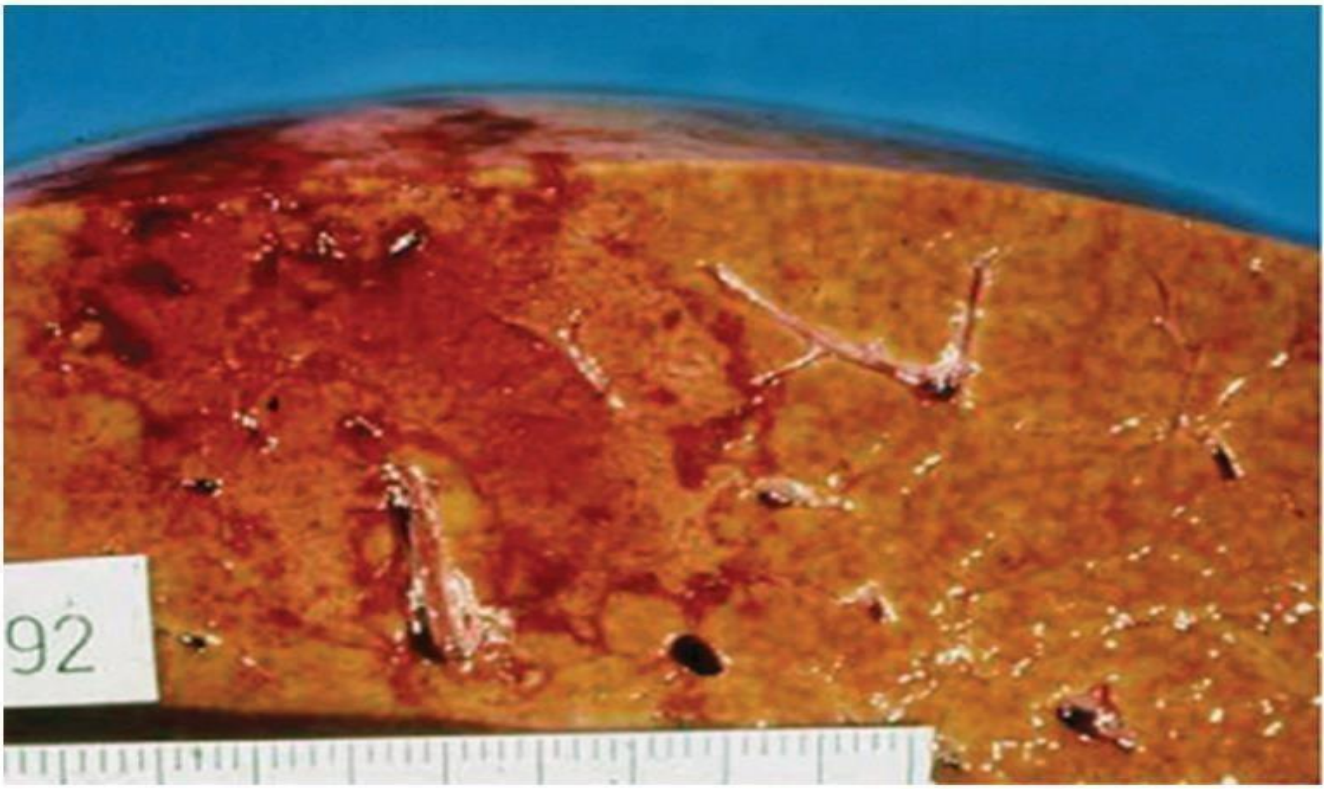


FIGURE NO 3: Areas of ischemia and infarction, periportal haemorrhage

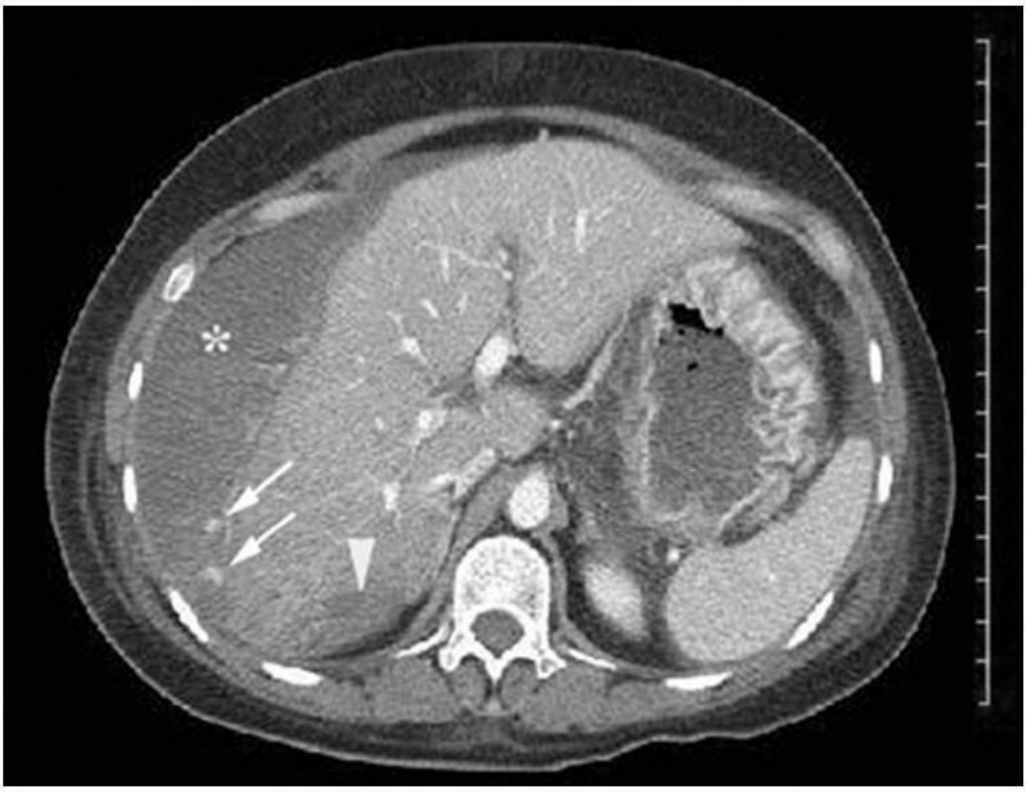


FIGURE NO 4: Abdominal CT showing a large subcapsular hematoma

Kidney[24,25]:-

- Glomerular endotheliosis results in higher serum creatinineconcentrations because itreduces filtration.
- There is an increase in urine sodium concentration and a decrease in urinecalcium.
- The level of plasma uric acid rises.
- A rise in urine protein excretion.

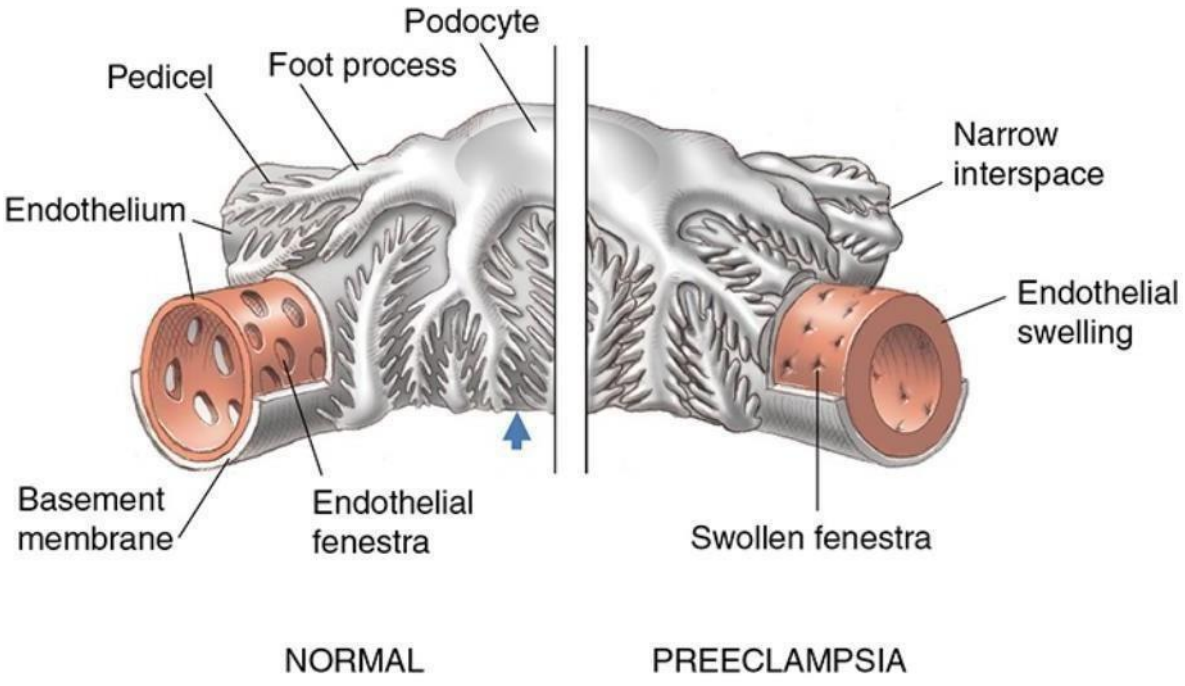


FIGURE NO 5: Diagram displaying the endotheliosis of theglomerular capillaries.

Wide endothelial fenestrations can be seen in the capillaries of the healthy glomerulus on the left side, the pedicels coming from the podocytes are widely spread shown by arrow mark.

The right-hand figure shows glomerulus with "preeclampsia syndrome-induced" alterations. The pedicels that are now touching one another, the endothelial cells, and their fenestrae have all swelled and narrowed.

Placenta[26]:-

- Chorionic villi congestion and proliferative endarteritis in the placenta
- An increase in infarcts and haemorrhages
- Increased syncitial knots and proliferation of cytotrophoblastic cells.

Brain[27,28]:-

- Cytotoxic oedema is brought on by cerebral vasoconstriction brought on by autoregulation. whenthis fails, it causes vascular permeability and vasogenicoedema..
- There may be visible infarction foci.
- Eclampsia and chronic hypertension increase the risk of cerebral hemorrhage.
- When a pregnant woman has significant hypertensionwith complications, an MRI should be done[29]

Hemodynamic changes in normal pregnancy and HDPs:

Circulating blood volume [30], cardiac output [31] and peripheral vascular resistance [32] are typical modifications in the mother's cardiovascular adaptation to pregnancy.

A state of low peripheral resistance and high cardiac outputis frequently used to describe a healthy pregnancy. Maternalcardiovascular function, including heart rate, vascular characteristics, myocardial contractility, and function, is significantly altered in this hemodynamic condition. There has been conflicting descriptions of myocardial function during pregnancy: it can be normal, depressed, or increased.

[33,34].

Preeclampsia, however, will always have an impact on the cardiovascular system, as evidenced by the hypertension, capillary leakage, proteinuria, and edemas that are related to this illness.It appears that endothelial dysfunction is a major factor in the preeclampsia pathogenesis.. Endothelial dysfunction seems to play a key role in the pathogenesis of preeclampsia .

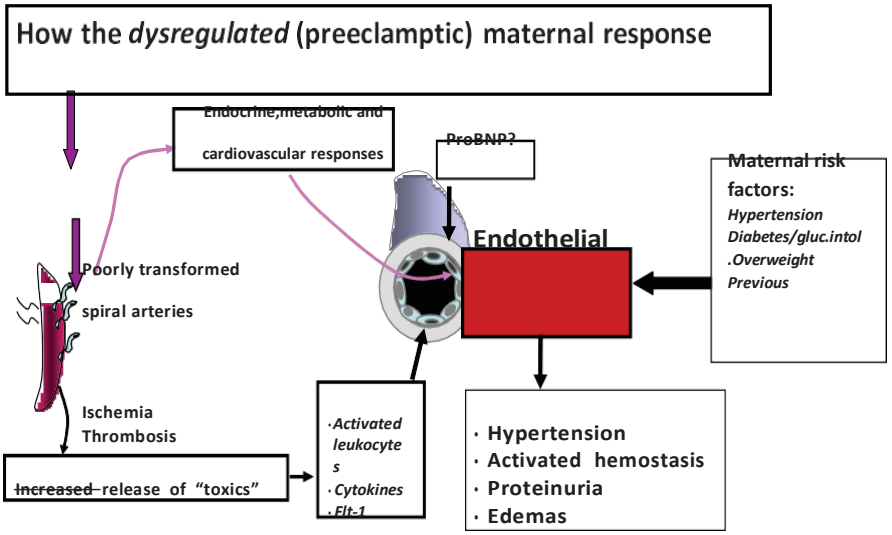


FIGURE NO.6 Preeclamptic maternal reaction to trophoblast affecting maternal systemic circulation.

High cardiac output and low peripheral vascular resistance are characteristics of a healthy pregnancy, as previously mentioned. In preeclampsia, the situation is frequently reversed, with a hemodynamic profile more analogous to the non-pregnant state with high vascular resistance [35,36]. Other vascular parameters, such as total arterial compliance and proximal aortic stiffness, have been investigated in the preeclamptic population. Noninvasive experiments utilising pressure and flow estimates reveal that overall arterial compliance is reduced, and proximal stiffness increases in preeclampsia. [37,38]. Furthermore, some evidence suggests that enhanced sympathetic activity in these patients [39] may contribute to the normal hemodynamic condition. There is significant evidence that endothelial dysfunction plays a fundamental role in the pathogenesis of preeclampsia, which conveniently explains

many of the cardiovascular, inflammatory, hematological, and clinical features of the preeclamptic patient. [40].

1.1 Types of hypertensive diseases during pregnancy:

I. **Chronic Hypertension in Pregnancy:** Chronic hypertension is defined as high blood pressure that is occuring during the first 20 weeks of pregnancy or that do not reduce by the 12week postpartum period evaluations[1]. There are two recognized levels of severity: mild, upto 179 mm Hg systolic and 109 mm Hg pressure), and severe,(>180mm Hg systolic or 110 mm Hg diastolic pressure)[41]. Approximately 5% of pregnancies are complicated by chronic hypertension, and as more women delay childbearing, the prevalence of this medical condition is increasing. The intensity and persistence of high blood pressure are closely correlated with the rates of complications. For example, preeclampsia with superimposition is more likely to occur in patients with high hypertension during the first trimester. Every patient with hypertension should have prenatal testing, repeated laboratory testing during pregnancy, and regular ultrasound scans to monitor growth[42]. While many women with chronic hypertension do well in their pregnancies, they are more likely to experience a number of pregnancy-related problems, including superimposed preeclampsia, fetal growth restriction, placental abruption, premature birth, and cesarean section[1,3].

II. **Gestational Hypertension:** The development of hypertension after 20 weeks of

gestation is called gestational hypertension, formerly known as pregnancy-induced hypertension or PIH[7] (Pregnancy Induced Hypertension). Clinical symptoms include; High blood pressure (systolic ≥ 140 or diastolic ≥ 90 mm Hg) that was previously normal, Urine containing no protein, and Absence of preeclampsia or eclampsia manifestations. Nearly 50% of women diagnosed with gestational hypertension between 24-35weeks of pregnancy are prone to develop Preeclampsia[3].

There is still no established diagnostic test that can determine the risk of developing gestational hypertension. The primary sign used to diagnose gestational hypertension is elevated blood pressure. some women may not exhibit any symptoms at all. Symptoms linked to gestational hypertension are Edema, Sudden gain of weight, Blurred vision ,light sentivity, vomiting, nausea, headaches, and raised blood pressure[43].

One of the main causes of morbidity and mortality in mothers, fetuses, and newborns is PIH. Disseminated intravascular coagulation (DIC) , organ failure, abruptio placentae, and cerebrovascular events are among the risks that women with PIH encounter. These mothers face a higher risk of intrauterine growth retardation, preterm, and intrauterine death in their fetuses[44]. Treatment of HDPs always depends on blood pressure, age of gestation, symptoms, and risk factors related to it. When SBP (Systolic Blood Pressure) falls between 140 and 149 mmHg or DBP (Diastolic Blood Pressure) falls between 90 and 99 mmHg, non-drug therapy is advised.[1,3]. The blood pressure thresholds used by various

health organizations for medication management during pregnancy differ. Antihypertensive medication is advised in pregnancy when blood pressure is ≥ 150/95 mmHg, as per 2013 ESH/ESC guidelines. Women having a) gestational hypertension, with having proteinuria or not, b) preexisting hypertension along with superimposed gestational hypertension, or c) hypertension along with organ damage symptomatic or assymtomatic at any time during pregnancy, should start antihypertensive treatment at values ≥ 140/90 mmHg[45].

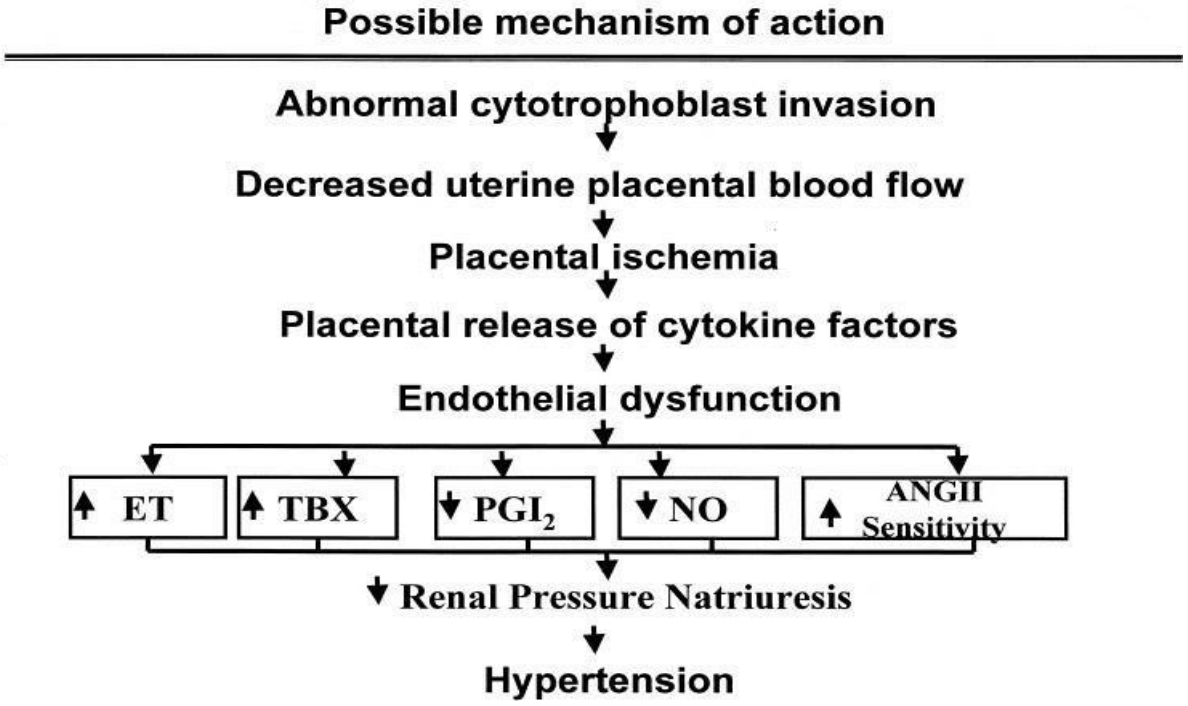


FIGURE NO .7 HDPs Possible mechanism of action

III. **Preeclampsia:** Preeclampsia is a pregnancy-related hypertension condition that accounts for 2% to 8% of all pregnancy-related problems worldwide. In low-

income nations, it causes 9% to 26% of maternal deaths, while in high-income countries, it causes 16% of deaths[46]**.** Preeclampsia is a multiorgan condition with complex unknown etiology. which is mainly characterized by the development of hypertension (≥ 140 mm Hg systolic or ≥ 90 mm Hg diastolic) plus proteinuria(> 0.3 g/24) after 20weeks of period of gestation[47].

The primary etiology of preeclampsia is unknown, even though the clinical presentation, diagnostic standards, and management of preeclampsia are already widely accepted. There are multiple theories for the pathophysiology of preeclampsia. The most widely accepted theory is the immunologic concept. The "30-15" criteria was once used to diagnose preeclampsia, which was defined as SBP more than 30 mm Hg over baseline BP and DBP more than 15 mm Hg above baseline BP. But because it is so unclear, the rule is no longer acceptable[3].

Similarly, generalized edema (affecting the hands and face) was used as a diagnostic criterion and is no longer regarded as a valid diagnosis due to its excessive variability[1]. Preeclampsia differs in severity from moderate to severe. Preeclampsia exposes the fetus and mother at risk. Delivery is usually not recommended until 37 to 38 weeks of gestation for women with mild preeclampsia, although it is to happen by 40 weeks[42].

Preeclampsia is a condition whose origin is unknown, but one essential factor in its development is placental insufficiency brought on by insufficient remodeling of the mother vasculature, which perfuses the intervillous space. This could

cause the placenta to go through a complicated process of ischemia-reperfusion, releasing harmful substances into the mother's circulation[48].

Cardiovascular problems, vascular endothelium damage, and an imbalance in angiogenesis are associated with the placental hypoxia/reoxygenation phenomena. Additionally, this process raises oxidative stress, which is detrimental to the health of both the mother and the fetus[49].

It has been suggested that pre-eclampsia is a two-stage condition caused by an imbalance between factors that promote and inhibit angiogenesis[50]. The main theory states that improper spiral artery remodeling causes cellular ischemia within the placenta, which consequently causes an imbalance between pro- and anti-angiogenic factors, ultimately leading to pre-eclampsia. All of the maternal organ systems are affected by the broad endothelial dysfunction caused by this disparity in favor of anti-angiogenic agents. Fetal growth limitation is another factor[51].

Despite much research in this area, the underlying cause of pre-eclampsia is still unknown. Research suggests that impaired endothelial function and cardiovascular and renal disease are the main underlying causes of hypertension and proteinuria, which are the cornerstones of the pre-eclampsia diagnosis. But treatment for preeclampsia is not much improved over the previous fifty years[51-56].

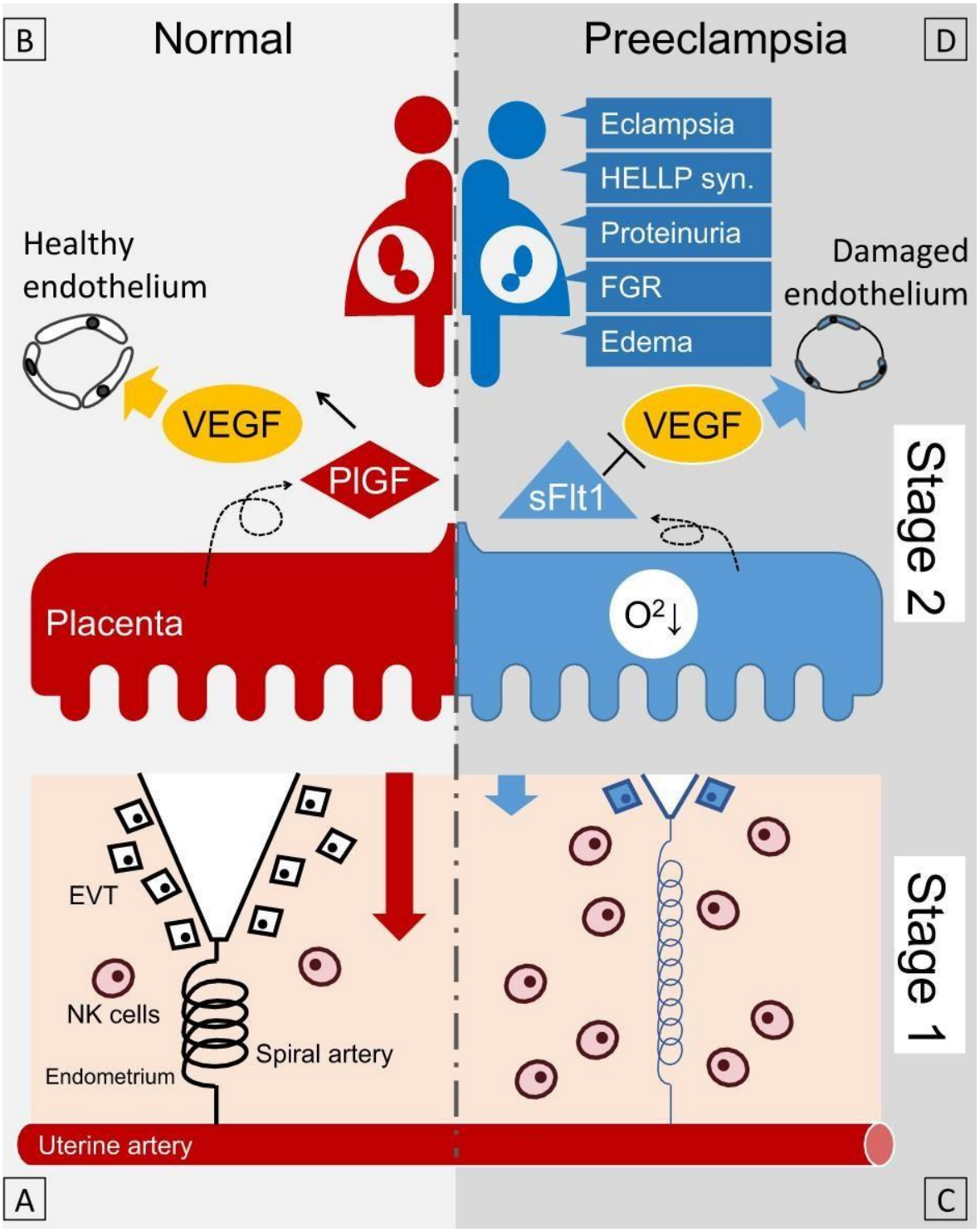


FIGURE NO.8 Schematic diagram of the two-stage theory of preeclampsia.

In an ordinary pregnancy, the right amount of extrauterine vein invasion (EVT) into the endometrium of the mother (red colored arrow) shows sufficient maternal blood flow from the spiral artery (A). B. PlGF, a placental secret, which stimulates Vascular

Endothelial Growth Factor(VEGF) and keeps the endothelium in good condition. C. preeclamptic pregnancy: However, partial invasion of the EVT (blue arrow) results in inadequate maternal blood supply from the spiral artery and resultant placental hypoxia. D. The placenta now secretes sFlt1, which inhibits VEGF and causes systemic endothelial dysfunction as well as several clinical symptoms[57].

Pre-eclampsia is indicated by proteinuria, a decrease in cardiac output and blood volume, and an increase in systemic vascular resistance[3]. The renin-angiotensin system (RAS) and aldosterone secretion are suppressed despite the drop in blood volume, or hypovolemia. This could imply that hypertension in PE is not always caused by the RAS, but rather that the suppression of RAS can result from hypertension. Early in pregnancy, abnormal placentation may cause the release of cytokines and anti-angiogenic substances, which could cause vascular dysfunction[58].

The placenta has been found to have a critical role in the pathophysiology of preeclampsia. A sequence of events culminating in this sickness is initiated by aberrant cytotrophoblast infiltration of spiral arterioles, which reduces uteroplacental perfusion[59]. Placental ischemia causes the release of soluble pro- or anti-angiogenic placental factors. These substances enter the maternal bloodstream and lead to endothelin-1 and superoxide production, an increase in vascular sensitivity to angiotensin II, and a decrease in the production of vasodilators such as nitric oxide. These effects are caused by malfunction of the maternal vascular endothelium[60]. The placenta releases more sFlt-1 as a result of the mother's elevated vascular resistance and endothelial dysfunction,

which lowers endothelial nitric oxide synthase and VEGF receptors. Both diastolic relaxation and endothelial function are impacted by this. Its hypertrophy may impact cardiac function. Proteinuria is caused by damage to the glomerular cells' podocytes and an increase in the excretion of podocyte-specific proteins in the urine. Additionally, sFlt-1 may impair thyroid hormone activity during or after pregnancy, potentially affecting liver function[61].

VEGF is bound and rendered inactive by elevated anti-angiogenic factors such as sFlt-1. According to research, the anti-cancer medication sunitinib, which inhibits VEGF, causes PE-like syndrome, which is typified by hypertension, proteinuria, and kidney injury[60]. Increases in endothelin-1 levels are also observed in such circumstances. It is discovered that endothelin-1 suppresses renin and is a separate factor in the hypertension and proteinuria observed in PE. Endothelin receptor blockers have been proven to stop the progression of PE in animal models of the illness. Therefore, the endothelin system may have a significant role in the development of PE's clinical symptoms[62].

Preeclampsia can also occur as a result of several risk factors. These include:

- Having multiple pregnancies, such as twins or triplets
- Pregnancy after 40 years of age
- Experienced preeclampsia in a previous pregnancy
- Having a preeclamptic family history
- Being obese
- Having a medical history of illnesses such as sickle cell disease, diabetes,

high blood pressure, kidney disease, lupus, or other autoimmune conditions

- Conceiving using in vitro fertilization

Predicting pre-eclampsia, which may indicate the degree of aberrant placentation, endothelial dysfunction, and feto-maternal unit perfusion, has been given substantial attention due to its significant impact on maternal and neonatal morbidity and mortality[50]. Numerous biomarkers aid in the disease's early identification. Early in pregnancy, lower mother blood levels of PP13 (Placental Protein 13) and PAPP-A (Pregnancy Associated Plasma Protein-A) may indicate the onset of pre-eclampsia. Additionally, elevated first-trimester levels of homocysteine, ADMA, s-Eng, leptin, and sFlt-1 indicate the disease's later stages of development. Elevated serum levels of ADMA, homocysteine, PAPP-A, and sFlt-1 are linked to a more severe case of pre-eclampsia. Finding these biomarkers could aid in improving pre- eclampsia patient monitoring and care[63].

In a healthy pregnancy, the maternal heart rate rises by 20% and the cardiac output rises by 30%–40% to make up for the decrease in systemic vascular resistance and to enhance the blood supply to the uterine arteries[64]. In normal pregnancies, vasodilation of the mother's systemic circulation can be observed as early as five weeks. Complete placentation and the growth of the uterine and placental circulations happen subsequently. Pregnancy is associated with a reduction in mean arterial pressure. Although most of the decrease in arterial pressure can be seen early in pregnancy, it persists significantly in the second trimester. The significance of correlating hemodynamic evaluations with preconception values instead of early pregnancy rates isstressed because these changes

occurred very early in pregnancy. The third trimester reveals an increase in arterial pressure, which by the end of the postpartum period returns to preconception levels [65-67].

However, placental ischemia in pre-eclampsia results from poor spiral artery remodelingof the placenta, which releases pro-inflammatory and antiangiogenic substances into the mother's blood. These elements then lead to vasoconstriction and endothelial dysfunction, which raise arterial blood pressures in pre-eclampsia.

Patients exhibiting preeclamptic symptoms should undergo prompt diagnostic testing after a complete medical history and physical examination. This consists of laboratory testing for pregnancy-induced hypertension, which includes a complete blood count to screen for thrombocytopenia, a complete metabolic panel to check for impaired liver function, and renal insufficiency[66]. A urinalysis is performed to determine proteinuria. The only possible treatment for preeclampsia is delivery. The purpose of corticosteroids is to accelerate the maturation of fetal lungs. A cesarean section or vaginal delivery are two possible delivery methods. A cesarean section is recommended when the fetus is in distress, when the oxytocin challenge test results in late deceleration, when labor induction fails, and when there are other symptoms such as a tight pelvis and malpresentatio[68]. The following drugs are typically used to treat severe preeclampsia: Antihypertensive medications: can reduce blood pressure. anticonvulsant drugs such as magnesium sulfate which helps to prevent seizures, and Corticosteroids to help the lungs of an infant develop before delivery[69] .

IV. **Eclampsia:** Eclampsia is a rare condition but a serious preeclampsia condition.

Less than 3% of patients with preeclampsia experience eclampsia. It requires immediate medical evaluation and creates difficulties during pregnancy. Eclampsia can occur suddenly in a patient with slightly elevated blood pressure and no proteinuria, or it can be the development of convulsions in a patient with pre-existing pre-eclampsia. The specific reason is unknown, however, edema andcerebral ischemia have been suggested. A seizure of eclampsia may occur in antepartum (53 percent of cases), intrapartum (19 percent), or postpartum (28 percent of cases) [69]. Although it is thought to be a complication of preeclampsia sometimes preeclampsia cannot necessarily manifest. These seizures may put a pregnant woman in a coma or result in confusion and disorientation. Sometimes it can result in a stroke or even death. Preeclampsia is typically treated prior to itsprogression to eclampsia.

The likelihood of developing eclampsia can be decreased by receiving treatment for preeclampsia. You can lower your risk by obtaining timely medical treatment,attending all prenatal checkups, and leading a healthy lifestyle. Some conditionscan increase the risk of developing preeclampsia and eclampsia. If in case of higher risk, starting low-dose aspirin in the first trimester may help lower the riskof developing preeclampsia.

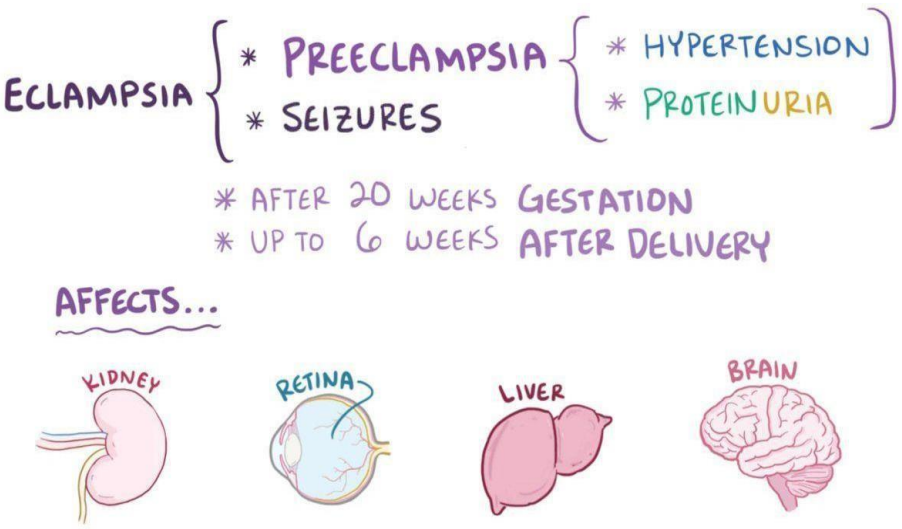


FIGURE NO.9 Eclampsia and its effect

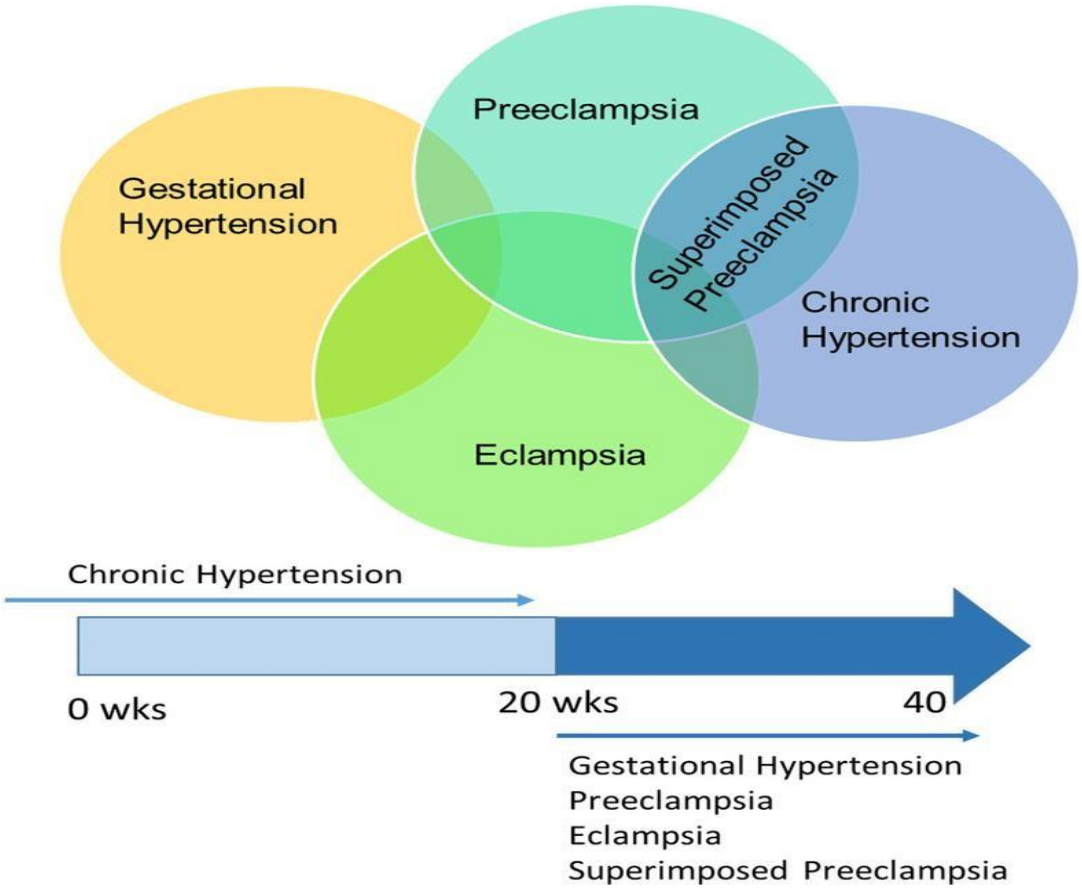


FIGURE NO .10 Hypertensive disorders of pregnancy

Management:-

Pre conception care:

- Due to their teratogenic effects, ACE-I (Angiotensin converting enzyme-I), Atenolol, Statin's, Thiazide's should be stopped using.

Evaluation[70]:-

- Thorough examination, including daily monitoring for headache, vision changes, orepigastric pain;
- Daily weight measurement to detect rapid weight gain;
- Readings of blood pressure every four hours;
- Increasing haemoglobin percentage.
- A urine test for proteinuria
- Dropped platelet count
- Schistiocytes on the peripheral smear;
- Elevated INR and APTT (in DIC)
- Aberrant LDH.
- Raised Serum creatinine levels
- Raised transaminases, bilirubin levels
- A lower level of albumin
- A fundus examination
- An assessment of foetal size, health, and amnionic fluid index.

Prenatal Care: - Per NICE45b (National Institute of Health and Clinical

Excellence) recommendations

- From the 12th week of pregnancy until delivery, Women should take low dose aspirin daily if they have at least "1 severe or 2moderate risk factor" for preeclampsia.
- They should also limit their physical activity
- Undergo weekly or biweekly antenatal visits for blood pressure checks and anti-hypertensive therapy.
- Women with preeclampsia who also have complications are also advised to stay in thehospital.

Criteria for managing mild preeclampsia at home:-

- Systolic of less than 150 mm Hg, Diastolic of less than 100 mm Hg
- Normal lab findings and no maternal symptoms
- 1 g or less of protein in the urine in 24 hours
- Reassuring foetal condition with sufficient growth;

1.2 Diagnosis for Hypertensive Disorders of pregnancy

- Laboratory investigations such as complete blood cell count, Blood Urea Nitrogen (BUN), creatinine, uric acid, ALT, and AST both as baseline and serial tests.
- Echocardiography for evaluation of maternal changes and fetal health.

- Doppler ultrasonography and ultrasound to assess fetal growth and amniotic fluid volume. Early uteroplacental insufficiency may be identified by umbilical artery systolic/diastolic ratios determined by Doppler ultrasonography
- Prenatal examination (biophysical profile, nonstress test). The biophysical profile is a measure of the health of the fetus. Well-oxygenated fetuses exhibit typical behaviors such as wiggling, twisting, flexing and extending their extremities, and breathing. Hypoxic fetuses remain motionless in an attempt to conserve oxygen.
- A 24-hour urine collection for protein evaluation.

Preventing seizures, lowering blood pressure to protect the mother's end organs, and accelerating delivery are the objectives of treatment [71,72]. There are significant changes in the cardiovascular system during pregnancy. The heart must adjust to them by increasing cardiac output (up to 50%), decreasing peripheral vascular resistance, and increasing plasma volume. During labor and delivery, the heart has to respond to an acute increase in workload. Once these alterations are resolved, the heart must then readjust in the weeks following pregnancy [73].

High blood pressure can be harmful to both the mother and the fetus, even though many pregnant women suffer from it and have healthy babies without any serious complications. Compared to women with normal blood pressure, those with pre-existing or chronically high blood pressure are more prone to experience specific complications during pregnancy. High blood pressure may result in modest to severe consequences.

Low birth weight and premature delivery can result from high blood pressure, which can also damage the mother's heart, kidneys, and other organs. In the worst situations, the mother experiences preeclampsia, also known as "toxemia of pregnancy", which places the mother's and the fetus' lives in danger.

Women with hypertensive disorders of pregnancy (HDP) can evaluate their heart function using two-dimensional echocardiography (2D-echo), a traditional evaluation method. Pregnancy-related hypertension and pre-eclampsia (PE) are two HDPs that can significantly alter a mother's heart's geometry and function. When determining who requires closer postpartum care and identifying cardiac damage, 2D-echo can be helpful.

To evaluate fundamental cardiac parameters such as valvular structures, chamber size, ventricular function, and congenital variations, conventional 2D imaging is performed. Doppler echocardiography is utilized in addition to 2D imaging to evaluate for valvular disease to detect shunts. To evaluate diastolic function, peak early (E) and late

(A) diastolic blood flow velocities at the level of mitral valve are two typically reported values that Doppler can identify. Pregnant women can be examined using advanced echo techniques that can detect modest changes in heart function, such as subclinical systolic and diastolic abnormalities[74]. An echocardiogram (echo) is a procedure that provides a graphic outline of the heart’s movement. It frequently combines color Doppler and Doppler ultrasonography with echo to assess blood flow via the heart's valves. Radiation is not used during an echocardiogram, An echo differs from techniques such as CT scans and X-rays that utilize radiation.

Types of echocardiogram:

- **Transthoracic echocardiogram (TTE):** A transthoracic echocardiography, or TTE, is a test that produces images of the heart using ultrasonography, or sound waves. TTE can assess the health of the heart and determine the root causes of symptoms related to the heart. The examination is either minimally invasive or noninvasive.
- **Transesophageal echocardiography (TEE):** Transesophageal echocardiography (TEE) is a kind of echo test that uses sound waves to produce images of the heart. Many conditions, such as blood clots and heart infections, can be detected using a TEE.
- **Exercise stress echocardiogram**: An exercise stress echocardiography is a pre- and post-exercise ultrasound of the heart. It creates visuals that demonstrate how the heart works under pressure.

The heart can be represented using a variety of techniques. Depending on the particular circumstance, the techniques are

- **Two-dimensional (2D) ultrasound.**
- **Three-dimensional (3D) ultrasound.**
- **Doppler ultrasound.**
- **Color Doppler ultrasound.**
- **Strain imaging.**
- **Contrast imaging**

Echo is the least expensive and invasive technique for evaluating heart structure and

various functions are accessed they are as follows;

Systolic dysfunction: The percentage of the end-diastolic volume ejected during systole, or ejection fraction, is used to determine LV systolic dysfunction. Usually, this is visually estimated using all of the available echo views. An ejection fraction of 50% to 80% is considered normal; however, end-stage heart failure is compatible with values as low as 5%.

The E/A ratio: Two waves are typically observed when using PW Doppler to analyze flow over the MV. These are the early [E] wave, which is the passive filling of the ventricle, and the atrial [A] wave, which is the active filling with atrial systole. Traditionally, the Ewave travels at a faster speed than the Awave. Two abnormalities, however, may occur under circumstances that restrict the LV's compliance: reversal, in which the A wave is greater than the E wave. It can be caused by hypertension, left ventricular hypertrophy (LVH), or slow filling caused by advancing age. exaggerated of normal - a tall, thin E wave with a short or missing A wave. This suggests infiltrative cardiac disease, constrictive pericarditis, or restrictive cardiomyopathy.

Diastolic dysfunction: Reversed E/A ratio, thickened ventricle, and normal LV cavity size are typical echo findings with diastolic dysfunction.

Aortic stenosis: Calcification or the appearance of a bicuspid valve can be used to diagnose aortic stenosis (AS). Using Doppler to measure the high-velocity flow over the valve, one can assess the severity of the stenosis [75].

Since echocardiography is rarely used for uncomplicated pregnancies, there isn't much data available on the echo features of healthy pregnant women. Normal echo values have been suggested by a few cohort studies; however, the reported findings are not consistent because the values were collected at different times of pregnancy. Furthermore, since both cardiac preload and afterload vary with positional alterations of the gravid uterus, variations in patient positioning may also have an impact on measurements. Therefore, while interpreting echo findings in pregnant women, it is crucial to take into account information regarding gestational age and patient posture (e.g., supine versus left lateral decubitus)[76,77].

Additionally, diastolic function parameters show more pronounced changes in women with HDP, such as:

- Diminished ratio of E/A mitral inflow
- A higher E/e′ ratio
- thicker LV walls
- elevated LV mass index
- elevated RV systolic pressure estimate
- Unusual RV tension

The evaluation of HDP-related cardiac remodeling advantages significantly through echocardiography, particularly when strain imaging is used, as it may be able to identify minute alterations in cardiovascular function before developing hypertension or other medical conditions. (Shahul S et al.,2018). Echo can differentiate between the

anticipated eccentric LV hypertrophy associated with a normal pregnancy and HDP-related increased LV mass and concentric hypertrophy, which is particularly common in women with preeclampsia. Echo can also detect chamber expansion associated with HDP, most prominently in the left atrium [79].

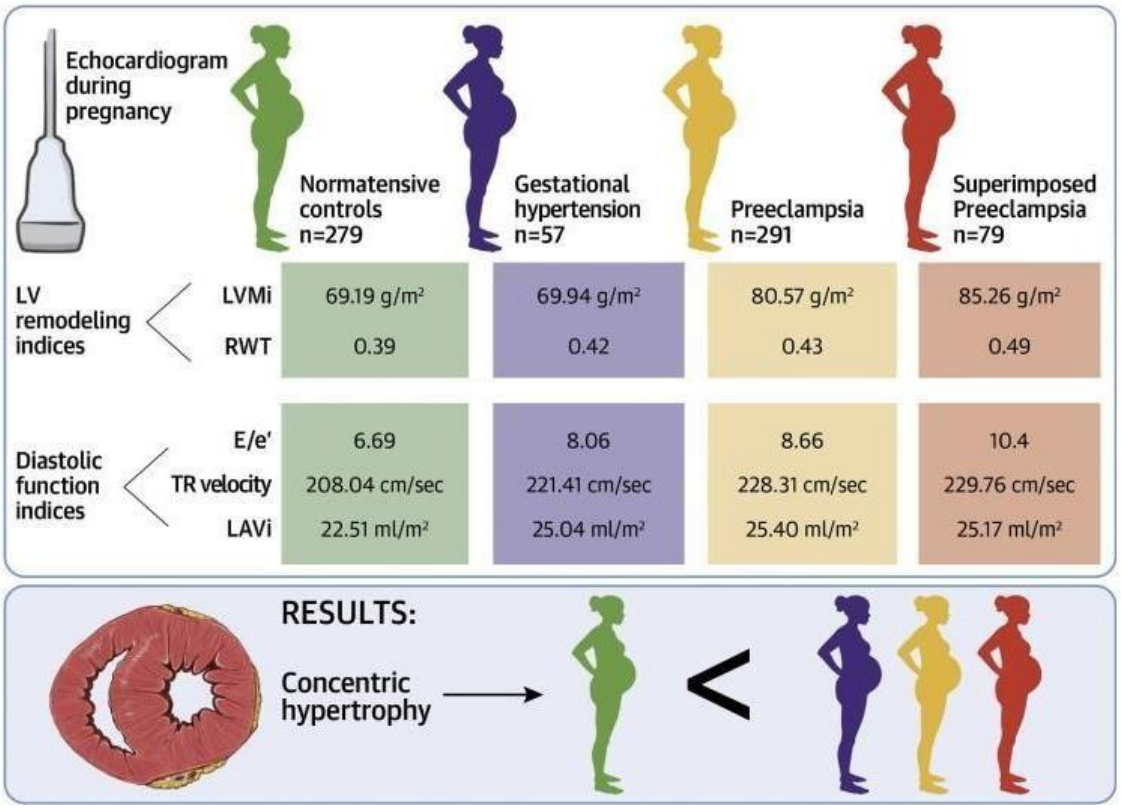


FIGURE NO 11. Echocardiographic Changes in Women across different subtypes of Hypertensive Disorder of Pregnancy.

The range of HDP complications can be observed in the variations in echo data for systolic function in patients. For example, systolic function remains unchanged in gestational hypertension. On the other hand, preeclampsia is more frequently linked to systolic dysfunction; more severe or early-onset preeclampsia is associated with more dysfunction. The majority of data for all forms of HDP indicate a correlation with diastolic dysfunction, especially in pre-eclamptic patients. Despite the absence ofroutine

monitoring in current practice, the echo abnormal findings linked to HDP emphasize the significance of imaging in women who have HDP or who are at risk for it. Echocardiograms should be performed on these women at least once during pregnancy to detect any minor heart abnormalities before they become clinically worse. Individuals who match the criteria for serial echocardiography and have severe signs of preeclampsia are particularly vulnerable to pulmonary edema and other cardiovascular conditions [80].

This study explores the importance of understanding the heart's function in hypertensive mothers for timely diagnosis, better management, and a good prognosis. It highlights the feasibility of using echocardiography as a routine investigation for hypertensive disorders during pregnancy.

Role of 2Decho in normal pregnancy and hypertensive disorders of pregnancy:-

There are various components to current research on hemodynamics and vascular characteristics in normal pregnancy and preeclampsia. To determine the typical ranges of hemodynamic parameters, first a thorough understanding of the natural sequence of physiological changes during healthy pregnancy is required. Such information is important for health professionals who work with women of childbearing age because it provides a theoretical foundation when dealing with abnormal pregnancies or women who have pre-existing medical issues that may be affected by pregnancy. Second, it is becoming increasingly evident that the different subgroups of preeclampsia are pathogenetically at least partly diverse [81] and require additional definition.

Third, the large increase in risk of cardiovascular disease later in life in previous preeclamptic women calls for data that can provide pathophysiological knowledge and enhance health recommendations to women at risk. Given that the cardiovascular system is influenced in normal pregnancy, becomes deranged in preeclampsia, and preeclampsia is a predictor of future cardiovascular risk, investigations of the cardiovascular system in pregnant women should be a key component of women's health studies.

Cardiovascular adaption studies in pregnant women are particularly difficult because they must be conducted with no fetomaternal danger or discomfort. To reduce fetomaternal risk and changes in normal maternal cardiovascular physiological state, investigations should be brief, harmless, and not require sedatives.

Noninvasive approaches have been developed to provide thorough assessments of systemic arterial characteristics and left ventricular (LV) function. Systemic vascular characteristics, such as peripheral resistance, total arterial compliance, and characteristic impedance, can be determined using simultaneous echocardiography and external arterial pressure waveform recording. [82-84]. Understanding LV performance necessitates not only an examination of the LV's characteristics, but also an investigation into the influence of the arterial system on left ventricular function. The connection between the LV and the arterial system, known as ventriculo-arterial coupling (Ea/ELV), is a major driver of cardiovascular performance and cardiac energetics, although it has yet to be thoroughly studied in either normal or pathological pregnancy.

Preeclampsia has a higher impact on the heart than gestational hypertension, with the most significant changes occurring in early-onset, severe disease[85]. Early research into cardiac anatomy and function can lead to improved maternal and foetal health outcomes.

Henceforth, A safe, non-invasive method for evaluating the structure and function of the cardiovascular system during pregnancy is echocardiography[86]. The temporal variability of echocardiography is minimal, while being operator dependent and requiring training to get reproducible data.[87,88]. Pregnant women with hypertension who undergo echocardiography reveal increased left ventricular mass and remodelling, which in certain cases results in diastolic dysfunction. For a more accurate assessment of chamber deformation, a number of methods have been employed, including tissue Doppler analysis, pulse Doppler analysis (PDA), two-dimensional and M-mode echocardiography, and other more recent methods.[88].

Pregnant women have an increased cardiac output accompanied with a decreased systemic vascular resistance.[89,90]. Hypertension causes generalized vasospasm and increased peripheral vascular resistance further leading to increased afterload and reduced left ventricular ejection fraction[89,90]. This leads to cardiac remodelling and increased left ventricular mass index. left ventricular ejection fraction (LVEF) in normal pregnancy is 55-70%[91]. Less than 50% is considered as decreased LVEF. Stroke Volume (SV) normally increases in pregnancy up till the end of the second trimester after which it decreases. Stroke Volume in normotensives is 73.3±14.19 ml, whereas it is 70.8±3.22 ml in patients with hypertension[92]. Left Ventricular mass(LV

Mass) in normotensive is 90.6±19.8 whereas it is 106±29.4 in hypertensive pregnant women[93] .

Intrapartum management[94,95]:

- The goal of intrapartum treatment is to keep diastolic and systolic blood pressure at or below 110mmHg and 160mmHg, respectively.
- Start giving antihypertensive if you have mild hypertension and a serious illness or organ malfunction.
- Patients with mild to severe hypertension and a co-morbid disease are advised to receive antihypertensive treatment.
- monitoring the fetal heart rate continuously
- Eclampsia prophylaxis provided to women with severe preeclampsia or imminent eclampsia
- Vaginal delivery recommended unless there are obstetric indications.
- If the bishop score is low, prostaglandins should be used for induction.
- Avoid using ergometrine
- Practice active management of the third stage of labour (AMTSL).

Timing of Delivery ACOG (American College of Obstetricians and Gynecologists)

- Patients with well controlled HT (With drugs):- 37 to 39 period of gestation.

- When mild preeclampsia is present, the result of labour induction after 37 weeks is superior to expectant treatment.
- When Hypertension is severe can terminate at 36 - 37 weeks.

MANAGEMENT OF GESTATIONAL HYPERTENSIONSUPPORTIVECARE:

- Most of the day should be spent with less physical activity; complete bed rest is not required. (venous stasis and risk of thromboembolism, muscle disuse atrophy are due to long duration bed rest)
- Calorie and protein intake should be adequate but not excessive, and sodium and hydration intakes shouldn't be restricted or mandated.
- Gestational hypertension without risk factors patients are instructed how to perform daily foetal movement counts
- They should have clinical visits every week for the assessment of maternal and fetal well being
- Gestational hypertension with risk factors should be managed as severe preeclampsia and admitted to the hospital to complete her evaluation and start medical treatment.
- Initial evaluation includes full blood count with platelets, LDH and liver enzymes, urea and electrolytes
- Ultrasound for fetal growth, amniotic fluid volume and umbilical and cerebral Doppler every 2 weeks and weekly Cardiotocography(CTG)
- They require treatment with antihypertensive agents.

- The aim of the treatment is to avoid main complications like ischaemicor haemorrhagic stroke, congestive heart failure, myocardial infarction, renal injury, pulmonary oedema .
- Gestational hypertension by itself is not an indication for caesarean section except in severe cases unresponsive to treatment or with FGR
- If cervix is ripe, vaginal delivery is the best option, but if cervix is unripe and the cervical length is >2.5cm caesarean delivery may often be Indicated to avoid a prolonged induction
- Induction can still be an option with unripe cervix if the BPs are well controlled.

MANAGEMENT OF MILD PREECLAMPSIA

- Close supervision of the pregnant woman is need for this hospitalizationis needed, once the blood pressure is under control weekly antenatal visits or twice in a week is needed
- Goal in management of mild preeclampsia is to prevent it progression to severe PE and also organ dysfunction, continuing pregnancy till < 37 weeksof gestational age.
- Measurement of BP at least “4 times” a day
- Weight measurement alternate day
- Urinary “dipstick evaluation for protein” in the urine first voided in the morning daily
- Complete blood picture for platelet count, AST (Aspartate Transaminase) & ALT (Alanine Transaminase) weekly/two times a week. Coagulation profile isnot required when the platelet count is normal.

- The patient should be questioned about any concerning symptoms, such as a severe headache, discomfort in the right upper quadrant, or frequentnausea and vomiting.
- Daily fetal movement count with NonStress Test (NST) and weekly/biweekly amniotic fluid evaluation
- Antihypertensive treatment

According to NICE clinical guidelines (2010, amended 2019) Should be started at BP 150/100mmHg First line - Oral therapy used like

1. Labetalol given orally 100 - 400 mg every 8 - 12 hours

Alternately Methyldopa or Nifedipine can be given

- If BP below 150/100 mmHg no immediate need for antihypertensives unless markers of severe disease are present
- Persistent high levels >160/110 mm Hg is most common indication fordelivery in women with sever preeclampsia.

2. Hydralazine (10 - 25mg twice a day)

- Commonly used for rapid lowering of elevated blood pressure
- Acts directly on arteriolar smooth muscles to reduce PVR
- Most frequent side effects are decreased uteroplacental perfusion and hyperdynamic circulation indicated by late decelerations in patients with previously normal FHR (Fetal Heart Rate) tracing

3. Methyldopa (250 – 500 mg tid or qid)

- Central and peripheral antiadrenergic action

Role of Glucocorticoids[40N]

- It is recommended, if birth is before 34 weeks, 2 doses ofbetamethasone 12 mg IM .
- Significantly reduces neonatal complications like RDS, intraventricular haemorrhage and death.

Delivery

- Induction of labour should be attempted in women with mildpreeclampsia once pregnancy reaches 37 weeks.(ACOG 2020)
- Frequent monitoring of blood pressure done hourly.
- In suspected situations of fetal growth restriction, continuous electronic fetal monitoring should be carried out.
- 3rd stage of labor managed with oxytocin or prostaglandins to preventpostpartum haemorrhage

MANAGEMENT OF SEVERE PERECLAMPSIA

≥ 34 weeks

- Treat with magnesium sulfate for prevention of seizures
- Antihypertensive administration
- Deliver after stabilization of patient

Indications for Delivery in Women less than 34 weeks who are managed expectantly:

- **Prompt delivery after maternal stabilisation and after single dose corticosteroid therapy**

- Uncontrolled severe hypertension
- Persistent headache, refractory to treatment
- Persistent epigastric pain
- Eclampsia
- HELLP syndrome
- Pulmonary edema
- Placental abruption
- DIC
- Stroke
- Myocardial infraction
- Nonreassuring fetal status
- Fetal demise

- **Delay delivery 48hr if possible to allow corticosteroid for lung maturation :**

- PPROM (Preterm Prematue Rupture of Membranes)
- FGR(Fetal Growth Restriction)
- Oligohydramnios
- "Reversed end-diastolic Doppler flow" seen in Umbilical artery
- Worsening "renal parameters"

≤ 34 weeks

- Keep in high risk antepartum area for intensive fetal and maternalmonitoring isneeded

for blood pressure and urine output, cerebral status is done.

- Symptoms like Epigastric pain/tenderness, Shortness of breath are monitored.
- Antihypertensive administered with an aim to lower SBP between 140 -155 mmHg and DBP between 90 -105 mmHg.
- Corticosteroids will be administered (12 mg betamethasone IM two doses 24hoursapart).
- Delivery should be done at least if possible 12 - 24 hours after second steroid dose

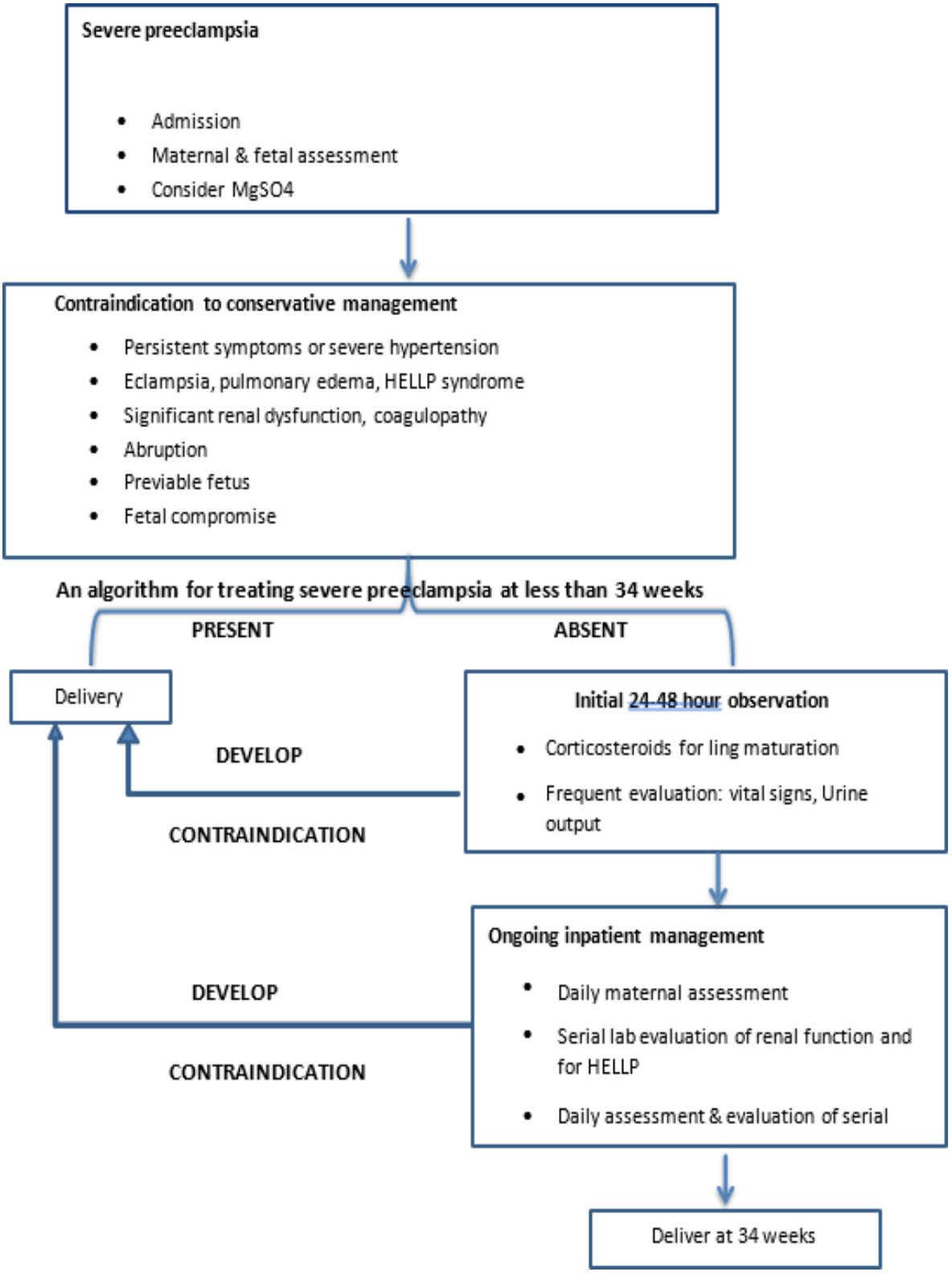


FIGURE NO 12: An algorithm for treating severe preeclampsia at less than 34weeks

Eclampsia

- It is defined as onset of seizures in a pre-eclamptic woman during pregnancy or puerperium which cannot be assigned to other factors and/or an inexplicable comatose state.
- In Eclampsia, maternal mortality ranges from 1 to 5% and about 5–12% of instances involve perinatal death.

Impending Eclampsia:-

It is advised for women who show the signs / symptoms listed below.

- Headaches (occipital/frontal)
- Vision blurriness
- Vomiting and nausea.
- Pain in the right upper quadrant and/or in the epigastrium
- Reduced urine output
- Test results showing DIC

Management[97,98abc]:-

- MgSO4 is used as a treatment and a preventative measure.
- Oral suctioning, airway clearing, and oxygen administration
- Blood pressure management
- Baby delivery

MGSO4 REGIMENS

Pritchard Regimen

- 4 gm of 20% mgso4 iv over not less than 3 minutes

- 20 ml syringe + 4 ampoules of mgso4 + add to 12 ml of NS
- Immediately followed by 10 gm of 50% gmso4 IM (5g in each buttock)
- Maintenance dose → 5 gm of 50% mgso4 IM 4rth hourly in alternate buttocks

Zuspan Regimen

- Loading dose 4 g IV (administered over 5 to 10 mins)
- Maintenance dose 1 -2 g/ hour by controlled infusion pump up to 24hours after last seizure

Sibai Regimen

- 6gm MgSo4 over 20 mins followed by 2 gm MgSo4 IV infusion
- Continuous IV regimen 4 -6 g LD of MgSo4 in 100 ml IVF slowly over 15 to 20 mins followedby 1 - 2 gm/hr in 100 ml of IV infusion as maintenance.

Low dose regimen of SARDESAI

- Loading dose of 4 gm (20 ml in 20% solution) of mgso4 was given IV 4 -6 minutes
- Maintenance does is 2gm of 20% w/v every 3 hours
- Can be given IM route as well

Other anticonvulsants

- Levetiracetam
- Phenobarbitone
- Phenytoin regime
- Diazepam regime
- Lytic cocktail regime

Antihypertensive for urgent control of severe hypertension (ACOG -2020a)

1. **Hydralazine :**

   5mg IV or IM → 10mg (15-20 minutes apart) - Maximum upto 3 doses

   (or)

   Constant infusion 0.5-10mg/hr

   o Side effects – tachycardia, headache, hypotension

2. **Labetalol :**

   10mg→20mg→40mg→80mg (at 10-15 minutes interval)

   (or)

   Constant infusion 1-2 mg/min

   o Side effects – asthma precipitation, bradycardia, hypotension

3. **Nifidipine** : 10mg→20mg at 20 minutes interval for 2 doses

   o Side effects – tachycardia , headache

4. **Diuretics** :

- Women who are eclamptic and have concurrent pulmonary oedema should not beprescribed diuretics during the antepartum phase.
- They may form an integral part of the postpartum care.
- Placental perfusion is compromised with the use of diuretics.
- Women with severe preeclampsia and eclampsia usually have an acute expansion of intravascular volume during delivery due to the substantial amount of IV fluids that

they receive. The auto transfusion that follows the contraction of the uterus during delivery adds additional volume to the intravascular space. Finally, following delivery large amounts of fluid accumulated in the interstitial space start to mobilise towards the intravascular space. This is a perfect setup for congestive heart failure and pulmonary oedema particularly in women with renal function impairment.

- Therefore, before delivery "diuretics are not used to lower blood pressure"
- Furosemide or similar drugs are used before delivery solely to treat pulmonary oedema.
- After vaginal or caesarean delivery, the patient may begin receiving furosemide 20–40 mg IV every 10–12 hours. Once the patient is able totolerate oral intake, the medication can be continued orally for several days.

DELIVERY

- Delivery is the only definitive treatment for eclampsia
- The mode of delivery depends, on multiple factors like period ofgestation, presenting part & cervical examination findings
- Cervix -favourable, options include Artificial Rupture of Membranes (ARM) or labour inductionusing Prostaglandins/oxytocin.
- Cervix - unfavourable with alive fetus, options like caesarean can be considered.
- Caesarean delivery can be necessary for prolonged fetal bradycardia,an unripe cervix, or for a gestational age under 30 weeks.

HELLP SYNDROME

This is a complication of pre-eclampsia characterised by the development of thrombocytopenia (<1lakh/mm^3) with elevated liver enzymes and hemolysis

Diagnosis:

- About 82%–85% of patients with HELLP have mild-to-severehypertension and 85%have significant proteinuria.
- HELLP may have an atypical onset or features with about 15% lack either hypertension or proteinuria.
- "HELLP SYNDROME DIAGNOSIS CRITERIA":

1. Hemolysis

- Peripheral blood smear showing abnormality like burr cells, schistocytes.
- Increased bilirubin levels "≥ 1.2 g/dl"
- Reduced "serum haptoglobin" levels
- Elevated LDH more than "twice the upper limit of normal (>600 U/L)"

2. Elevated liver enzymes

- Elevated AST, ALT "≥ twice the upper limit of normal (≥72 IU/L)"

3. Reduced platelet count (<1lakh/mm^3)

Table no 1: Mississippi classification of HELLP syndrome[99]

Class	Severity of thrombocytopenia	Platelet count/mm^3
I	Severe	<50,000
II	Moderate	50,000 to 1,00,000
III	Mild	100,000 to 150,000

Maternal complications[100]:

- Abruptio placentae with DIC,
- Acute renal failure
- Pulomonary edema

Perinatal complications[100]:

- Prematurity (can lead to Respiratory distress syndrome- RDS, intracranial haemorrhage, necrotising enterocolitis and bronchopulmonary dysplasia)
- Growth restriction and
- Abruptio placentae

Hepatic rupture

- The signs and symptoms are those of significant circulatorycollapse, and it can happen either antepartum or postpartum.
- Intraabdominal bleeding - signs of peritoneal irritation and progressive hypovolaemia will be seen.
- The pregnancy must be terminated immediately.
- At the time of the laparotomy, the laceration is almost always found on the diaphragmatic aspect of the right lobe of the liver. It frequently coexists with subcapsular petechiae and subcapsular haematomas.
- The prognosis for pre-eclamptic patients with liver rupture is ominous.

- Usually Attempts at surgical repair or excision → extension of thelaceration →more bleeding, consumption coagulopathy and ultimately death.
- Least manipulation of the hepatic tissue have better results. The bleeding hepatic surface should be covered with Avitene, Oxycel or Gelfoam and then packed with surgical sponges placed above the haemostatic agent. One of the sponges is brought outside the abdominal incision to facilitate removal on the second or third postoperative day Management
- The clinical course of HELLP syndrome is frequently characterised by a gradual,occasionally rapid, deterioration in the status of the motherand foetus.
- Pregnant Women should be “delivered regardless of their gestational age”because of the dangerous nature of this condition and the elevated rates of maternal morbidity and mortality.
- It few studies it has been shown that corticosteroids due to their anti- inflammatory action and immunosuppressive action it might modify the proinflammatory condition of pre-eclampsia with severe features and may favourably change the clinical course. The risk of maternal death, severe maternal morbidity, and perinatal or newborn death remained unchanged. Improved platelet count was the only way that the treatment affected each patient's specific result. The evidence for corticosteroids being used to slowdown the HELLP syndrome disease's progression is insufficient.
- Investigations should be done at least at “12-hour intervals”.
- AST > 2,000 IU/L or LDH > 3,000 IU/L suggest of increased risk of mortality.

- Platelet count: - Generally decreases at an regular rate of nearly “40% per day”, and liver enzymes levels increased. The lowermost platelet count mostly occurs after delivery at a mean of 23 hours.
- HELLP syndrome might achieve peak condition in the first 2 daysfollowing delivery, with a descending trend in haematocrit values.
- If the platelet count continues to drop with increasing liver enzymes after 4 days of delivery, the initial diagnosis of HELLP syndrome shouldbe reassessed.
- Supportive care alone, 90% of patients will have platelet count > 1,00,000 /mm^3 and are duction in liver enzymes values <7 days followingdelivery.
- Women with HELLP syndrome are also at increased risk of pulmonaryoedema, acute RDS and renal failure.
- Immediate delivery:-
  - If gestational age is ≥34 weeks (or)
  - At any weeks of gestation if there is uncontrollable hypertension,acute renal failure, pulmonary oedema, non-reassuring fetal status, abruptio placenta, severe liver parameters abnormality, bleeding is present.
- All other cases require administration of magnesium sulphate, steroids for the prevention of intraventricular bleed and RDS in the fetus and delivery within 24hours after the second steroid dose.
- Delivery should not be delayed further even if there is some apparentimprovement in the patient situation during the time required for steroidadministration.

- Vaginal delivery - only if the cervix is ripe, the gestational age is >/=32 weeks of gestation, reactive FHR without any indications for caesarean delivery. Labour should proceed rapidly and cervical changes should be seenshortly after the initiation of induction. If vaginal delivery is not foreseen within 12 hours after the onset of induction, it is better to perform caesareansection.
- Platelets are given when the platelet count is below 50,000/mm3 andparticularly if the patient shows signs of altered haemostasis. The plateletcount is raised approximately 10,000/mm^3 by each unit of pooled platelets.
- An upward trend in platelet count and a downward trend in LDH shouldbe apparentby the fourth postpartum day in patients recovering without complications.
- Plasmapheresis -For deteriorating patients despite the conventional therapy.
- A double-blind, placebo-controlled clinical trial with adequate number ofsubjects demonstrated that "dexamethasone treatment does not improve the outcome in women with HELLP syndrome". Outcomes assessed included are"duration of hospitalization , recovery time of abnormal lab results and complications such as acute renal failure, pulmonary oedema, eclampsia and death". In the same study, an unplanned subgroup analysis suggested abeneficial effect in platelet count recovery in women with severe HELLP(platelet count <50,000mm^3)
- At present, corticosteroids are not recommended for the treatment of HELLP.

Complications of pre-eclampsia:

Pulmonary oedema

- Pulmonary oedema is a rather common complication of severe preeclampsia and eclampsia, affecting approximately 3% of thesepatients.
- Mostly due to increased use of crystalloid solutions (for intravascular volume expansion).
- Usually occurs in the postpartum period and is characterised by deep respiratory distress, significant fall in oxygen saturation and on auscultation of chest diffuse ralesare seen.
- Treatment include propped up position, administering oxygen by nasal prongs or a rebreathing mask, restriction of intravenous and oral fluids andfurosemide 20–40 mg IV every 6 hours. Central haemodynamic monitoring (central venous pressure of Swan– Ganz catheter) has been recommended by the ACOG (2002a) only in severely pre-eclamptic women with accompanying severe cardiac disease, renal disease or both or in cases of refractory hypertension, oliguria and pulmonary oedema.

Acute renal failure

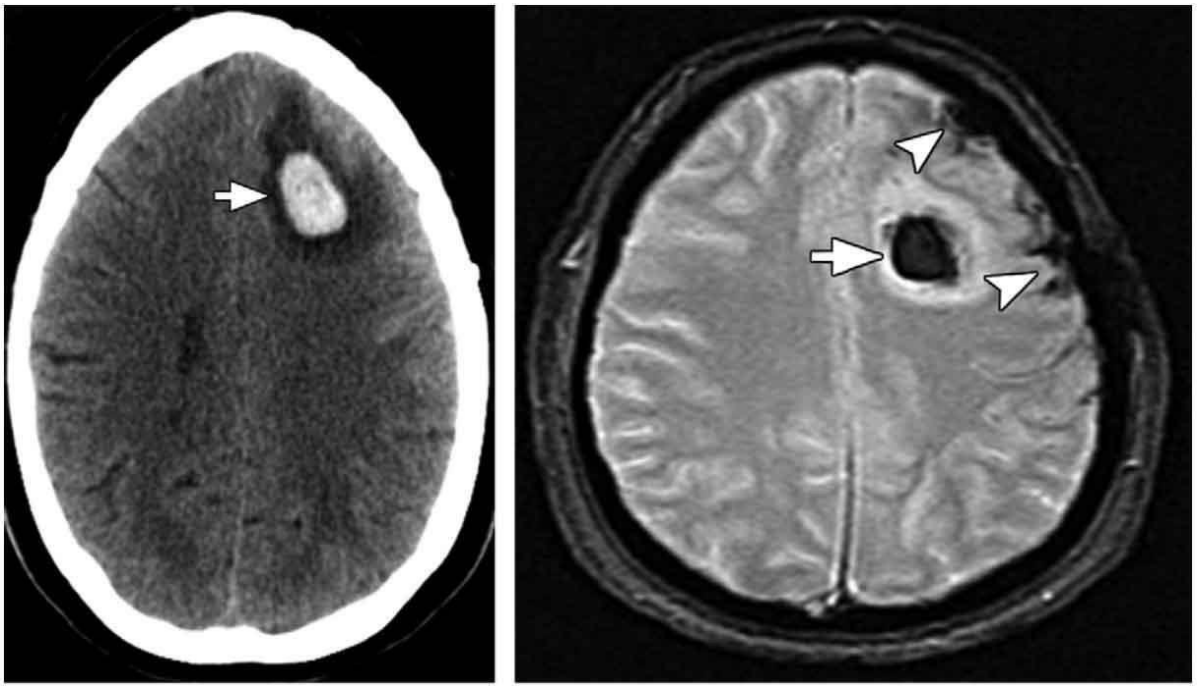
- In severe pre-eclampsia Oliguria is mostly due to prerenal in origin.
- The majority develop oliguria are volume depleted and they usually respond to an increase in the rate of intravenous fluid administration. Occasionally, some patients do not respond to the fluid challenge and it is necessary to make a rapid assessment ofthe pathophysiology of the process.
- In patients with uncontrolled hypertension with raised haematocrit reflecting haemo-concentration need aggressive treatment with vasodilatorsto effect afterload reduction

and decrease renal artery vasospasm.

- Normotensive or mildly hypertensive women with low haematocrit values have expanded intravascular volume and need aggressive diuresis.
- In older and obese patients, there is large increase in plasma volume with normal or decreased cardiac output. These women are at significant risk of pulmonary oedema and require fluid restriction and aggressive preload reduction with diuretics.
- In other patients, there is a contracted intravascular volume due to low plasma oncotic pressure and endothelial cell damage with leaking of seruminto the interstitial space. These women usually respond to interruption of pregnancy and expansion of intravascular volume.
- Rarely, oliguria is of renal origin and commonly due to Acute TubularNecrosis (ATN) that occur in pre- eclampsia setting complicated with severe abruption, DIC. many patients may require dialysis. The remote prognosis of properly managed ATN in patients with pre- eclampsia is good. Establishment of adequate urinary output is an important priority because the longer the low urinary output persists, the greater the possibility that the patient will develop severe or irreversible renal damage.
- If vaginal delivery cannot be anticipated to occur in a few hours, it isbetter to performa caesarean section.
- Usually delivery is followed by disappearance of the renalvasospasm and briskdiuresis.

Intracranial bleeding

- It is the foremost cause of mortality in pre-eclampsia.
- Underestimation of severity of the disease, extended outpatient treatment, failure to use antihypertensive drugs to treat extreme elevationsof BP and discharge from the hospital before obtaining adequate control ofthe hypertension is the most frequent cause of deaths.
- An important clinical observation from the analysis of 28 women who sustained a stroke in association with pre-eclampsia and eclampsia was thatthe main correlation of this event was the systolic, not the diastolic BP[101]. This suggests that antihypertensive therapy may be indicated when the systolic BP reaches 150, not 160mm Hg, in pre-eclamptic women.
- The diagnosis is suggested by a deepening stupor and sensorimotor deficits and becomes highly probable if focal neurologic signs, such asunilateral pupil dilation,are present.
- By CT or MRI scan the diagnosis is confirmed.
- Recovery is more of an exception than the norm, and the prognosis is very bad.
- In most cases, coma becomes more profound, respiratory paralysisappears andfinally the electroencephalogram shows loss of electrical activity.
- Severe occipital and temporal headaches are important symptoms inpregnant patients because they are frequently harbingers of convulsions.
- These headaches are usually secondary to inadequate BP control andthey are an indication for aggressive treatment with hypotensive agents



CT showing intracranial haemorrhage with surrounding edema in left frontal lobe	MRI showing Arrow – frontal lobe hemorrhage Arrowheads - SAH

FIGURE NO 13. Visual disorders[38]

- Blindness may happen in patients with severe PE and eclampsia and may persist for several days, although quick recovery after delivery is therule.
- In most cases, examination of the optic fundi does not show severeretinopathy, since the problem is usually caused by multiple micro haemorrhages and micro infarcts occurring in the occipital lobe.
- Cortical blindness is equivalent to a seizure, and patients with these symptoms should be treated as having eclampsia.
- The funduscopic examination of patients with pre-eclampsia usuallydoes not reveal more than focal or generalised vasospasm and, in some cases, retinal oedema, which

frequently is missed in the examination because it begins in the periphery of theretina.

- Papilloedema in pre-eclampsia is highly unusual and demands a re- evaluation to rule out the possibility of an intracranial tumour or bleeding.
- Diplopia is a symptom that may occur, and it is caused by functional impairment of the sixth cranial nerve paralysis. This finding requires a CT scan to rule out a tumour in the brainstem area. Like most lesions caused bypre-eclampsia, the sixth nerve paralysis improves after delivery and eventually disappears several weeks later.

Abruptio placentae

- About 7% of all patients with eclampsia will have abruption.

PREECLAMPSIA SCREENING TESTS FOR PREDICTION AND PREVENTION

I. **Modification of the placental perfusion and vascular resistance functions**

- Doppler ultrasonography,
- The rollover test, and mean arterial blood pressure
- Testing for angiotensin II binding,
- Isometric exercise testing,
- Angiotensin infusion tests
- Serum renin levels
- The response of platelet calcium to Argipressin,
- Monitoring of ambulatory BP for 24 hours

II. **Changes in the fetal placental unit function**

- Inhibin A levels,
- The Alpha fetoprotein (AFP), and
- HCG
- Reduction in plasma protein A related with pregnancy
- Serum Estriol levels

Renal parameter changes

- Raised Serum U.A;
- Elevated microalbuminuria levels.
- Urinary presence of kallikrein
- Increased micro transferrinuria levels.

IV. Changes in endothelial and oxidative stress function

- Raised Fibronectin levels
- Raised Homocysteine
- Raised Endothelin
- Thromboxane is raised

The different predictors can be broadly divided into two categories: non-laboratory approaches an1.d laboratory approaches.

NON-LABORATORY APPROACHES:

History :- "High risk factors for PE" include

- Primigravida
- Maternal age extremes

- Overweight or obese

Preeclampsia-complicated prior pregnancy;

- Multifetal pregnancy;
- Ethnic group

2. **Provocative Pressor tests include**:

a. **Test with Angiotensin II infusion** -

- Between 28 and 30 weeks, this test is performed.
- Preeclampsia is predicted by angiotensin infusion test with sensitivity of 90%, specificity of 87%, and 78% PPV in high-risk individuals whenthe diastolic blood pressure rises by more than 20 mmHg.
- Drawback:
  - ❖ Difficult to complete as a mass screening technique
  - ❖ Costly
  - ❖ Time-taking
  - ❖ Not dependable

b. **Roll over Test:-**

- Performed between 28 to 32 weeks period of gestation
- With the patient in the left lateral posture, the blood pressure is first measured.

  A positive test is considered to occur when the patient's diastolicblood pressure rises by more than 20 mmHg when lying supine.
- Sensitivity is <88%, Specificity is of 5-95% and Positive predictive value is <93%.

- The substantial range in results makes this test useless for clinical usage.

c. **Isometric handgrip test[102]:-**

- Performed between 28 to 32 weeks period of gestation
- Patient is advised to squeeze a hand ball for about 3 minutes, it is suggested that their diastolic blood pressure threshold rises by20 mmHg.
- Has Sensitivity of 81% and Specificity of 96% with Positive predictive value of 81%

d. **Mean Arterial pressure**:-

- Patients who have mean arterial pressure in the second trimester greater than90 mmHg are at increased risk for pre-eclampsia.
- "The predictive value varies widely".

USG Doppler:-

- Pre-eclampsia can be detected as early as 18 weeks in apregnancy using uterine and umbilical artery Doppler velocimetry.
- In patients who are at risk of developing pre-eclampsia, the diastolic waveform notches characteristically, indicating higher peripheral resistance as a result of inadequate trophoblastic invasion of spiral arterioles.
- It has Sensitivity of 78% and Positive predictive value of 28%

LABORATORY APPROACHES[103]:-

1. **Foetal placental unit: – indicating endocrine dysfunction**

- Placental protein 13,
- Alpha Fetoprotein(AFP),

- Estriol levels
- Pregnancy associated plasma protein A
- Inhibin A and
- Activin A levels
- Corticotrophin-Releasing Hormone(CRH)
- hCG.

2. Endothelium dysfunction indicators

a. Serum Fibronectin levels

b. Inhibitors of plasminogen activator,

c. Cell adhesion molecules,

d. Serum thrombomodulin,

e. Endothelin-1

f. Coagulation factors, platelets

g. Uric acid levels

h. Atrial natriuretic peptide,

i. Haematocrit

3. Urinary Tests:

a. Microalbuminuria: - sensitivity of 7%-90% and specificity of 29%-97%.

b. calcium excretion[104]

c. urine calcium[105] _____

creatinine

d. Allikrein creatinine

e. Fasting urinary albumin and creatinine ratio

4. Angiogenic factors:-

a. Decreased levels of VEGF and placental growth factors, which are pro-angiogenic factors

b. An increase in the antiangiogenic molecules "sFlt-1 and sEng"

5. Cell free fetal DNA

- Maternal plasma may contain cell-free DNA (cfDNA) from the placenta. Apoptosis of cytotrophoblasts is thought to be increased in preeclampsia which releases cfDNA.
- The overall cfDNA levels and preeclampsia prediction were not correlated according to an MFMU (Maternal Fetal Medicine Units) Network study.

6. Others :-

- Other markers under investigation include first-trimesterestimated placental volume, serum cystatin-c levels, and glycosylated HbA1c.
- Serum and urine proteins as well as cellular metabolites can be studied using proteomic, metabolomic, and transcriptomic technologies. preliminary research shows they have potentialpredicting ability

PLATELETS:-

- Preeclampsia includes thrombocytopenia and platelet dysfunction as essential

symptoms. Platelet activation results in increased destruction of the cells and decreased blood levels. Platelet volume increases as a result of immaturity. Preeclampsia has been said to beearly predicted by platelet volume.

SERUM URIC ACID:

- Because of reduced clearance, preeclampsia has higher serum uric acidcontent. The severity of the illness and the prognosis for the fetus are related to serum level. The increase in serum levels happens somewhat latein the disease's progression. As a result, unreliable as a predictor.
- Specificity ranged from 77 to 95%, and sensitivity from 0 to 55%.
- Raised serum uric acid is generally better understood as a sensitive signof decreased renal function rather than a predictive, diagnostic, or unique feature of preeclampsia.

SERUM FIBRONECTION LEVELS[8N]:

- The glycoprotein known as fibronectin is crucial for “cellular adhesions, migration, phagocytosis, and homeostasis”. It is found in connective tissueas well as the basement membrane. Endothelial cells and extracellular matrix release it into the bloodstream after endothelial damage. It has beensuggested that cellular fibronectin levels greater

than “3.8 ug/mL within 22to 26 weeks of gestation” may aid in the early diagnosis of preeclampsia inprimigravida. There were discrepancies in the sensitivity, specificity, and positive and negative predictive values between investigations. According to a systemic review, “neither cellular nor total fibronectin were clinically helpful in predicting PIH”.

RAISED HOMOCYSTEINE LEVELS:

- Homocysteine is reported to be increased in preeclampsia and causes oxidative stress, endothelial cell dysfunction, and other problems. Although there was a 3–4 fold increased risk of preeclampsia in pregnant women withelevated levels “at 14–16 weeks”, the findings have not been consistent.

SERUM INHIBIN-A and ACTIVIN-A:

- It's unclear how they contribute to the development of preeclampsia. They are produced by the placenta's trophoblast cells, and their levels reachtheir peak at 8 weeks before dropping off till term. They exhibit placental bed formation with trophoblast invasion. Patients who later develop preeclampsia experience an increase in maternal serum levels between 13 and 18 weeks.

ALPHA-FETOPROTEIN (AFP):-

- AFP in the maternal serum rises up until 30 weeks of pregnancy. Numerous studies have proven that there is a link between increased maternal AFP and hypertensive disorders of pregnancy. At 12 weeks ofgestation, AFP peaks at 3 mg/ml and then starts to fall.

ANGIOGENIC & ANTI-ANGIOGENIC FACTORS:-

- The formation of placental vascular tissue involves a number of proangiogenic and antiangiogenic components. In preeclampsia, factors like placental growth factor (PLGF) and (VEGF) are diminished. Early pregnancy doesn't typically show this distinction. A study showed that placental growth factor is a poor predictor of the development of severe preeclampsia later on. "Worsening hypoxia atthe uteroplacental interface stimulates excessive levels of antiangiogenic factors". At least two antiangiogenic peptides are overproduced by the trophoblastic tissue of pregnant women who will eventually develop preeclampsia.

i. Soluble Fms-like tyrosine kinase (sFlt-1) functions as a receptor for (VEGF) and (PLGF).

ii. Endoglin is a TGFβ co-receptor. Endothelial nitric oxide dependentvasodilatation is reduced as a result of the inhibition of TGFB binding toendothelial receptors. There is still no known reason for the placental overproduction of antiangiogenic proteins.

iii. Before the development of a clinical condition, "soluble endoglin and soluble fms like tyrosine kinase 1 (SFlt-1) are elevated". Their clinical effectiveness is not advised until it is more clearly demonstrated.

Prevention:-

Preeclampsia prevention is a topic with a large body of literature. Instead thanfocusing on preventing preeclampsia's sequelae, there is some debate about whether or not preventing preeclampsia per se is a worthwhile objective.

Primary Avoidance:

- Primary avoidance, while ideal, is lone feasible when the precise etiology is understood. By changing some of the risk variables, primaryprevention is some what attainable.
- Better To have children at an "age when the endothelium is still ableto handle the inflammatory stress associated with the pregnancy state".
- It is advised to have pregnancies with "low risk men", more common in "nulliparous"women and to stay with the same partner because the disease process more common in "mutliparous women with change of partners".
- Preeclampsia may occur less frequently if obesity is prevented and/or effectively controlled.
- Women with conditions including "diabetes, chronic hypertension, renaldisease, and others" should have their underlying illness under control before trying to get pregnant.

Secondary prevention:

Secondary prevention's fundamental criteria are

a. comprehension of pathophysiological processes
b. The accessibility of screening techniques
c. Techniques for modifying and intervening with pathophysiology

Non-pharmacological methods of interventions

a. Bed rest;
b. alterations in lifestyle
c. Consistent physical activity

Dietary Modifications

a. Limiting salt in the diet
b. Nutritional protein and energy intake
c. Management of obesity
d. A alteration in food habits
e. Omega-3 fatty acids found in fish oil have been demonstrated in few trials to be helpful in preventing preeclampsia. Fish oils are unlikely to be helpful in preventing preeclampsia, according to atrial (FOTIP).
f. Consumption of alcohol.
g. Supplementing with arginine was reported to be advantageous,however the research was limited.
h. Toki-shakuyuku-san (TS), a Japanese herbal remedy, possibly will be helpful in

management and prevention of PE.

Pharmaceutical Involvements

a. Hypertension medications.

b. Diuretics like furosemide

c. Supplementing with zinc.

d. Magnesium Sulphate

e. There is no evidence to support the benefits of folic acid or any otherB vitamin in preventing preeclampsia.

f. Low-dose aspirin:

- Treatment with "low-dose aspirin (50–150 mg/day)" duringpregnancy reduces the production of platelet TX-A2 with only minor effects on prostacyclin.
- ACOG (2020) recommendations : low-dose aspirin should be given between12 and 28 week period of gestation to help preventpreeclampsia in:-

❖ Women with one or more of the following high risk features:

- Previous preeclampsia
- chronic hypertension
- overt diabetes mellitus
- any renal disorders,
- autoimmune diseases,
- Multifetal pregnancy.

❖ Supplementation can be considered in women with

>1 of these equalities:

-Nulliparity

-Age > 35 years,

- Obese women
- Family history of PE
- Vulnerable sociodemographic,
- Previous low-birthweight or growth-restricted baby.

- The CLASP (collaborative low dose aspirin) research is the largesttrial to date. Overall, there was a 12% decrease in the incidence ofpreeclampsia when low dose aspirin was used (non-significant). Women who use aspirin have a slightly greater risk of placenta abruption (Statistically Not Significant). Antiplatelet medications used to prevent preeclampsia did not vary from the control groupin a meta-analysis. The outcomes of the current trials do not support the usual therapeutic or preventive use of aspirin therapyfor pregnant who are deemed to be at risk for PE. Women who face the risk of developing preeclampsia with early onset are theonly population in which low dose aspirin may be indicated.

g. Only women with antiphospholipid antibody syndrome should take heparin and low-dose aspirin; they should not be recommended on aregular basis.

h. Calcium supplement: Calcium consumption and the likelihood of preeclampsia are inversely correlated. Levin d's study, which involved supplementing with 2g/day, revealed no advantages. The Cochrane study did find a slight decrease in preeclampsia, with the benefit beinghighest in high-risk mothers who consume little calcium.

i. Nitric oxide donors – preeclampsia affects Nitric oxide production, Dataon how NO donors affect preeclampsia prevention are scarce and contradictory.

j. Antioxidants like Vitamin - C, E, lycopene, selenium, and garlic, have been utilised in numerous trials with positive outcomes. Antioxidant supplements, however, may not have an impact on therisk of preeclampsia or clinical outcomes according to a 2008 Cochrane analysis.

Exercise

Cardiovascular medications:

a. Diuretic drugs

b. antihypertensive

Anti-oxidants like Vitamins C, E, and D[106,107].Anti-thrombotic medications[108,109]:

a. "low-dose aspirin"

b. "Aspirin with Dipyridamole or heparin or ketanserin"

AIMS AND OBJECTIVES

1) To study the echocardiographic changes in pregnant women with HDPs compared to normal pregnant women.

2) To study the correlation between Echocardiographic abnormalities and fetal and maternal outcomes of pregnant women with HDPs with normal pregnant women

MATERIALS AND METHODS

A. **Study Design**: Prospective observational study

B. **Study Period:** September 2022 – April 2024

C. Study Area:

The study was conducted in women between the age group 18 -35yrs with HDPs with a normal singleton pregnancy at 28 to 42weeks gestation admitted to the Department of Obstetrics & Gynecology, Shri B.M. Patil Medical College Hospital and Research Centre,

B.L.D.E. (DEEMED TO BE UNIVERSITY), Vijayapura, Karnataka, India

D. **Sample Size:** 142

E. Sample Size Calculation:

Using G*Power ver. 3.1.9.4 software for sample size calculation, The Echocardiographic parameter LVESV (mL) Cases (Mean=34.67, SD=10.12) and Control (Mean=29.27,SD=7.25), this study requires a sample size of 142 for each group (i.e., a total sample sizeof 71, assuming equal group sizes). So to achieve a power of 95% for detecting a difference means (t-tests - Means: Difference between two independent means (two groups)) with a 5% level of significance.[3]

F. Method of Collection of

DataInclusion Criteria

- Women with a normal singleton pregnancy from 28 weeks to 42 weeks period of gestation.
- Women with gestational hypertension (B.P.≥140/90mmHg after 20 weeks of pregnancy) from 28 weeks to 42 weeks period of gestation.
- Women with mild preeclampsia (gestational hypertension with proteinuria)
- Women with severe preeclampsia (gestational hypertension with proteinuria withend-organ damage)
- Women with imminent eclampsia (preeclampsia with imminent signs) from 28weeks to 42 weeks period of gestation.

Exclusion Criteria

- Women who are not consenting to the study.
- Women with chronic hypertension.
- Women with eclampsia.
- Women with Gestational age less than 28 weeks.
- Women in active labor.
- Multiple pregnancy, severe anemia patients.
- Known cardiac disease (e.g., structural heart disease, coronary heart disease, cardiomyopathies, etc.
- Connective tissue disorders.
- Renal impairment.
- Diabetes mellitus.

Institutional Ethical Clearance was obtained from Shri B.M. Patil Medical College Hospital

andResearch Centre, B.L.D.E. (DEEMED TO BE UNIVERSITY), Vijayapura, Karnataka, India. Written informed consent was obtained from all patients before the collection of data.

G. Data Collection Procedure

1) All singleton pregnant women from 28 weeks to 42 weeks gestation meeting the inclusioncriteria were screened for hypertension by measuring their blood pressure using a standardauscultatory method with the help of a mercury sphygmomanometer. Systolic and diastolicblood pressure was measured in the sitting position in the right arm at the level of the heart.[6] Heart rate is measured along with mean arterial pressure.

2) The normotensive patients were divided into one group which served as controls, and a departmental Echocardiography was performed.

3) The patients with B.P.> 140/90mmHg on two occasions 4 hours apart were considered hypertensive and were divided into another group serving as cases.

4) Bed bedside urine albumin test was done by taking 2ml urine with 2ml of sulphosalicylic acid added and checked for cloudiness to differentiate gestational hypertension from preeclampsia and as a routine protocol, all basic investigations and departmental ECHO were performed.

4) In patients who are in early pregnancy (28 weeks to 32 weeks), repetition of 2DECHO wasperformed in regular intervals.

5) Data was compared in terms of echocardiographic findings and structural anomalies on echocardiography in normal pregnant women with HDPs.

6) Data was also compared in terms of complications of maternal outcomes like Congestive Cardiac Failure (C.C.F.), Atrial fibrillation, hypertensive crisis,

cardiomyopathy, Intensive Care Unit(I.C.U.) admissions, maternal mortality, and complications of fetal outcomes like Intra Uterine Growth Retardation (I.U.G.R.), preterm, Neonatal Intensive care Unit (NICU) admissions, Intra Uterine Deaths.

7) A cardiologist's opinion was taken in patients with abnormal echocardiographic findings, and a decision regarding the plan of treatment was suggested accordingly.

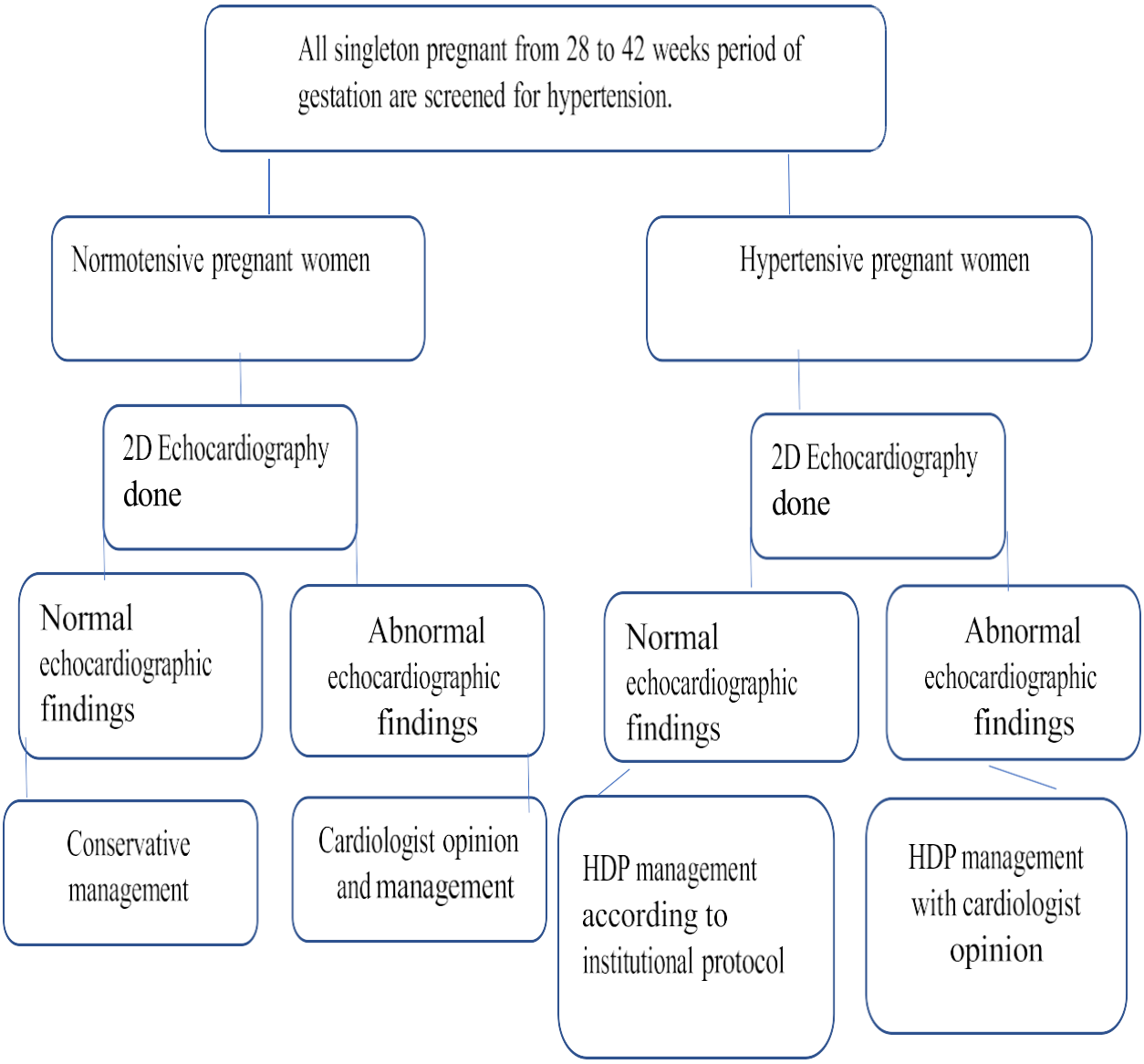


Figure no. 14 Systemic flowchart of methology

2D Echo Analysis:

Echocardiograms were performed with the GE Vivid T8 ultrasound machine (GE

Healthcare,USA), using the cardiac sector probe 3Sc-RS (1.3-4.0 MHz) with the capability of the M-

mode, 2D, Doppler, transesophageal echocardiogram (TEE), and strain analysis.

The measurements of echocardiographic parameters were conducted in accordance with the guidelines provided by the European Association of Cardiovascular Imaging and the American Society of Echocardiography. The previously mentioned guidelines were followedfor analysing echo parameters; in the HPD cases and normal pregnancy**.**

The following echo parameters were measured: TAPSE (tricuspid annular plane systolic excursion); left atrial dimension, area, and volume; right atrial area and volume; E/A ratio; tricuspid regurgitation jet velocity; left ventricular ejection fraction; left ventricular end-diastolic and end-systolic dimension; right ventricular dimension and wall thickness. The tricuspid regurgitation jet velocity (TRV), or the retrograde blood flow over the tricuspid valve during systole, was measured in order to determine the pulmonary artery systolic pressure. TRV calculates the systolic pressure of the RV and correlates it to the pressure in the pulmonary artery. A cutoff point of 2.5 m/s was selected for the TRV.

Brief Procedure of 2D Echo:

1. The patient lies on a table, and the chest region was covered with gel.
2. A transducer that transmits high-frequency sound waves was applied to the chestregion by the sonographer or technician.
3. The sound waves reflect off the heart and pass through the chest, painting an imageof the anatomy and operation of the heart.
4. The pictures were recorded for later examination and shown on a monitor.

The overall process takes thirty to forty-five minutes. and following the procedure, the patient was get back to their usual activities immediately.

Figure no.15 Picture of 2Dechocardiography machine

Figure no.16 Picture of Echocardiogram room

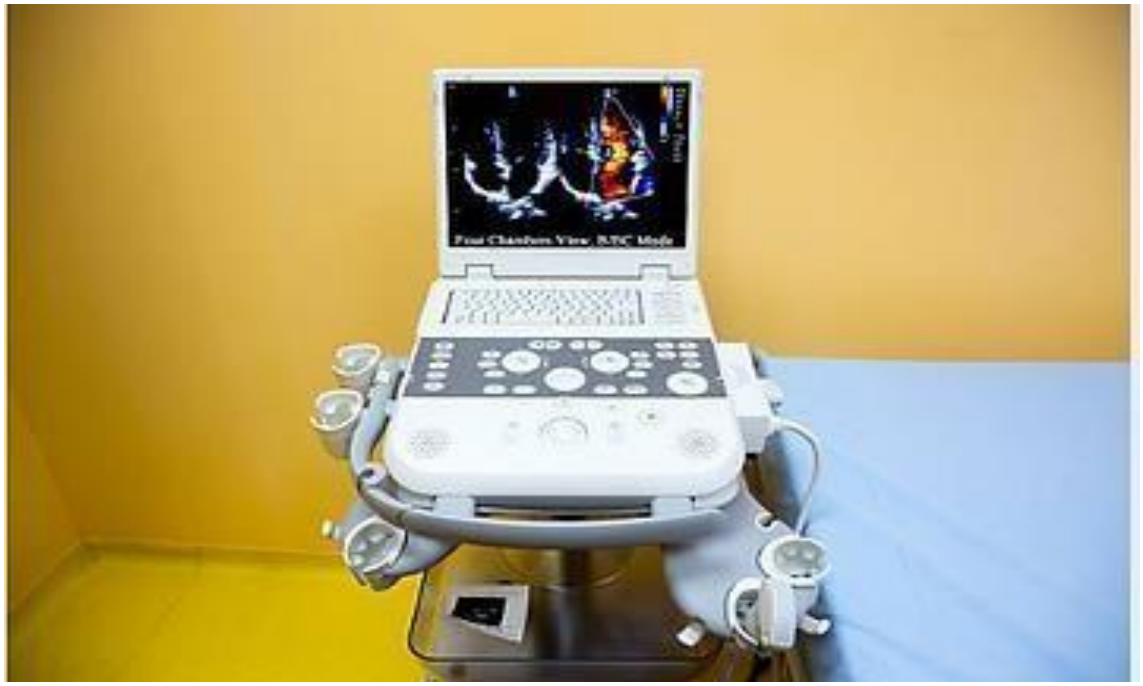


Figure no. 17 GE Vivid T8 Ultrasound Machine

Statistical Analysis

The statistical analyses are performed using a statistical package for the social sciences (S.P.S.S.) (Version 20). Results are presented as Mean, SD. For normally distributed continuous variables an independent sample t-test. For not normally distributed variables, the Mann- Whitney U test is used. Categorical variables Chi-square test/Fisher's exact test. If $p<0.05$ will be considered statistically significant. All statistics are performed two-tailed.

REVIEW OF LITERATURE

Alhuneafat L et al. (2024) identified women who underwent echocardiography during or after their first year of pregnancy within the integrated health network. These women were younger than eighteen, had pulmonary embolism, cancer, autoimmune conditions, and structural heart problems. The SPE group showed significant findings, with higher adjusted LV mass index and E/e' than controls. The most common signs of abnormal diastolic filling and LV remodeling in SPE and PRE are associated with HDP. During or after pregnancy, echocardiography may be used to identify these abnormalities in these high-risk expectant mothers.[110]

Giorgione J et al. (2022) study examined 13 women with HDP who underwent two transthoracic echocardiographic (TTE) examinations before and during the early postpartum period. The findings showed that maternal hemodynamic changes did not significantly affect peripartum TTE indices in HDP women, suggesting that suboptimal findings may be due to chronic pregnancy cardiovascular load changes or pre-existing cardiovascular impairment[111].

Giorgione V et al. (2022) studied thirty women who are HDPs diagnosed and

who received two successive transthoracic echocardiographic (TTE) exams before giving birth and in the early postpartum period were included in a prospective longitudinal research. They discovered that 70% of the patients had Left Ventricular (LV) concentric remodeling or hypertrophy. The LV mass indexand relative wall thickness were not significantly different before and after delivery. With identical left-atrial volume, lateral E′, and E/E′ ratio before vs. after delivery, the LV diastolic function did not show any peripartum fluctuation. The systolic function indicators, which included the global longitudinal strain and theLV ejection fraction did not alter from pre- to post-delivery. Their findings demonstrated that in women with HDP, peripartum TTE indices were not significantly impacted by maternal hemodynamic alterations related to delivery. Chronic pregnancy cardiovascular load alterations or pre-existing maternal cardiovascular impairment are likely to be the cause of suboptimal maternal echocardiographic findings in HDP. The long- term maternal cardiovascular disease legacy of HDP may be connected to the severity and persistence of myocardial dysfunction during the postpartum phase

[112].

Vernekar et al. (2021) undertook a prospective study involving 300 cases of hypertensive disorders of pregnancy[113]. Echocardiographic parameters were compared between hypertension instances, and their effect on the outcome for

thefetus and mother was investigated. 14% of the women exhibited concentric left ventricular hypertrophy, 5% had grade II diastolic dysfunction, and 13% had Grade I diastolic dysfunction. Preeclamptic women were more likely to experience diastolic dysfunction. LV ejection fraction was less than 60% in 9.3%of the women. Of the newborns, 25% needed to be admitted to the NICU, and 55% weighed less than 2.5 kg. Knowing the anatomy and physiology of the heartin women with hypertension is crucial for prompt diagnosis, improved treatment,and a favorable prognosis. A useful, safe, non-invasive method for risk stratification and management guidance is echocardiography [113]

Parikh P et al. (2021) conducted a retrospective study and analyzed 150 cases ofpregnant women with hypertension, focusing on the impact of structural and functional abnormalities on fetal and maternal outcomes. The research revealed that there were more abnormal echocardiographic values in a preeclampsia case including lowered fraction of Left Ventricular Ejection Fraction, decreased stroke volume, and increased Left Ventricular mass. Echocardiography is a valuable toolfor stratifying risk and managing pregnant women with gestational hypertension,chronic hypertension, and preeclampsia. Early changes in cardiac function and morphology correlate with disease severity and adverse outcomes, suggesting that studying cardiac structure and function can improve maternal and fetal outcomes[114].

Calabuig AM et al. (2021) carried out a cross-sectional investigation on Gestational Diabetes Mellitus(GDM)- affected women and controls who were measured between 26 and 40 weeks of pregnancy. Every woman had an echocardiogram, and the left atrium, right ventricle, and left ventricles 3D volumes were measured. There were 246 controls and 123 GDM-afflicted womenin the study population. In comparison to the control group, the GDM women hada higher body mass index, a higher systolic blood pressure, and an older age. AllGDM-afflicted women had satisfactory glycemic control. The global longitudinalstrain of the left ventricle was lower in GDM-affected women than in controls. Ejection percent, left ventricular mass, diastolic function as determined by left atrial strain, and 3D functional indices did not significantly differ across the groups. Women with GDM exhibit less distortion of the left and right ventricles than women with an uneventful pregnancy. 3D echocardiography volumetric assessment does not yield further information regarding the heart function of themother. One sensitive echocardiographic method for identifying early functionalabnormalities in the heart in women with GDM is strain imaging [115].

Badenoosh B et.al.(2021) study found a significant relationship between diastolic disorder, systolic artery pressure, and T.R. gradient in women with latepreeclampsia, but not with other echocardiographic variables. The research indicates that elevated T.R. is a significant echocardiography finding in late

preeclamptic patients[116].

Parikh PM et al. (2020) retrospective analysis examined 150 cases of hypertension during pregnancy[7]. Data was analyzed to compare the complications observed in hypertensive women with normal echocardiogram scans to those who had structural and functional abnormalities in the examination. The key Echocardiographic parameters were compared between hypertension condition and their influence on the outcome for the fetus and mother. Preeclampsia was associated with a higher incidence of abnormal echocardiographic parameters than gestational hypertension or chronic hypertension. These characteristics include reduced stroke volume in 28.6% of cases, increased left ventricular massin 26.6% of cases, and decreased left ventricular ejection fraction in 10% of instances. They concluded that preeclampsia affects the heart more than gestational hypertension and that early onset, severe illness exhibits the most marked alterations. Better outcomes for the mother and fetus can be achieved byresearching the anatomy and function of the heart in the first trimester [117].

Mostafavi A., et al., (2019) compared healthy pregnant women and those with preeclampsia, focusing on LV strain using 2D speckle-tracking echocardiography. The findings indicated that preeclampsia patients had significantly higher LV end-diastolic, LV end-systolic, and right ventricular dimensions. The study concluded that preeclampsia development is associated

with increased right and left ventricular diameters and decreased ventricular systolic function, as evidenced by a decline in global circumferential strain[118]

Levine L et al. (2019) carried out a prospective blinded longitudinal cohort study in which pregnant African American women with preterm (< 37 weeks) preeclampsia with severe features were compared to normotensive pregnant women (controls) who were matched for race, gestational age, maternal age, and body mass index. Transthoracic echocardiograms were performed on patients and controls both at the time of diagnosis and again between 4 and 12 weeksafter delivery. Results: At the time of preeclampsia diagnosis, there was more abnormal cardiac function as in preeclampsia cases compared to controls. These findings persisted for 4–12 weeks postpartum. There were additional notable abnormalities postpartum. Conclusions: It wasobserved that there were significant cardiac function disparities amongAfrican American women, lasting into the postpartum period, betweenthose with severe preeclampsia and healthy pregnant women[119]

Zaman N et al. (2018) study compared the systolic and diastolic parameters of echocardiography in thirty pregnant women with preeclampsia with thirty women with a normal singleton pregnancy at 34 weeks gestation. To determine the circumferential and global strain, strain imaging was used. According to the study's findings, preeclamptic women differ significantly from pregnant

women with normal blood pressure in terms of systolic and diastolic dysfunction. Identification of the high-risk populations may be aided by early evaluation of these parameters[120]

Timpka S et al.(2016) in their prospective birth cohort study analyzed offspring who underwent echocardiography using linear regression and investigated the potential associations between offspring's cardiac shape and systolic/diastolic function and hypertensive diseases of pregnancy[3]. They also studied the rate of change in the mother & and SBP and DBP during pregnancy (gestational age of 8–18, 18–30, 30-36, and 36 weeks or more) related to the outcomes of the offspring using multilevel linear spline models. Greater relative wall thickness was linked to exposure to gestational hypertension and maternal preeclampsia. Additionally, a lower leftventricular end-diastolic volume (−9.0 mL; −15 to −3.1) was linked to preeclampsia. Investigation revealed no correlations between hypertensive problems during pregnancy and the heart function of the progeny. Greater infant left ventricular end-diastolic volume, left ventricular mass indexed toheight2.7, and E/A were all positively correlated with the mother's positive rate of systolic blood pressure change between weeks 8 and 18[121]

RESULTS

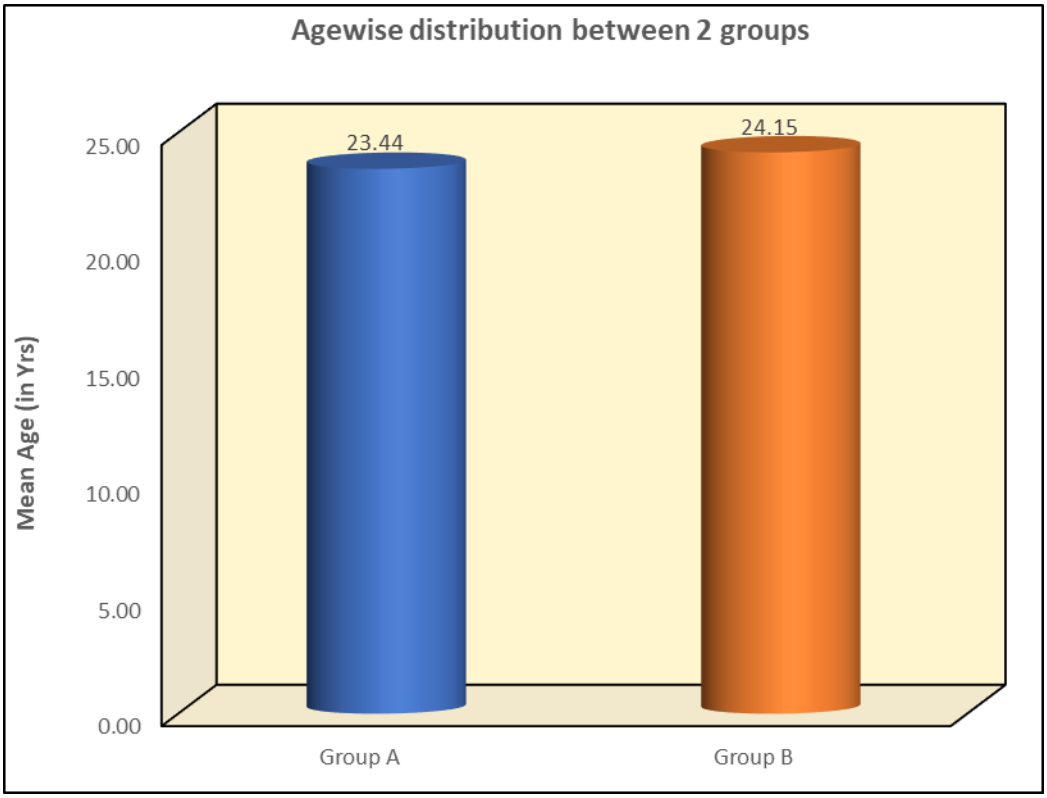
TABLE NO.2 Baseline Characteristics between Hypertensive & Normotensive Pregnant Women						
Variable	Category	Group A		Group B		p-value
		Mean	SD	Mean	SD	
Age	Mean	23.44	3.90	24.15	4.40	0.31[a]
	Range	18 - 36		18 - 37		
Gestational Age	Mean	37.39	2.53	37.82	2.21	0.29[a]
	Range	31 - 42		31 - 41		
		n	%	n	%	
Obstetric Scores	Primigravida	34	47.9%	27	38.0%	0.24[b]
	Multigravida	37	52.1%	44	62.0%	
Smoking	Yes	0	0.0%	0	0.0%	..
	Nil	71	100.0%	71	100.0%	
Race / Ethnicity	Asian	71	100.0%	71	100.0%	..
	Others	0	0.0%	0	0.0%	

Note: a. Mann Whitney Test & b. Chi Square Test

Group A: Hypertensive Pregnant Women & **Group B:** Normotensive Pregnant Women

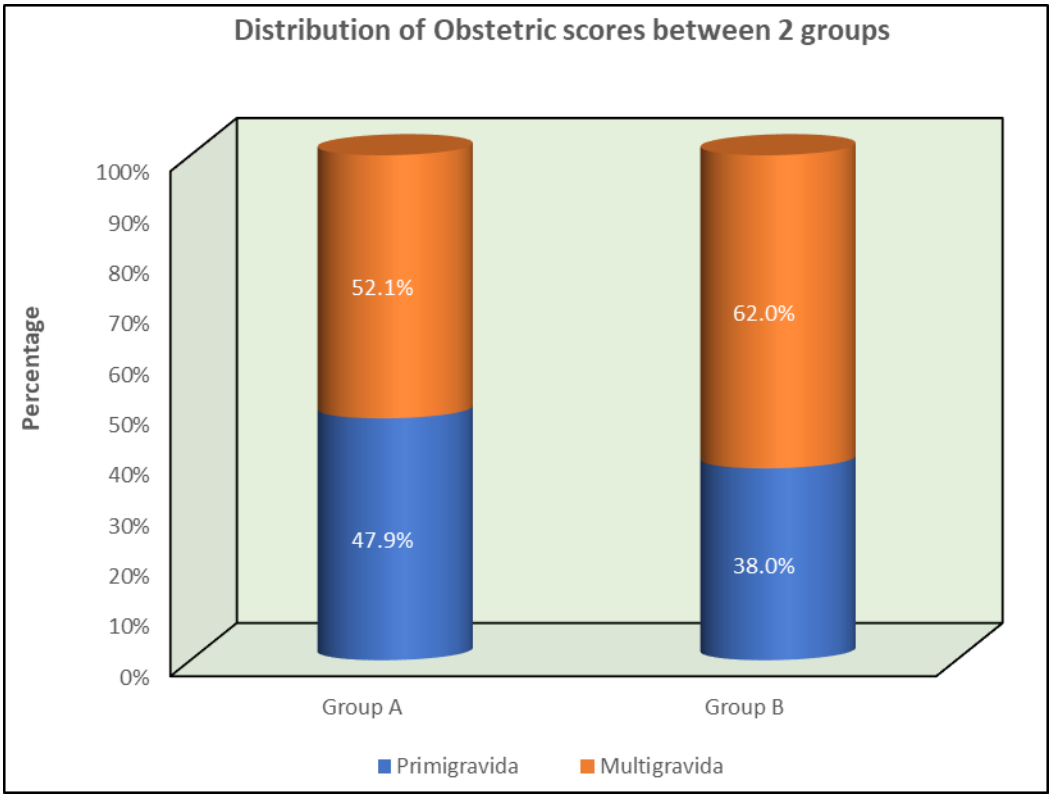
The mean age in Group A Pregnant women was 23.44 ± 3.90 and in Group B was 24.15 ± 4.40, with age range of 18 – 37 years. There was no significant difference noted in the mean age of pregnant women between 2 groups. The mean Gestational age in Group A was 37.39 ± 2.53 weeks, in Group B was 37.82 ± 2.21 weeks, with gestational age ranging between 31 – 42 years and there was no significant difference observed with respect to mean gestational age between 2groups.

In Group A & Group B, majority of the pregnant women were Multigravida (52.1% & 62.0%) and there was no significant difference in the obstetric scores between 2 groups. In both groups, there were no pregnant women with smoking history and all were from Asian background.



Group A: Hypertensive Pregnant Women & GroupB: Normotensive PregnantWomen

Figure no.18 Agewise distribution between HDPs group and Normotensive group



Group A: Hypertensive Pregnant Women & Group B: Normotensive Pregnant Women

Figure no.19 Distribution of obstretric scores between HDPs group and Normotensive group

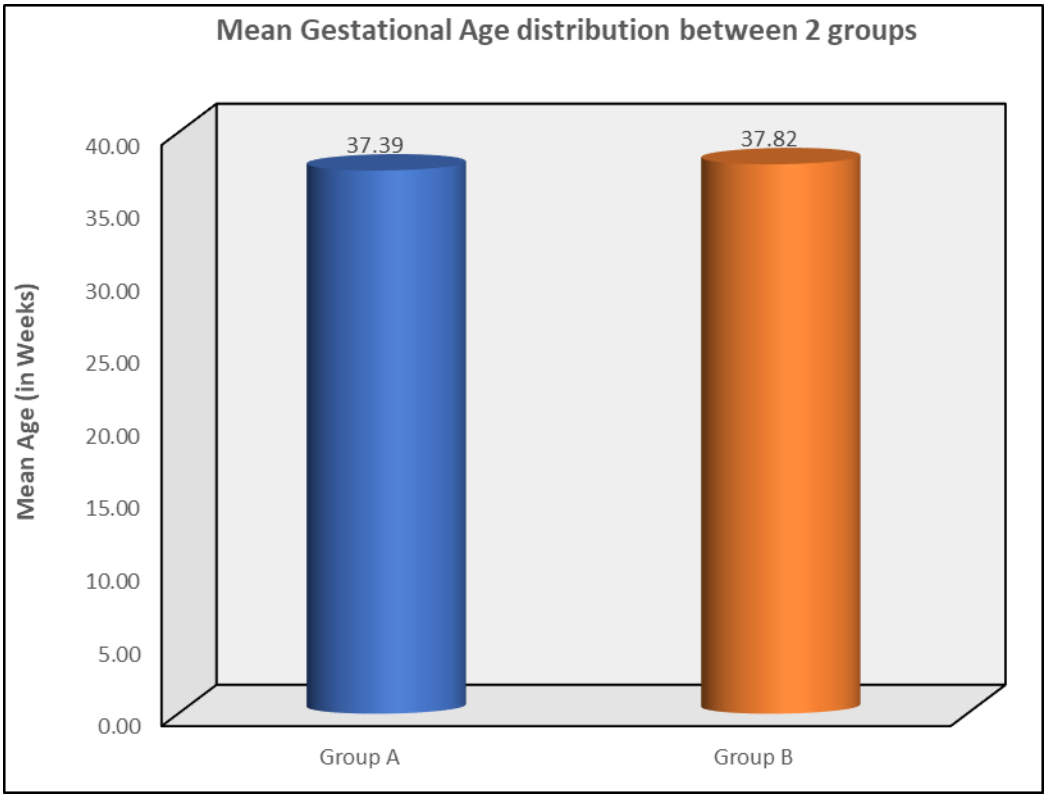


Figure no 20. Mean gestational age distribution between HDPs group and Normotensive group

TABLE NO.3 Distribution of Hypertensive Pregnant Women			
Variable	Category	n	%
Hypertensi veGroup	Chronic HTN with Superimposed PE	1	1.4%
	Gestational HTN	15	21.1%
	Imminent Eclampsia	6	8.5%
	PE without Severe Features	10	14.1%
	PE with Severe Features	39	54.9%

The most common category, comprising 54.9% of pregnant women (n=39), experiences preeclampsia with severe features, followed by 21.1% of pregnant women (n=15) have gestational hypertension, 14.1% of pregnant women (n=10) exhibit preeclampsia without severefeatures, 8.5% of pregnant women (n=6) are at risk of imminent eclampsia. The least

prevalent category, with only 1.4% of pregnant women (n=1), has chronic hypertension with superimposedpreeclampsia.

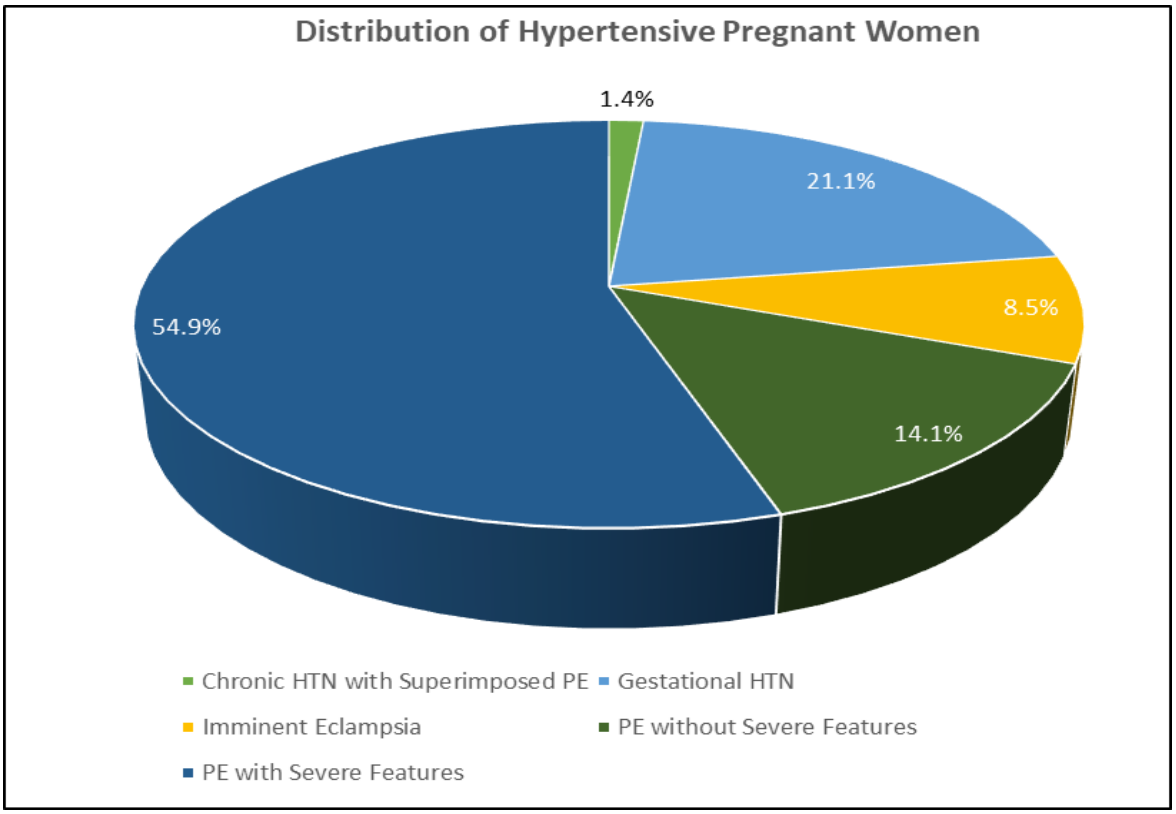


Figure no. 21 Distribution of HDPs

TABLE NO.4 Comparison of Maternal Status at different Trimesters between 2 groups using Chi Square Test

Variable	Category	Group A		Group B		p-value
		n	%	n	%	
1st Trimester	H/o Anemia	0	0.0%	1	1.4%	0.39
	H/o PV Leak	1	1.4%	0	0.0%	
	H/o Spontaneous Abortion	0	0.0%	1	1.4%	
	Uneventful	70	98.6 %	69	97.2%	
2nd Trimester	H/o of Anemia	1	1.4%	1	1.4%	0.57
	H/o of High BP Recordings	1	1.4%	0	0.0%	
	H/o Cervical Encirclage	0	0.0%	1	1.4%	
	Uneventful	69	97.2 %	69	97.2%	

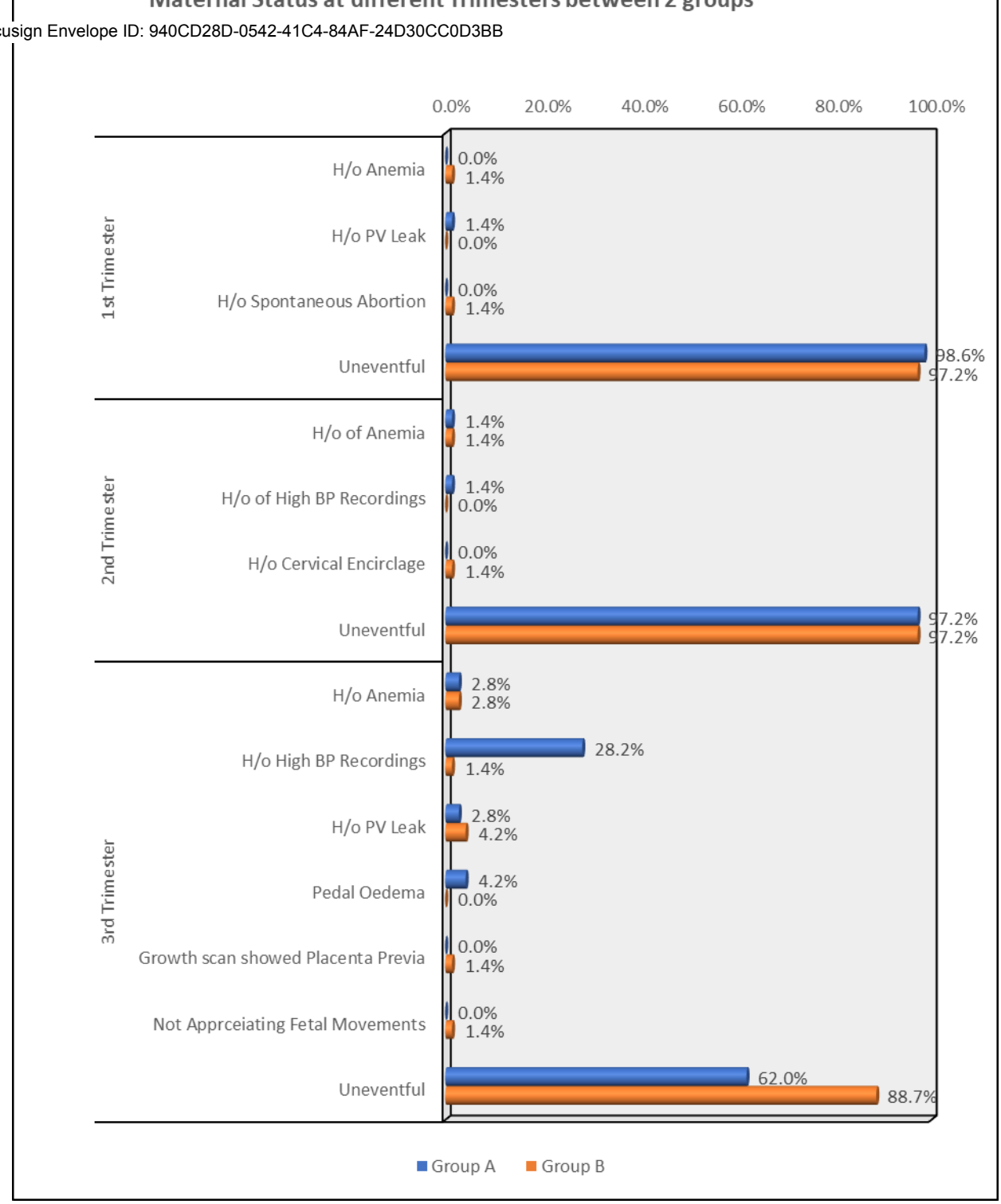
3rd Trimester	H/o Anemia	2	2.8%	2	2.8%	<0.001*
	H/o High BP Recordings	20	28.2 %	1	1.4%	
	H/o PV Leak	2	2.8%	3	4.2%	
	Pedal Oedema	3	4.2%	0	0.0%	
	Growth scan showed Placenta Previa	0	0.0%	1	1.4%	
	Not Appreciating Fetal Movements	0	0.0%	1	1.4%	
	Uneventful	44	62.0 %	63	88.7%	

* - Statistically Significant

In 1st Trimester, H/o Anemia was prevalent in similar rates in both Group A (1.4%) and Group B (1.4%), H/o PV Leak and H/o Spontaneous Abortion, occurred in one each patient in Group A& B. The majority (98.6% in Group A, 97.2% in Group B) had an uneventful first trimester.

In 2nd Trimester, H/o Anemia was prevalent in similar rates in both Group A (1.4%) and GroupB (1.4%), H/o of High BP Recordings and H/o Cervical Encirclage occurred in one each patientin Group A & B. The majority (97.2% in both groups) had an uneventful Second trimester.

In 3rd Trimester, H/o Anemia was prevalent in similar rates in both Group A and Group B (2.8%in both groups), followed by, which was more common in Group A as compared to Group B (28.2% vs. 1.4%), H/o PV Leak and Pedal Oedema occurred in a few patients in both groups, ranging between 2.8 – 4.2%. In Group B, Growth scan showed Placenta Previa and Not Appreciating Fetal Movements was observed in one patient each (1.4%). Uneventful 3rd trimesterPregnancy was more prevalent in Group B as compared to Group A (88.7% vs. 62.0%). This difference was statistically significant with respect to maternal status between the 2 groups during the third trimester at $p<0.001$.



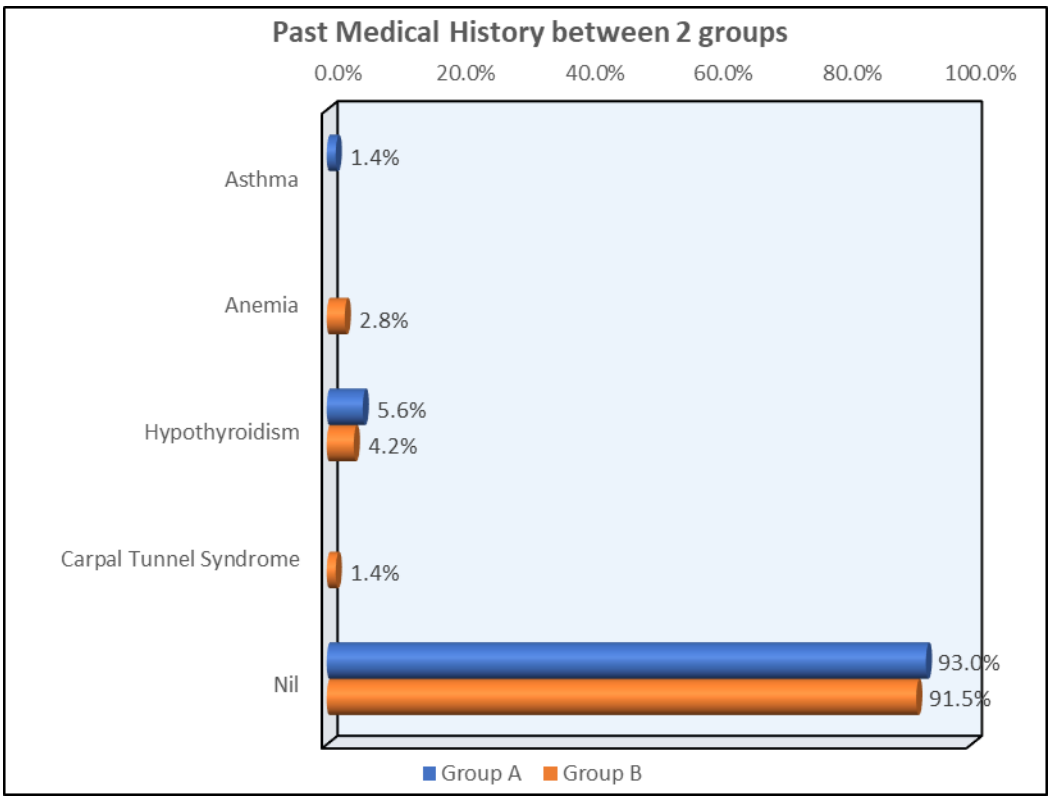
Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no. 22 Maternal status at different trimesters between HDPs and normotensive group

TABLE NO.5 Comparison of Past Medical History between 2 groups using Chi Square Test						
Variable	Category	Group A		Group B		p-value
		n	%	n	%	
Past Medical History	Asthma	1	1.4%	0	0.0%	0.39
	Anemia	0	0.0%	2	2.8%	
	Hypothyroidism	4	5.6%	3	4.2%	
	Carpal Tunnel Syndrome	0	0.0%	1	1.4%	
	Nil	66	93.0 %	65	91.5 %	

Group A showed 1.4% of patients have asthma (n=1) as compared to Group B with no cases reported with asthma (0%). Group B showed 2.8% of patients have a previous history of Anemia(n=2) as compared to Group A with no cases reported with previous Anemia (n=0). Group A, reported with 5.6% of patients have hypothyroidism (n=4)) as compared to Group B, with 4.2% of patients presented with hypothyroidism (n=3). This was followed by Group B reported CarpalTunnel Syndrome in 1.4% samples (n=1) as compared to Group A with no such cases been reported. No specific medical history was predominantly observed in both groups (93.0% in Group A, 91.5% in Group B). There was no significant difference in Past Medical History between the 2 groups.



Group A: Hypertensive Pregnant Women & Group B: Normotensive Pregnant Women

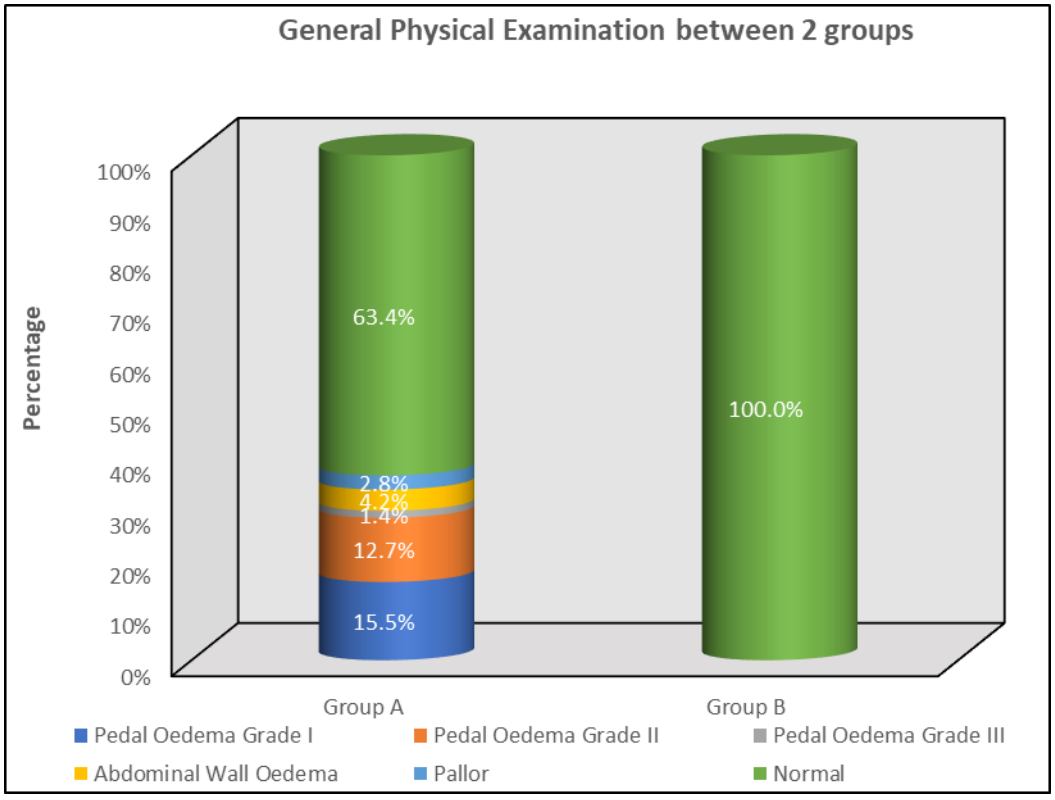
Figure no 23 Past medical history between HDPs and normotensive group

TABLE NO.6 Comparison of General Physical Examination between 2 groups using Chi Square Test

Variable	Category	Group A		Group B		p-value
		n	%	n	%	
Gen. Physical Exam	Pedal Oedema Grade I	11	15.5 %	0	0.0%	<0.001*
	Pedal Oedema Grade II	9	12.7 %	0	0.0%	
	Pedal Oedema Grade III	1	1.4%	0	0.0%	
	Abdominal Wall Oedema	3	4.2%	0	0.0%	
	Pallor	2	2.8%	0	0.0%	
	Normal	45	63.4 %	71	100.0 %	

* - Statistically Significant

Grade I Pedal Oedema was reported in 15.5% of patients in Group A and has higher occurrence than Group B (0%). This was followed by Group A, which reported with Grade II Pedal Oedema(12.7%) that also shows a higher occurrence compared to Group B (0%). Grade III Pedal Oedemawas reported only in patient in Group A (1.4%), while Group B has none. Abdominal Wall Oedema was reported in 4.2% and Pallor (2.8%), both occur in Group A but not in Group B. Group A (63.4%) has a lower proportion of normal findings compared to Group B (100%). The difference showed a significant difference in general physical examination outcomes between thetwo groups at p<0.001.



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 24. General physical examination between HDPs and normotensive group

TABLE NO.7 Comparison of mean clinical and Echocardiographic parameters between 2 groups using Independent Student t Test

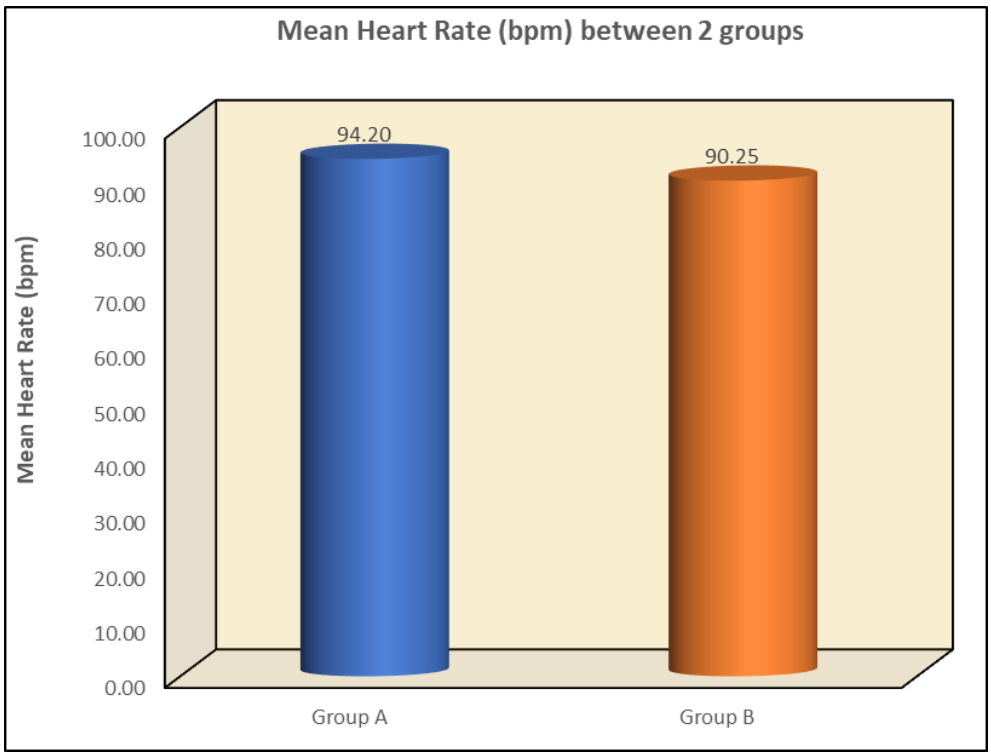
Parameters	Groups	N	Mean	SD	Mean Diff	p-value
HR	Group A	71	94.20	9.22	3.95	0.002 *

TABLE NO.7 Comparison of mean clinical and Echocardiographic parameters between 2 groups using Independent Student t Test						
Parameters	Groups	N	Mean	SD	Mean Diff	p-value
	Group B	71	90.25	5.25		
SBP	Group A	71	145.41	13.23	28.90	<0.001*
	Group B	71	116.51	9.95		
SBP	Group A	71	94.65	7.34	19.80	<0.001*
	Group B	71	74.85	5.88		
MAP	Group A	71	112.00	8.10	23.12	<0.001*
	Group B	71	88.88	6.25		
EF (%)	Group A	71	61.52	5.13	-4.07	<0.001*
	Group B	71	65.59	4.49		
LV ESV	Group A	71	26.62	5.32	0.20	0.81
	Group B	71	26.42	4.28		
LV EDV	Group A	71	70.47	14.57	1.72	0.44
	Group B	71	68.75	11.67		
Stroke Vol.	Group A	71	53.16	9.34	-2.54	0.07
	Group B	71	55.69	6.95		
LV Mass	Group A	71	131.62	18.47	1.42	0.62
	Group B	71	130.20	15.25		

* - Statistically Significant

The mean Heart rate in Group A was significantly higher (94.20 ± 9.22) as compared to Group B(90.25 ± 5.25) and the mean difference was statistically significant at p=0.002. The mean SystolicBP in Group A was significantly higher (145.41 ± 13.23) as compared to Group B (116.51 ± 9.95) and the mean difference was statistically significant at p<0.001. The mean Diastolic BP in GroupA was significantly higher (94.65 ± 7.34) as compared to Group B (74.85 ± 5.88) and the mean difference was statistically significant at p<0.001. The mean MAP in GroupA was significantly higher (112.00 ± 8.10) as compared to Group B (88.88 ± 6.25) and the mean

difference was statistically significant at p<0.001. The mean percentage of Ejection Fraction in Group A was significantly lesser (61.52 ± 5.13) as compared to Group B (65.59 ± 4.49) and the mean difference was statistically significant at p<0.001. However, the mean LV ESV, mean LV EDV, mean Stroke volume and mean LV mass did not demonstrate significant difference between 2 groups.

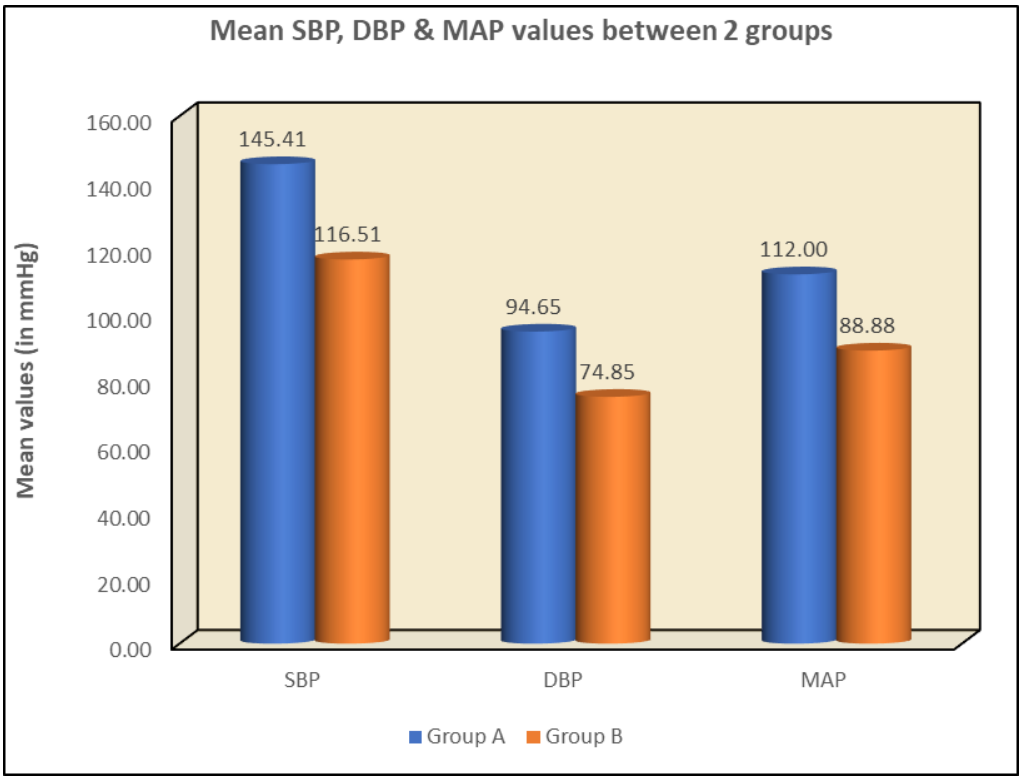


Group A: Hypertensive Pregnant Women

&

Group B: Normotensive Pregnant Women

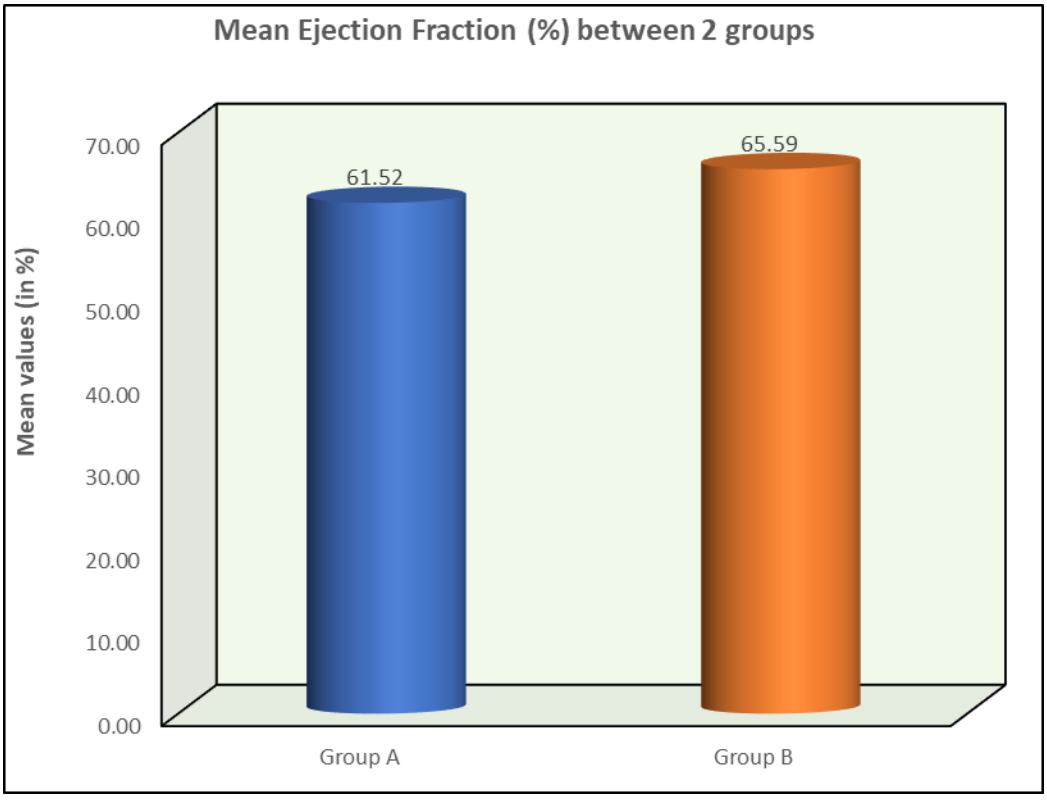
Figure no 25 Mean Heart Rate (bpm) between HDPs and normotensive group



Group A:
Hypertensive
Pregnant Women
&

Group B:
Normotensive
Pregnant Women

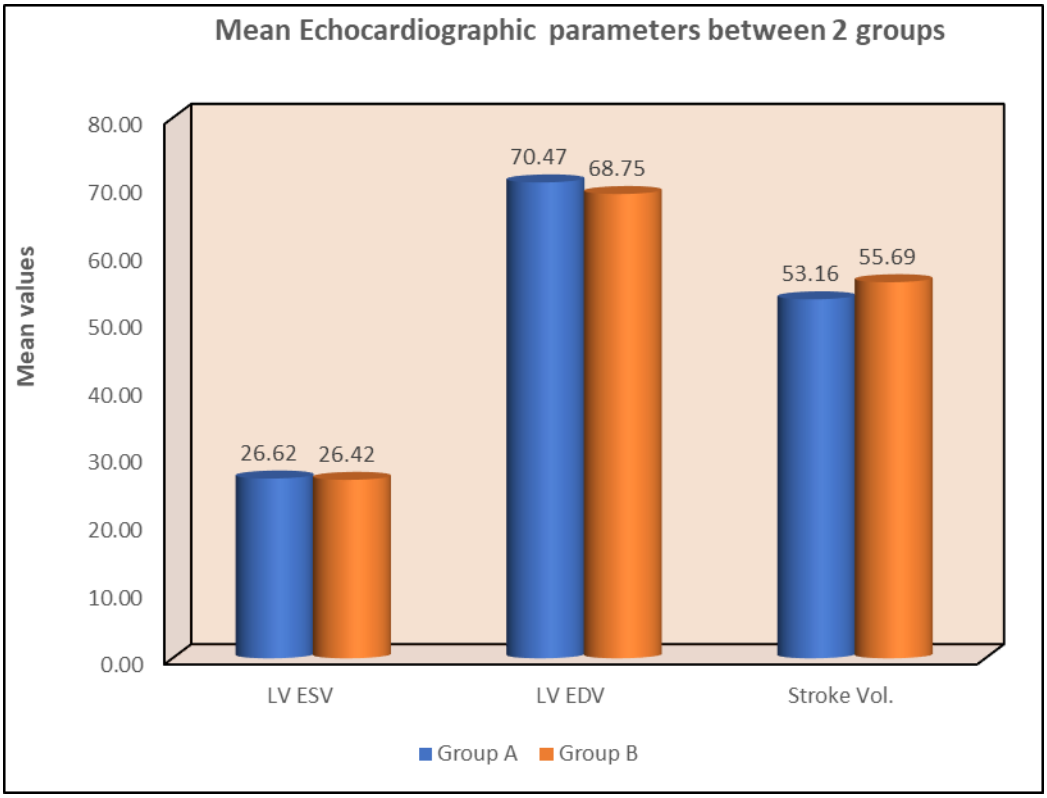
Figure no 26 Mean SBP, DBP and MAP values between HDPs and normotensive group



Group A:
Hypertensive
Pregnant Women
&

Group B:
Normotensive
Pregnant Women

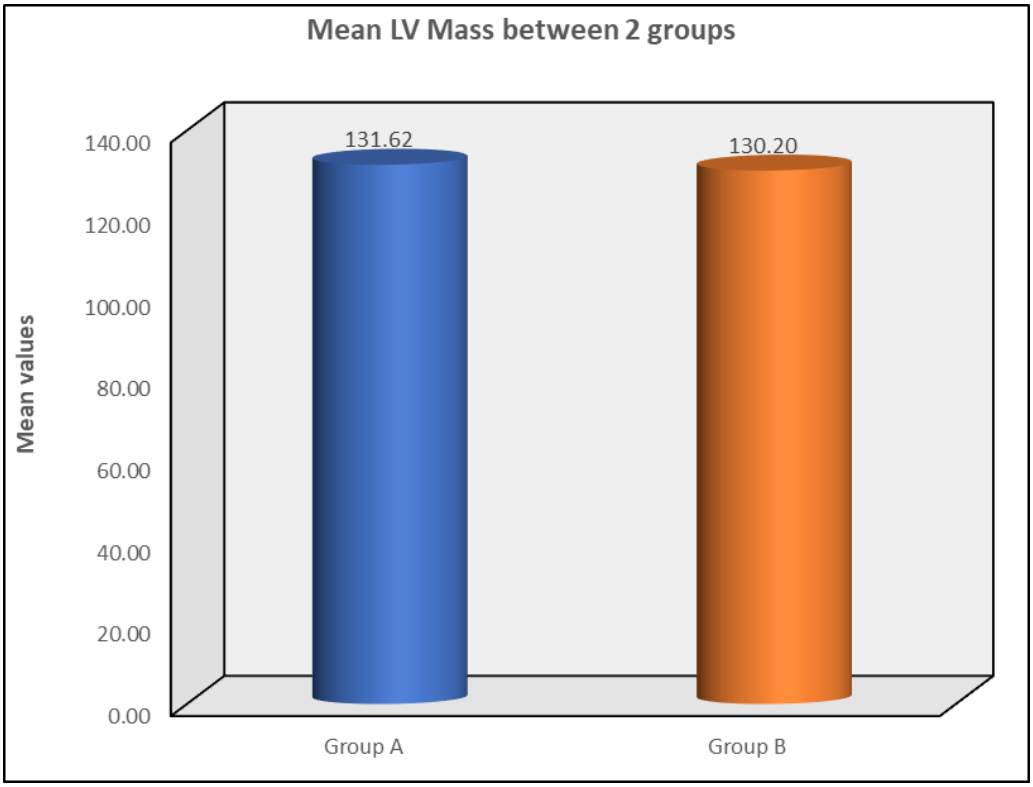
Figure no 27 Mean Ejection Fraction (%) between HDPs and normotensive group



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 28 Mean Echocardiographic parameters between HDPs and normotensive group



Group A: Hypertensive Pregnant Women &

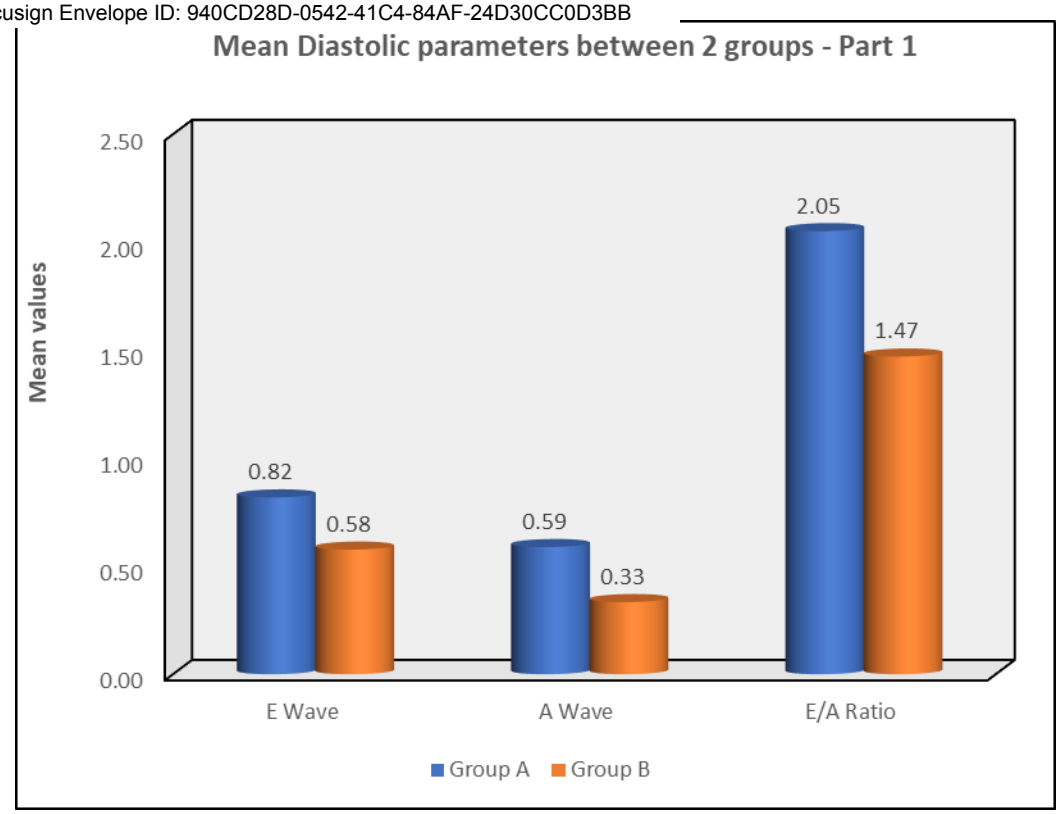
Group B: Normotensive Pregnant Women

Figure no 29 Mean LV Mass between HDPs and normotensive group

TABLE NO.8 Comparison of mean Diastolic parameters between 2 groups using Mann Whitney Test						
Parameters	Groups	N	Mean	SD	Mean Diff	p-value
E Wave	Group A	71	0.82	0.45	0.24	<0.001*
	Group B	71	0.58	0.28		
A Wave	Group A	71	0.59	0.35	0.25	<0.001*
	Group B	71	0.33	0.19		
E/A Ratio	Group A	71	2.05	2.40	0.58	0.07
	Group B	71	1.47	0.30		
Septal e'	Group A	71	0.119	0.163	-0.037	<0.001*
	Group B	71	0.156	0.133		
Lateral e'	Group A	71	0.121	0.030	-0.012	0.04*
	Group B	71	0.132	0.040		

* - Statistically Significant

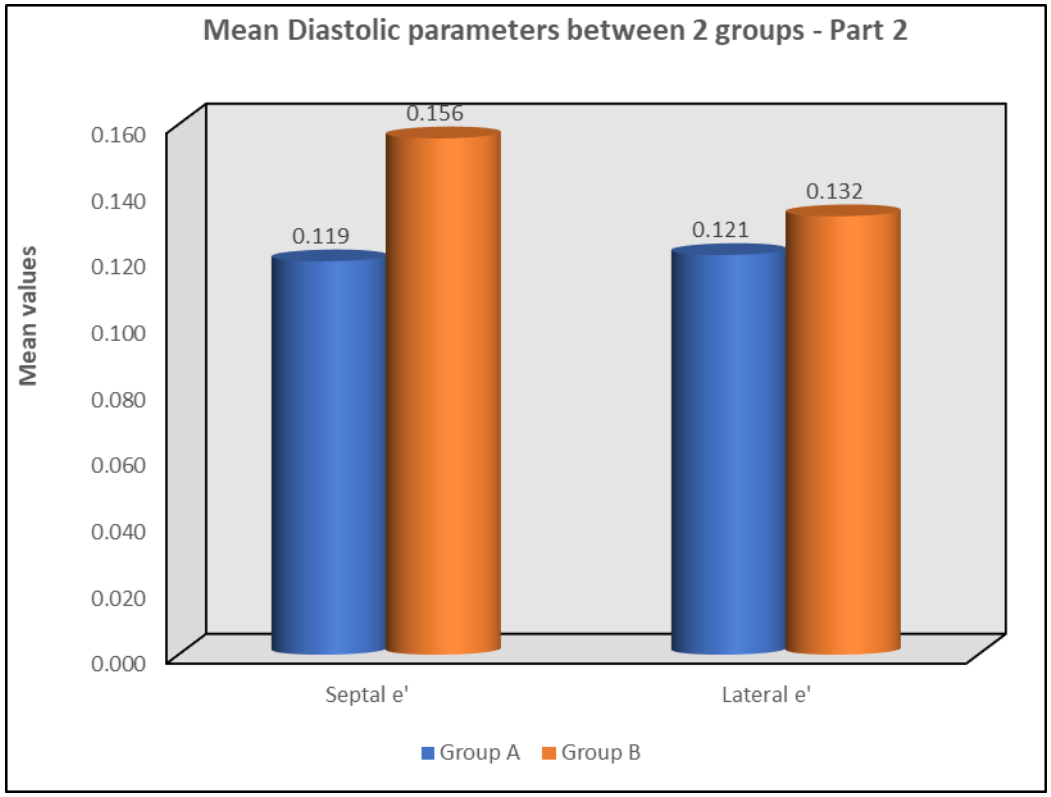
The mean E Wave in Group A was significantly higher (0.82 ± 0.45) as compared to Group B (0.58 ± 0.28) and the mean difference was statistically significant at p<0.001. The mean A Wavein Group A was significantly higher (0.59 ± 0.35) as compared to Group B (0.33 ± 0.19) and themean difference was statistically significant at p<0.001. The mean Septal e' in Group A was significantly lesser (0.119 ± 0.163) as compared to Group B (0.156 ± 0.133) and the mean difference was statistically significant at p<0.001. The mean Lateral e' in Group A was significantly lesser (0.121 ± 0.030) as compared to Group B (0.132 ± 0.040) and the mean difference was statistically significant at p=0.04. However, the mean E/A ratio did not demonstrate statistically significant difference between 2 groups.



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 30 Mean Diastolic parameters between HDPs and normotensive group- part 1



Group A: Hypertensive Pregnant Women &
Group B: Normotensive Pregnant Women

Figure no 31 Mean Diastolic parameters between HDPs and normotensive group- part 2

TABLE NO.9 Comparison of Mode of Delivery between 2 groups using Chi Square Test						
Variable	Category	Group A		Group B		p-value
		n	%	n	%	
Mode of Delivery	Full Term Vaginal Delivery	16	22.5 %	32	45.1 %	0.04*
	Preterm Vaginal Delivery	6	8.5%	4	5.6%	
	Emergency LSCS	48	67.6 %	32	45.1 %	
	Elective LSCS	1	1.4%	2	2.8%	
	Vaginal birth after caesarean section	0	0.0%	1	1.4%	

* - Statistically Significant

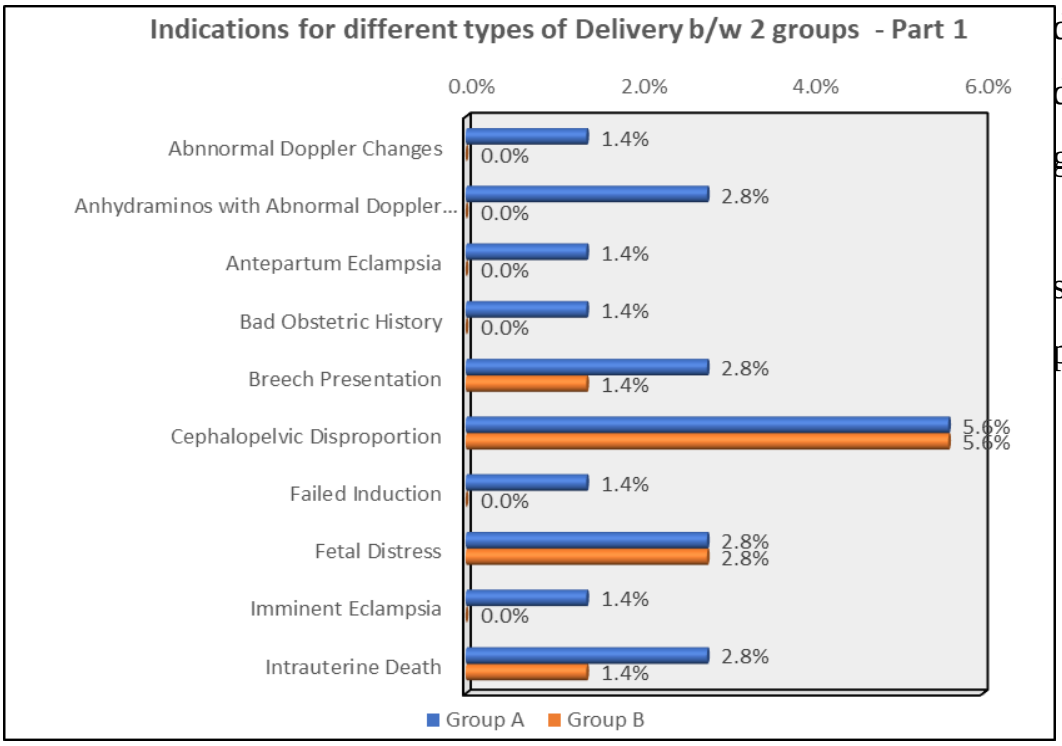
Group A had a lower proportion of full-term vaginal deliveries (22.5%) compared to Group B (45.1%). Contrastingly, Group A had a higher occurrence of emergency LSCS (67.6%) compared to Group B (45.1%). This was followed by Preterm Vaginal Delivery, which occurred in both groups, with 8.5% in Group A and 5.6% in Group B. This was then followed by Group A (1.4%)and Group B (2.8%) which had minimal elective LSCS cases and finally Group B with 1 case ofVaginal Birth After Caesarean Section (1.4%). The mode of delivery differs significantly between the two groups, with Group B having more full-term vaginal deliveries and fewer emergency LSCS cases. This difference indicates a significant difference in mode of delivery between the two groups at p=0.04.

TABLE NO.10 Comparison of Indications for different types of Delivery between 2 groups using Chi Square Test						
Variable	Category	Group A		Group B		p-value
		n	%	n	%	
Indication	Abnormal Doppler Changes	1	1.4%	0	0.0%	
	Anhydraminos with Abnormal Doppler Changes	2	2.8%	0	0.0%	
	Antepartum Eclampsia	1	1.4%	0	0.0%	
	Bad Obstetric History	1	1.4%	0	0.0%	

	Breech Presentation	2	2.8%	1	1.4%	0.001*
	Cephalopelvic Disproportion	4	5.6%	4	5.6%	
	Failed Induction	1	1.4%	0	0.0%	
	Fetal Distress	2	2.8%	2	2.8%	
	Imminent Eclampsia	1	1.4%	0	0.0%	
	Intrauterine Death	2	2.8%	1	1.4%	
	Maternal Request	0	0.0%	2	2.8%	
	Meconium Stained Liquid	0	0.0%	2	2.8%	
	Non-reassuring NST	2	2.8%	1	1.4%	
	Non-Progression of Labour	1	1.4%	3	4.2%	
	PE with Severe Features	20	28.2%	0	0.0%	
	Placenta Previa	0	0.0%	1	1.4%	
	H/o Previous LSCS	12	16.9%	13	18.3%	
	Prolonged PROM	1	1.4%	0	0.0%	
	Secondary Stage Arrest	1	1.4%	0	0.0%	
	Severe Oligohydramnios	1	1.4%	4	5.6%	
	Nil	16	22.5%	37	52.1%	

* - Statistically Significant

The comparison of different indications between 2 groups expressed that Pre-eclampsia with Severe Features was most predominantly seen in Group A (28.2%) as compared to no cases (0.0%) reported in Group B. Other than this, various indications occur in both groups, including Anhydraminos with abnormal Doppler changes, antepartum eclampsia, bad obstetric history, breech presentation, Cephalopelvic disproportion, failed induction, Fetal distress, imminent eclampsia, intrauterine death, maternal request, meconium-stained liquid, non-reassuring NST, non-progression of labour, Placenta Previa, h/o previous LSCS, prolonged PROM, secondary stage arrest and severe oligohydramnios, which have occurred between 2 groups varying between1.4% to 16.9% in Group A and 1.4% to 18.3% in Group B. Group A showed significantly lesser proportion of cases with no specific indication (22.5%) as compared to Group B (52.1%). Thesedifferences in the occurrence of various Indications for

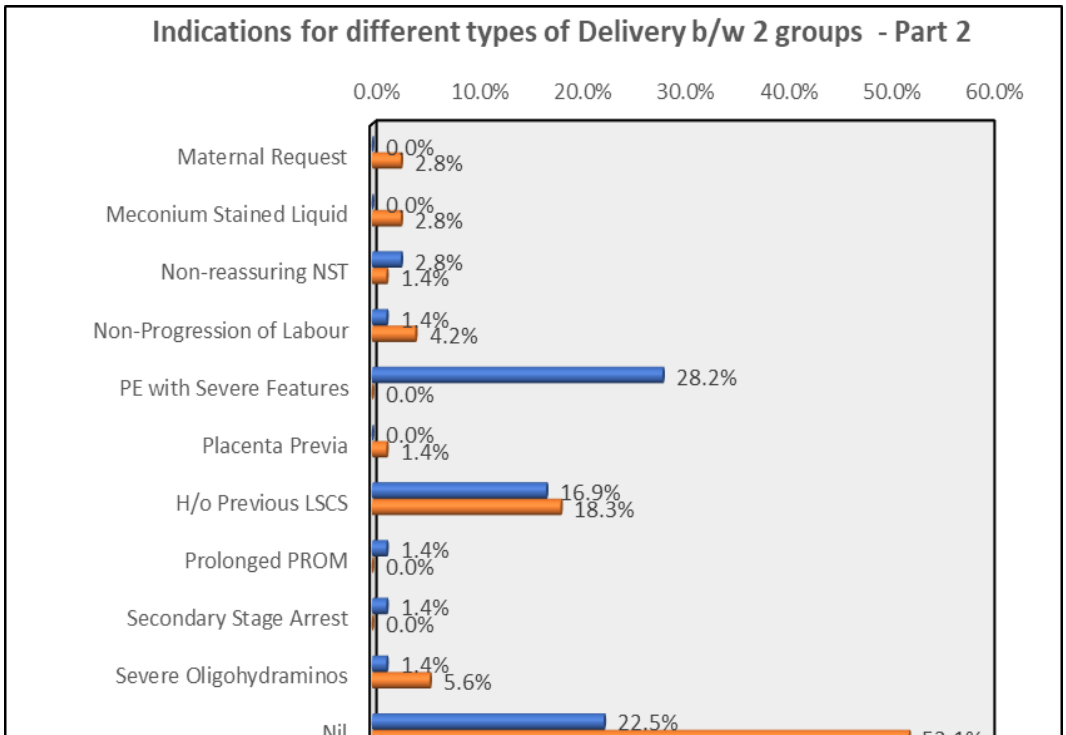


different modes of delivery between 2 groups was statistically significant at p=0.001.

Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 32 Indication for different types of delivery between HDPs and normotensive group- part 1



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

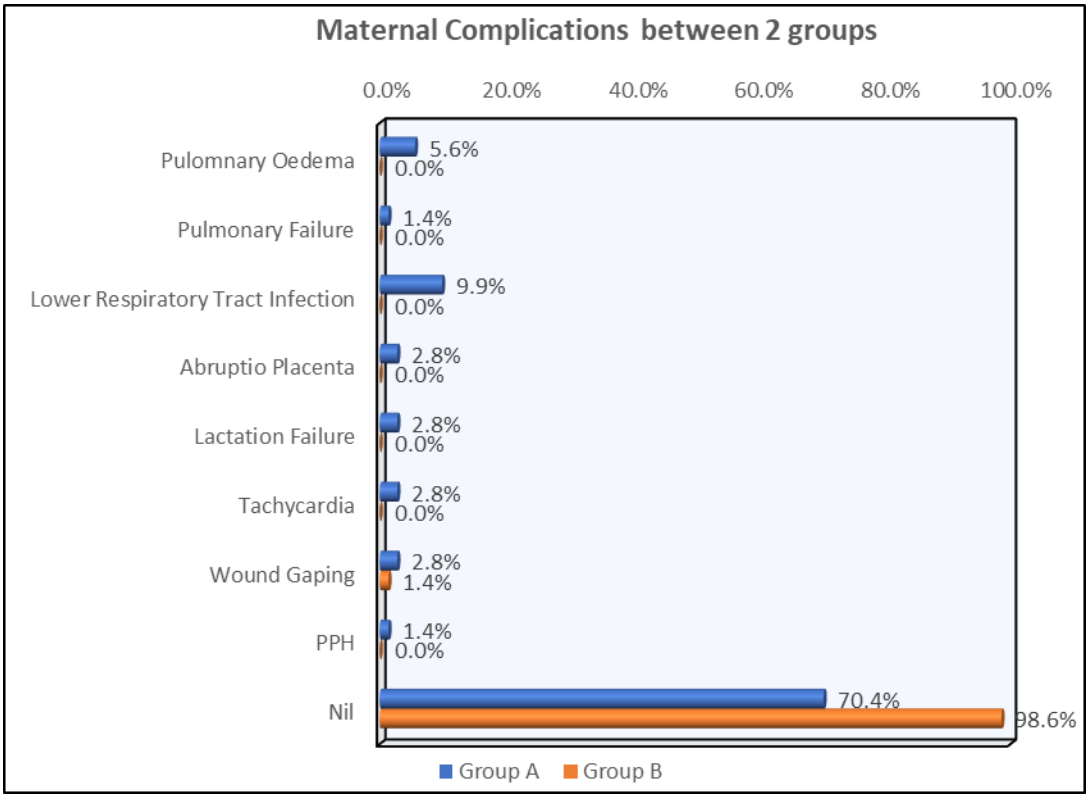
Figure no 33 Indication for different types of delivery between HDPs and normotensivegroup- part 2

TABLE NO.11 Comparison of Maternal Complications between 2 groups using Chi Square Test						
Variable	Category	Group A		Group B		p-value
		n	%	n	%	
Maternal Complications	Pulmonary Oedema	4	5.6%	0	0.0%	0.004*
	Pulmonary Failure	1	1.4%	0	0.0%	
	Lower Respiratory Tract Infection	7	9.9%	0	0.0%	
	Abruptio Placenta	2	2.8%	0	0.0%	
	Lactation Failure	2	2.8%	0	0.0%	
	Tachycardia	2	2.8%	0	0.0%	
	Wound Gaping	2	2.8%	1	1.4%	
	PPH	1	1.4%	0	0.0%	
	Nil	50	70.4%	70	98.6%	

* - Statistically Significant

A significant proportion of Group B Women have reported with No complications (98.6%) as compared to Group A (70.4). Group A has a significantly higher occurrence of lower respiratorytract infections (9.9%) as compared to Group B (0.0%). This was then followed by Pulmonary Oedema (5.6%), Wound Gaping, Abruptio Placenta, Lactation Failure, Tachycardia, and PPH with 2 cases each (2.8%) in Group A and finally Pulmonary Failure was observed in 1 case (1.4%) as compared to Group B, with no complications been reported except for Wound Gaping in 1 case (1.4%). This reveals that Group A exhibits significantly more maternal complications overall, with specific differences in lower respiratory tract infections, pulmonary oedema, and wound gaping as compared to Group B and the difference was statistically significant at

$$p=0.004.$$



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

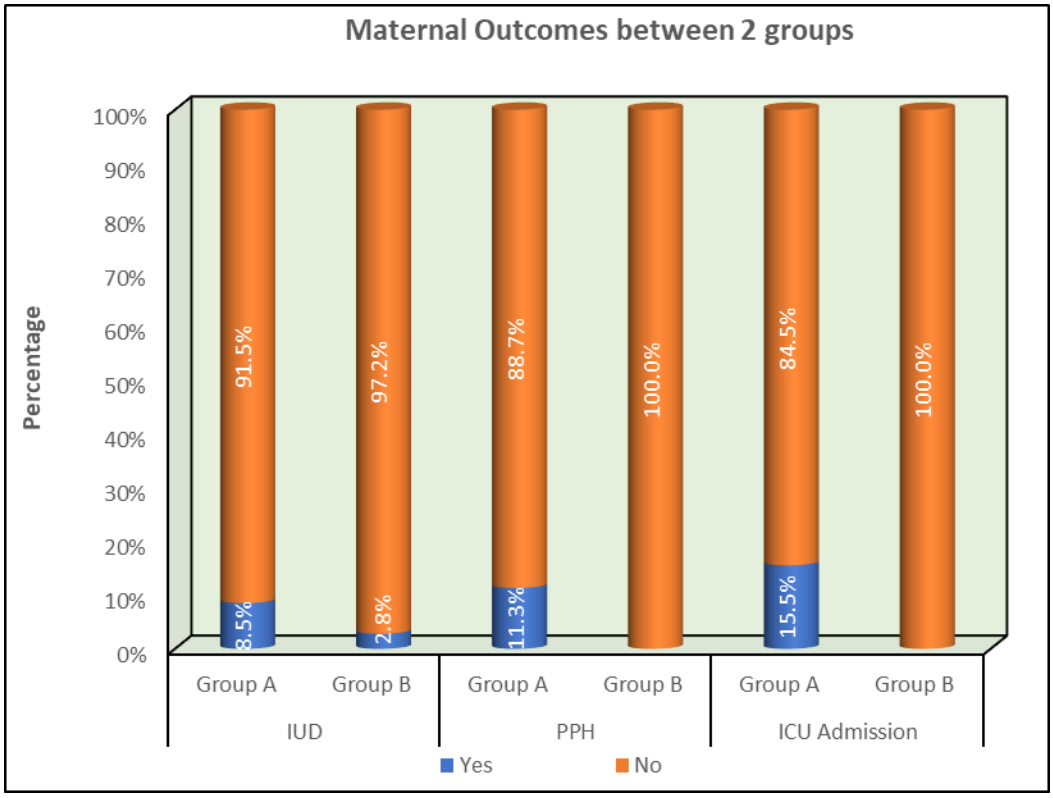
Figure no 34 Maternal complication between HDPs and normotensive group

TABLE NO.12 Comparison of Maternal Outcomes between 2 groups using Chi Square Test

Variable	Category	Group A		Group B		p-value
		n	%	n	%	
IUD	Yes	6	8.5%	2	2.8%	0.15
	No	65	91.5 %	69	97.2%	
PPH	Yes	8	11.3 %	0	0.0%	0.004*
	No	63	88.7 %	71	100.0 %	
ICU Admission	Yes	11	15.5 %	0	0.0%	0.001*
	No	60	84.5 %	71	100.0 %	

* - Statistically Significant

Maternal Outcome like Postpartum Haemorrhage (PPH) was significantly observed in higher proportion in Group A had (11.3%) as compared to Group B with no reported cases of PPH (0.0%). This difference in the occurrence of PPH between 2 groups was statistically significant at p=0.004. Similarly, Intensive Care Unit (ICU) Admission was significantly observed in higherproportion in Group A had (15.5%) as compared to Group B with no reported cases of ICU Admission (0.0%). This difference in the Incidence of ICU Admission between 2 groups was statistically significant at p=0.001. However, the Intra-uterine death was relatively seen in higherproportion in Group A (8.5%) as compared to Group B (2.8%), but there was no significant difference between 2 groups. In summary, Group A exhibited more PPH cases and a higher needfor ICU admission as compared to Group B.



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

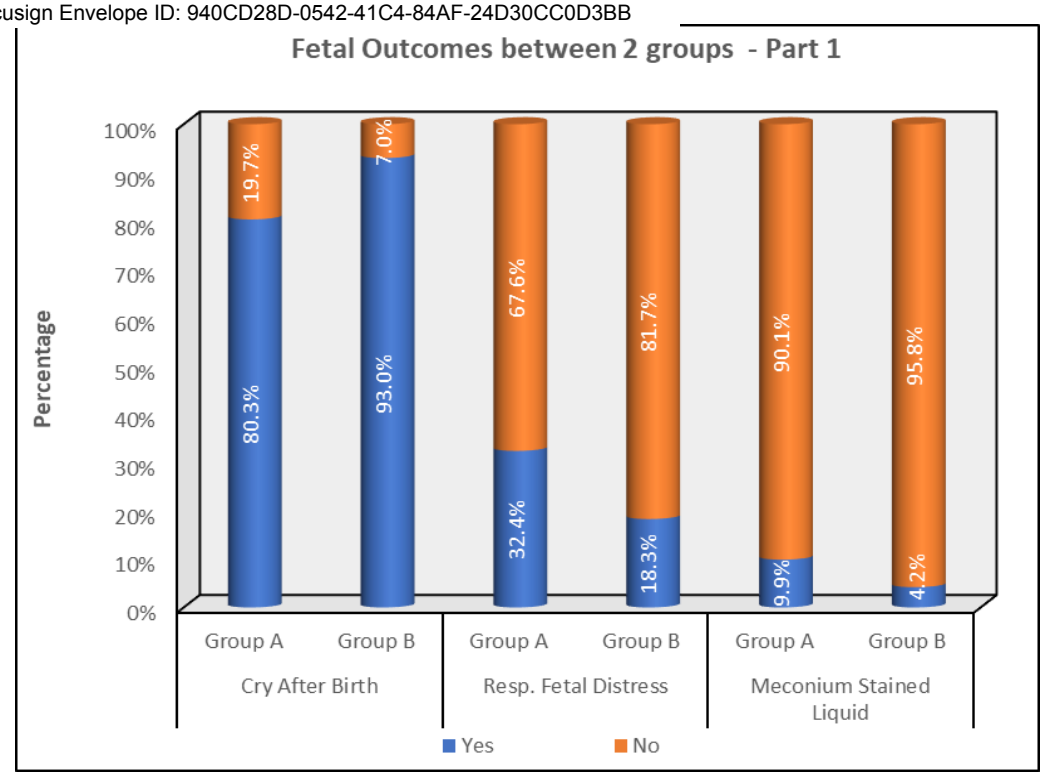
v

Figure no 35 Maternal outcomes between HDPs and normotensive group

TABLE NO.13 Comparison of Fetal Outcomes between 2 groups using Chi Square Test						
Variable	Category	Group A		Group B		p-value
		n	%	n	%	
APGAR Scores	> 7 Scores	57	80.3 %	66	93.0%	0.03*
	< 7 Scores	14	19.7 %	5	7.0%	
Resp. Fetal Distress	Yes	23	32.4 %	13	18.3%	0.04*
	No	48	67.6 %	58	81.7%	
Meconium StainedLiquid	Yes	7	9.9%	3	4.2%	0.19
	No	64	90.1 %	68	95.8%	
Fetal Growth Retardation	Yes	11	15.5 %	0	0.0%	0.001*
	No	60	84.5 %	71	100.0 %	
NICU Admission	Yes	29	40.8 %	16	22.5%	0.02*
	No	42	59.2 %	55	77.5%	
Neonatal Mortality	Yes	3	4.2%	2	2.8%	0.65
	No	68	95.8 %	69	97.2%	

* - Statistically Significant

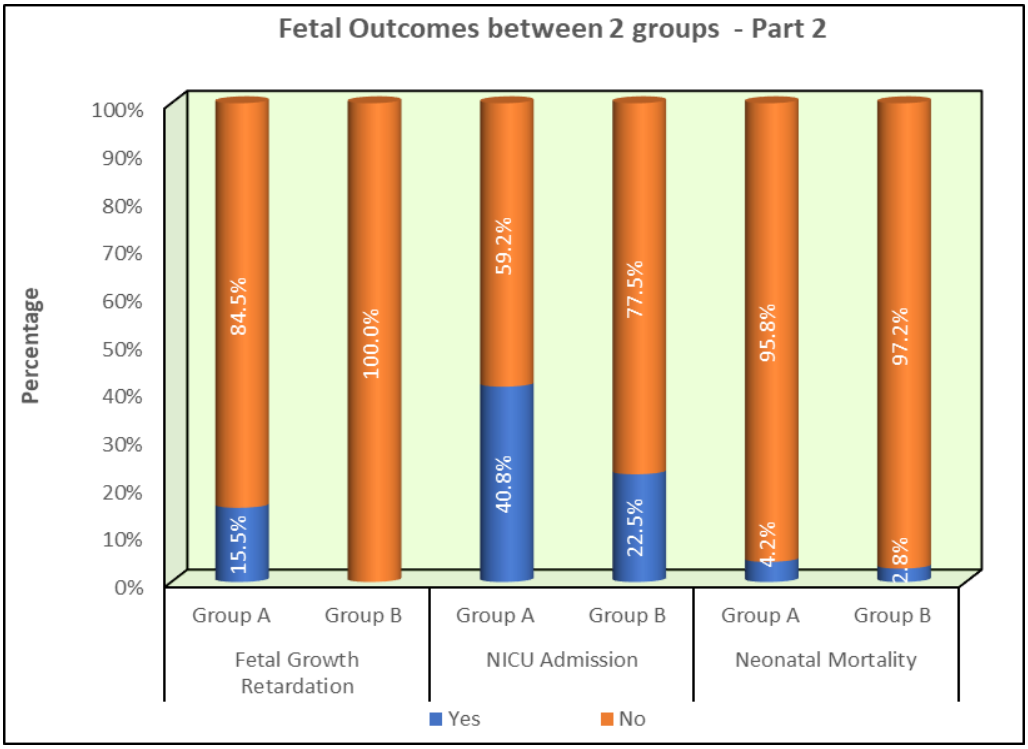
Among the Foetal Outcomes, APGAR Score of > 7 was significantly observed in higher proportion in Group B (93.0%) as compared to Group A (80.3%) and the difference was statistically significant at p=0.03. Contrastingly, in Group A Respiratory Fetal Distress (32.4% vs 18.3%), Fetal Growth Retardation (15.5% vs 0.0%) and NICU Admission (40.8% vs 22.5%) had been reported in higher occurrence as compared to Group B and the difference was statistically significant at p=0.04, p=0.001 & p=0.02 respectively. However, the Incidence of Meconium Stained Liquid & Neonatal Mortality did not demonstrate statistically significant difference between 2 groups.



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 36 Fetal outcomes between HDPs and normotensive group- part 1



Group A: Hypertensive Pregnant Women &

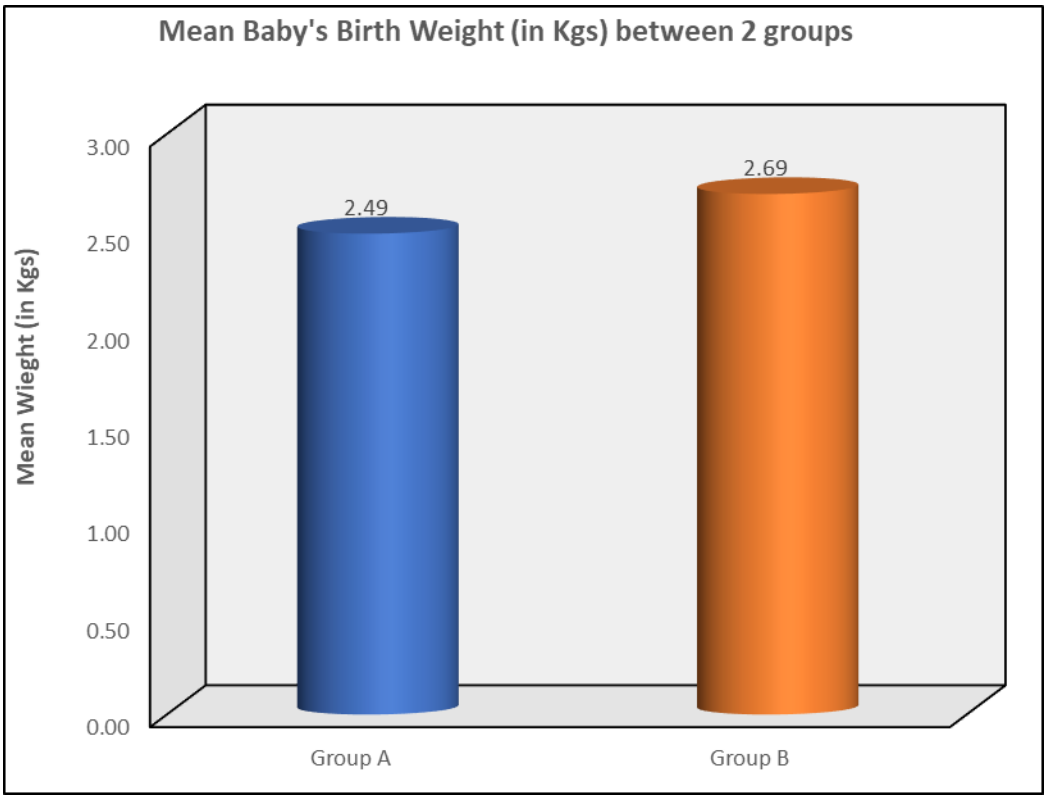
Group B: Normotensive Pregnant Women

Figure no 37 Fetal outcomes between HDPs and normotensive group- part 2

TABLE NO.14 Comparison of mean Baby's Birth Weight (in Kgs) between 2 groups using Independent Student t Test						
Parameter	Groups	N	Mean	SD	Mean Diff	p-value
Baby Birth Weight	Group A	71	2.49	0.56	-0.21	0.02*
	Group B	71	2.69	0.50		

* - Statistically Significant

The mean Baby's birth weight in Group A was significantly lesser (2.49 ± 0.56 Kgs.) as compared to Group B (2.69 ± 0.50 Kgs.) and the mean difference between 2 groups was statistically significant at p=0.02.



Group A: Hypertensive Pregnant Women &

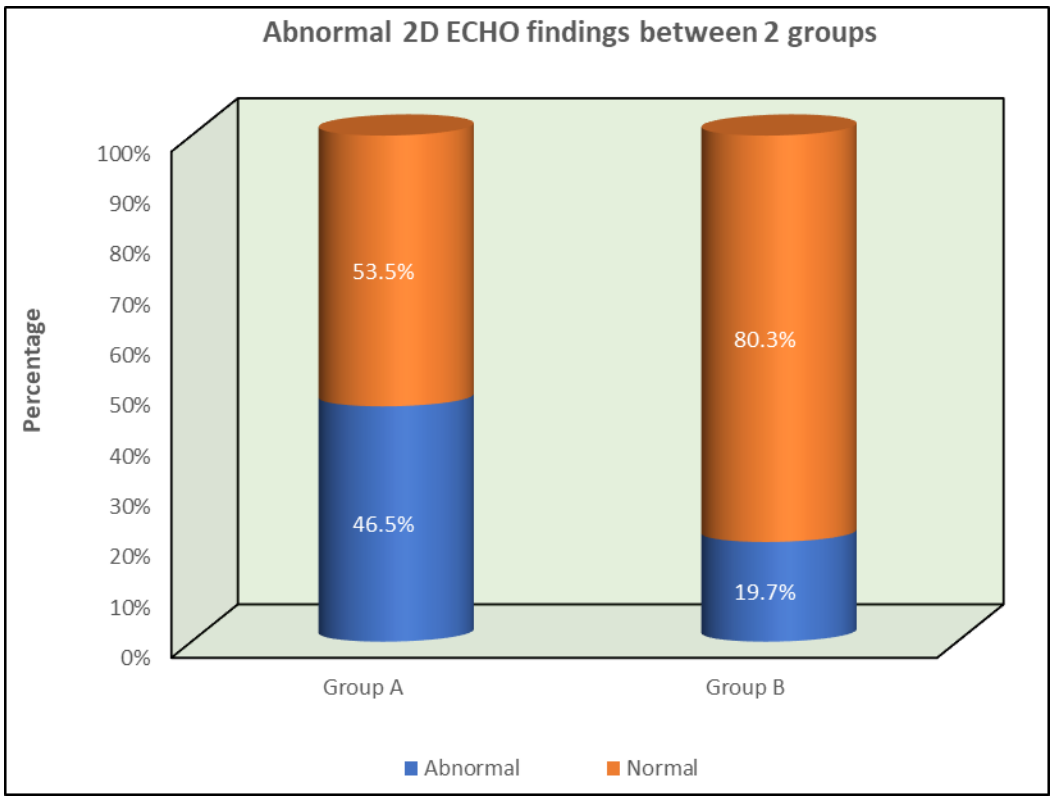
Group B: Normotensive Pregnant Women

Figure no 38 Mean Baby's Birth Weight (in Kgs) between HDPs and normotensive group

TABLE NO.15 Comparison of Abnormal 2D ECHO findings between 2 groups using Chi Square Test						
Variable	Category	Group A		Group B		p-value
		n	%	n	%	
2D ECHO	Abnormal	33	46.5%	14	19.7%	0.001*
	Normal	38	53.5%	57	80.3%	

* - Statistically Significant

Group A demonstrated with higher prevalence of Abnormal 2D ECHO findings (46.5%) as compared to Group B (19.7%) and the difference between 2 groups was statistically significant at p=0.001.



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

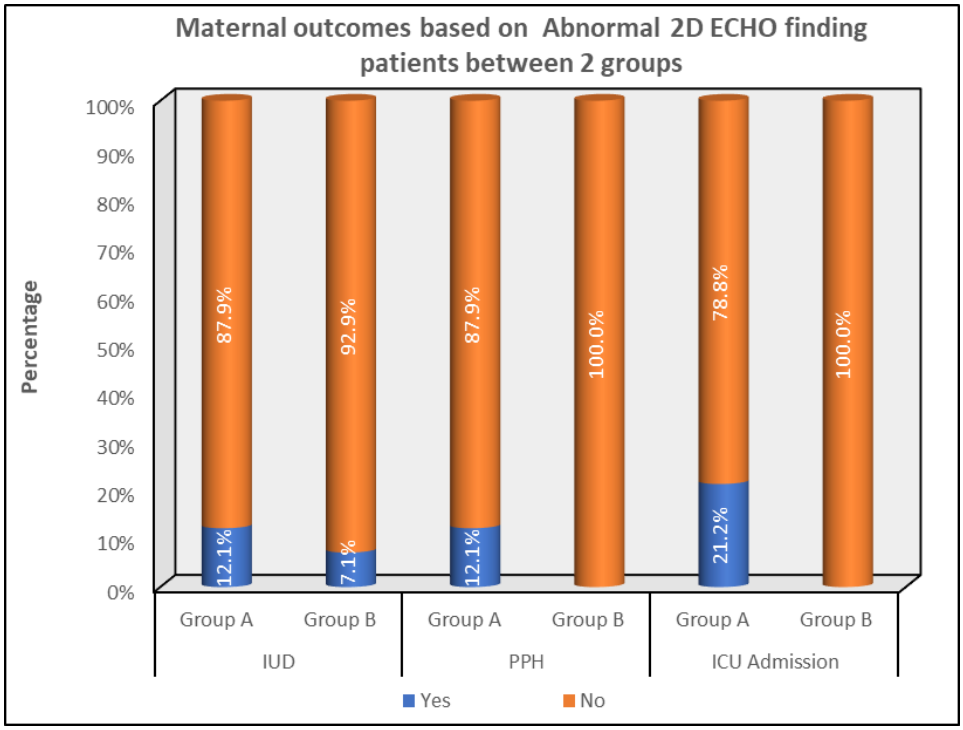
Figure no 39 Abnormal 2D ECHO findings between HDPs and normotensive group

TABLE NO.16 Comparison of Maternal outcomes based on Abnormal 2D ECHO finding patients between 2 groups using Chi Square Test

Variable	Category	Group A		Group B		p-value
		n	%	n	%	
IUD	Yes	4	12.1 %	1	7.1%	0.61
	No	29	87.9 %	13	92.9%	
PPH	Yes	4	12.1 %	0	0.0%	0.17
	No	29	87.9 %	14	100.0 %	
ICU Admission	Yes	7	21.2 %	0	0.0%	0.04 *
	No	26	78.8 %	14	100.0 %	

* - Statistically Significant

Among those samples with Abnormal 2D ECHO findings, a significantly higher proportion of women in Group A required ICU Admission (21.2%) as compared to Group B, with no cases reported which required ICU admission. This difference in the requirement of ICU Admission among abnormal 2D ECHO findings patients between 2 groups was statistically significant at p=0.04. However, other maternal outcomes like Intra-Uterine Death and Post-Partum Haemorrhage found no significant correlation with abnormal 2D ECHO findings among 2 groups.



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 40 Maternal outcomes based on Abnormal 2D ECHO findings between HDPsand normotensive group

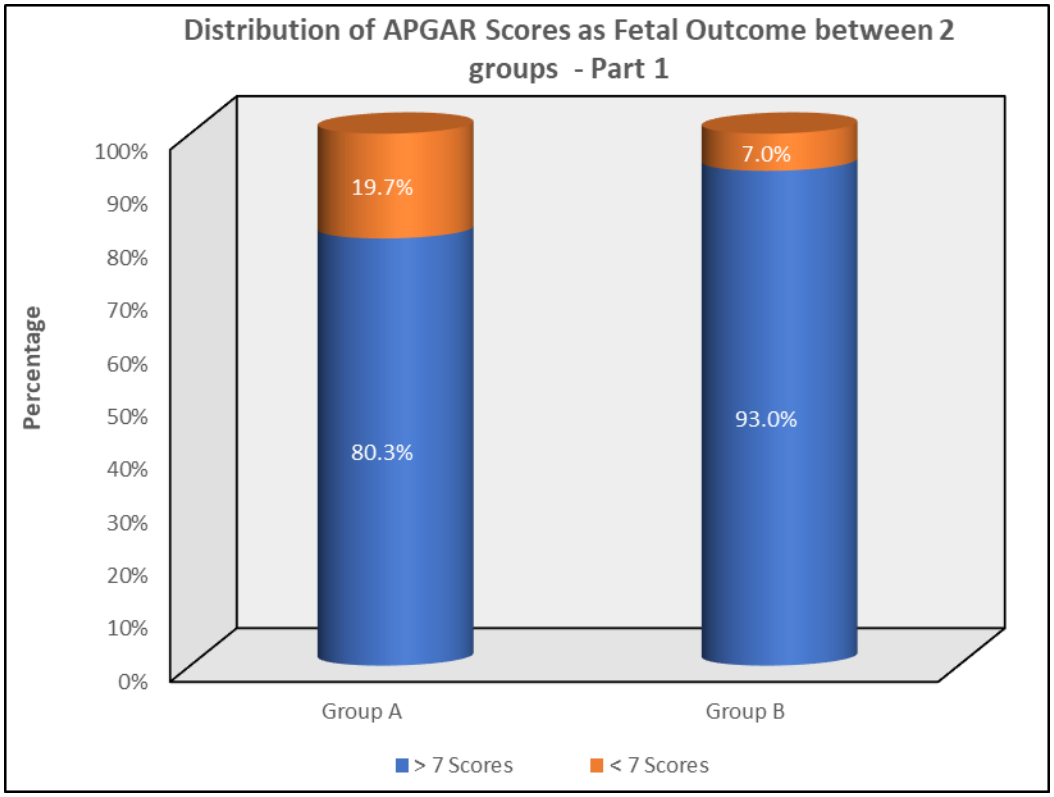
TABLE NO.17 Comparison of Fetal outcomes among Abnormal 2D ECHO finding patients between 2 groups using Chi Square Test

Variable	Category	Group A		Group B		p-value
		n	%	n	%	
APGAR SCORE	Yes	25	75.8%	13	92.9%	0.17
	No	8	24.2%	1	7.1%	
Resp. Fetal Distress	Yes	9	27.3%	3	21.4%	0.67
	No	24	72.7%	11	78.6%	
Meconium Stained Liquid	Yes	1	3.0%	1	7.1%	0.52
	No	32	97.0%	13	92.9%	
Fetal Growth Retardation	Yes	7	21.2%	0	0.0%	0.04*
	No	26	78.8%	14	100.0%	
NICU Admission	Yes	13	39.4%	4	28.6%	0.45
	No	20	60.6%	10	71.4%	

TABLE NO.17 Comparison of Fetal outcomes among Abnormal 2D ECHO finding patients between 2 groups using Chi Square Test						
Variable	Category	Group A		Group B		p-value
		n	%	n	%	
Neonatal Mortality	Yes	3	9.1%	1	7.1%	0.83
	No	30	90.9 %	13	92.9%	

* - Statistically Significant

Among the Fetal Outcomes, a significantly higher proportion of samples in Group A reported with Fetal GrowthRetardation (21.2%) as compared to Group B, with no cases reported with the same. This difference in the Fetal Growth Retardation among abnormal 2D ECHO findings patients between 2 groups was statistically significant at p=0.04. However, other Foetal outcomes like Cry After Birth, Respiratory Fetal Distress, Meconium Stained Liquid, NICU Admission and Neonatal Mortality found no significant correlation with abnormal 2D ECHO findings among 2 groups.

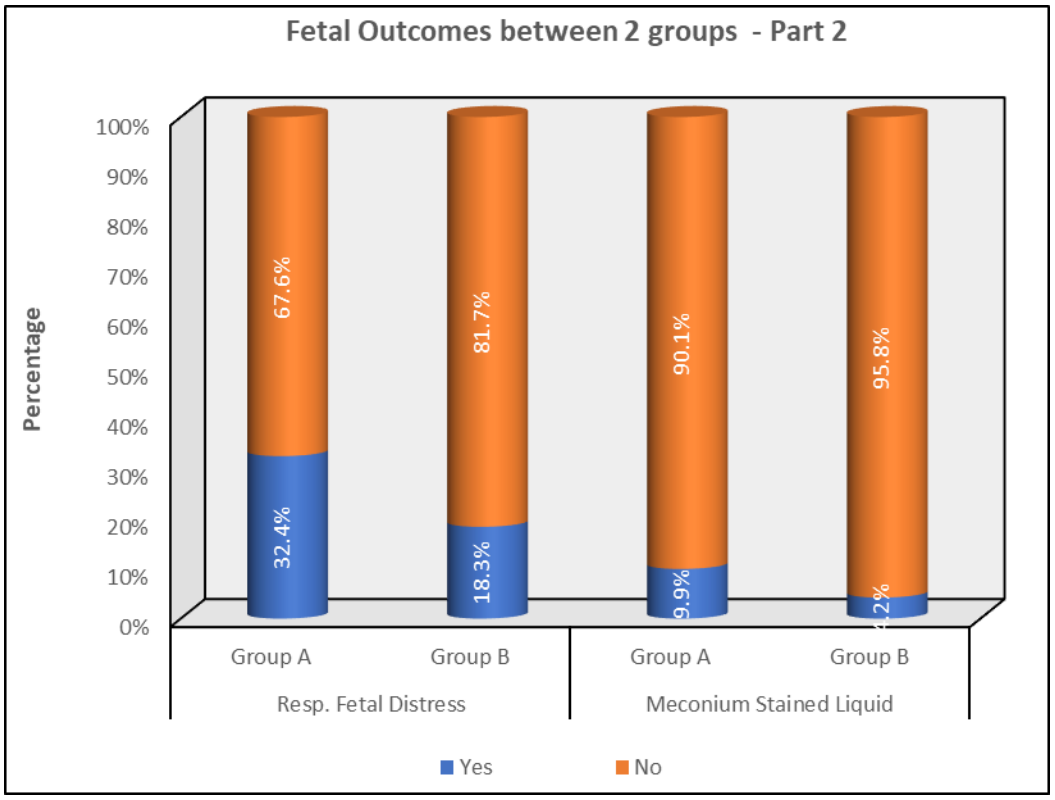


Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 41 Distribution of APGAR Scores as Fetal Outcome between HDPs and

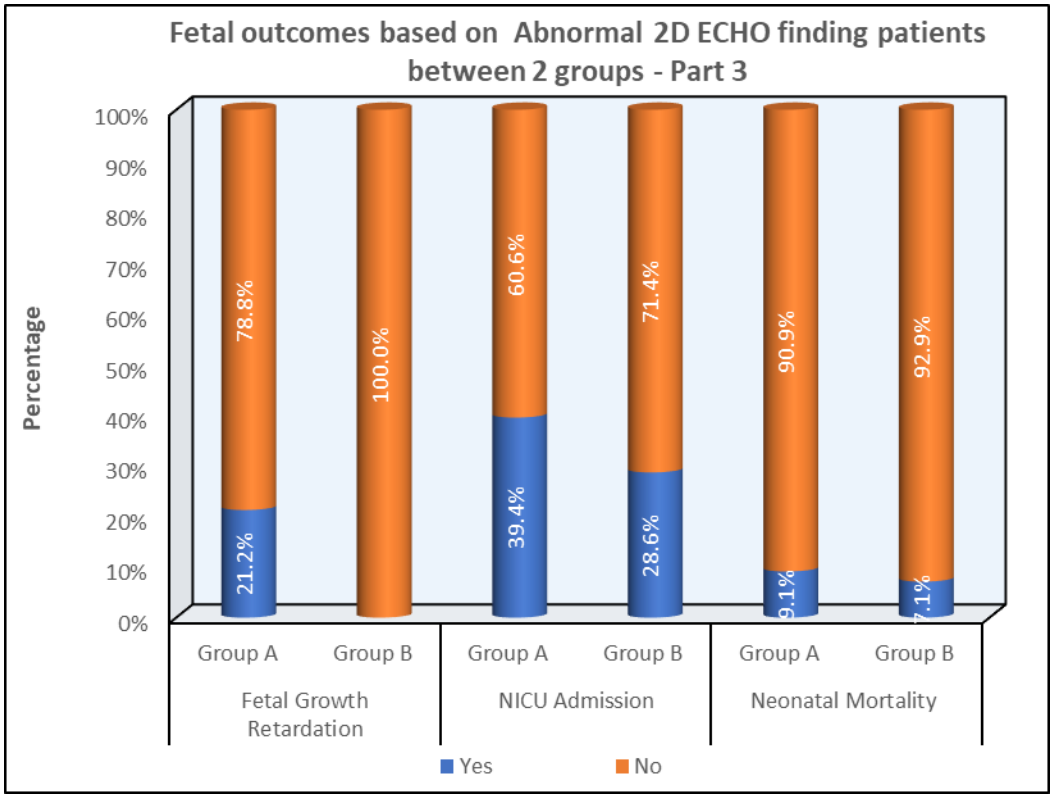
normotensive group- part 1



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 42 Fetal Outcome between HDPs and normotensive group- part 2



Group A: Hypertensive Pregnant Women &

Group B: Normotensive Pregnant Women

Figure no 43 Fetal Outcomes based on abnormal 2D ECHO findings patients between HDPs and normotensive group- part 3

TABLE NO.18 Comparison of mean Baby's Birth Weight (in Kgs) between 2 groups with Abnormal 2D ECHO findings using Independent Student t Test						
Parameter	Groups	N	Mean	SD	Mean Diff	p-value
Baby Birth Weight	Group A	33	2.35	0.54	-0.25	0.17
	Group B	14	2.60	0.59		

The mean Baby's birth weight in Group A was relatively lesser (2.35 ± 0.54 Kgs.) as compared to Group B (2.60 ± 0.59 Kgs.). However, the mean difference between 2 groups with Abnormal2D ECHO findings was not statistically significant.

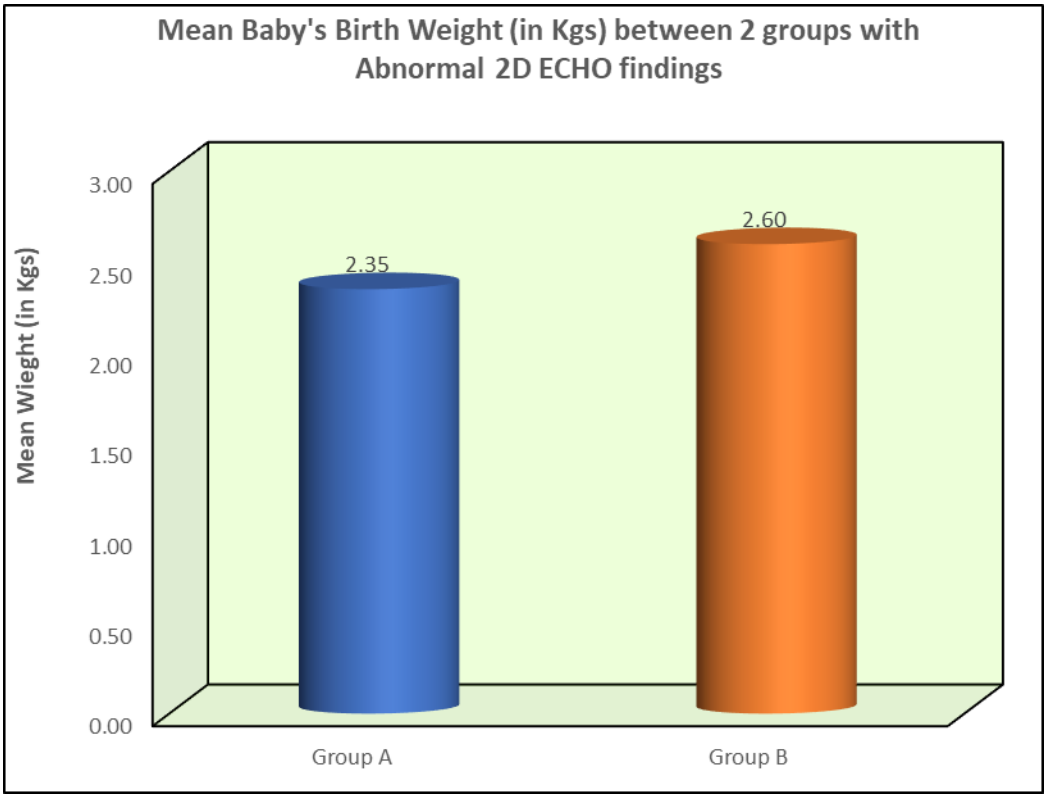


Figure no 44 Mean Baby's Birth Weight (in Kgs) between HDPs and normotensive groups with Abnormal 2D ECHO findings

DISCUSSION

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The Hypertensive Disorders of pregnancy include preeclampsia characterized by placental under perfusion, release of vascular toxins and other anti-angiogenic factors leading to widespread endothelial dysfunction and hypertension, proteinuria and edema. Echocardiography is a very useful tool for detecting anatomical, hemodynamic, and functional abnormalities in the cardiovascular system that occurin preeclamptic pregnancies[122]. Understanding these alterations could improve our comprehension of the pathophysiological causes of the condition and aid in the treatment of these patients This study was conducted to study the echocardiographicchanges in pregnant women with HDPs compared to normal pregnant women and also study the correlation between Echocardiographic abnormalities and fetal and maternal outcomes of pregnant women with HDPs and compare this with normal pregnant women. The Hypertensive (Group A) and Normotensive (Group B) Pregnant Women were in the age range of 18-37, with a gestational age of 31-42 weeks. Of the 142 patients in our study, 71 women (Group A) had hypertensive disorders of pregnancy; 34 of these women (48%) were primigravida and 37 of thesewomen (52%) were multigravida. In Group A & Group B, majority of the pregnant women were Multigravida (52.1% & 62.0%) and there was no significant differencein the obstetric scores between 2 groups. In both groups, there were no pregnant women with smoking history and all were from Asian background. Primigravidity isthe risk factor for the development of pre-eclampsia, according to a number of studies that looked into the risk of pre-eclampsia, which was traditionally thought to be a disease of the first pregnancy. However, multigravida women are also susceptible to the development of pre-eclampsia, even though the risk factors are less well defined**.Sibai et al.'s**[123] study, which showed similar distribution among multigravida women in a large multicentric

clinical trial conducted in the United States. In another study by **Bej P et al.**[124] in tertiary centre, Delhi bivariate analysis revealed that nulliparity, primigravida, twin pregnancies, a poor obstetrics history, and a history of abortion were not significantly associated with the development of PE . This finding is comparable to the distribution of women with HDP among multigravida as observed in our study. Majority of the Hypertensive Pregnant Women in this study had PE with Severe Features (54.9%). Similar outcomes for Asian women were discoveredby **Ling HZ et al.**[125] in a UK study, which stated that black and Asian women have higher peripheral vascular resistance (PVR) and lower cardiac output (CO) than do white women, with CO increasing through an increase in heart rate (HR) brought onby a static or declining stroke volume (SV). There is substantial evidence of racial disparities in the risks of unfavorable pregnancy outcomes and the long-term development of cardiovascular illnesses, including PE, among Asian pregnant women, which can be attributed to this least favourable hemodynamic profile. Less than a third of the Hypertensive pregnant women had high blood pressure readings in the third trimester whereas only a small percentage had high blood pressure in thesecond semester. Pre-eclampsia usually manifests clinically between 36 weeks of pregnancy and birth in the majority of instances. **Redman et al.**[126] review observes that while all forms of pre-eclampsia—whether primarily placental (early-onset) or maternal (late-onset)—are combined into a single diagnosis, it is acknowledged that the pathways to disease differ significantly between placental and maternal disease,aside from the shared presence of a placenta.

Our study reported comorbidities in the women with HDP that could aggravate their hypertensive state. In 3rd Trimester, H/o Anemia was prevalent in similar rates in both Group A and Group B (2.8% in both groups). The significance of this finding is related to the increased risk of preeclampsia in anaemic women. According to a study conducted in Eastern Sudan by **Ali AA et**

al.[127], anemic women with hypertension are more likely to suffer complications such placental abruption and maternal organ damage, as well as a more severe type of preeclampsia. **Johnson A et al**.[128] in a recent study in Tamilnadu, India found that the HDPs accompanied by anemia increases the risk of developing preeclampsia. A small percentage of patients in group A had a positive history of asthma and Hypothyridism. Hypothyroidism may be an independent risk factor for preeclampsia and fetal growth restriction. In a recent Nigerian study, **Nwabudike P et al.**[129] discovered that pregnant women who had hypothyroidism, subclinical hypothyroidism, or elevated TSH levels had a higher chance of developing hypertension. Pedal oedema was found in more than a third of Hypertensive women (Group A); mostly Grade I oedema (15.5%) and a small percentage (4.2%) having abdominal wall oedema but these findings were not observed among normotensive pregnant women. **Mac Gillivray et al.**[130] in a review reported that oedema is associated with a higher incidence of pre-eclampsia. According to several decades' worth of clinical findings, low extremity edema is typically the initial indication of future PE, and it often progresses into a more widespread form, as a study documented by **P'eter Tamas et al.**[131] Sometimes, after a few days or weeks, the blood pressure starts to rise. The echocardiographic findings from our study find consonance with the available literature. Statistically significant differences were noted in the mean clinical and Echocardiographic parameters between the two BP (94.65 ± 7.34), the mean MAP (112.00 ± 8.10) in Group A was significantly higher than in Group B. However, the mean LV ESV, mean LV EDV, mean Stroke volume and mean LV mass did not demonstrate significant difference between 2 groups. The mean percentage of Ejection Fraction in Group A was significantly lesser (61.52 ± 5.13) as compared to Group B (65.59 ± 4.49). These results were in line with the retrospective study carried out in the USA by **Kathryn et al.**[132], which revealed that 17 out of 57 pre-eclamptics had lower LVEF. In the same way, a

study carried out in Kanpur by **Zaman N et al.**[122] similarly demonstrated that preeclamptic patients had increased systolic and diastolic blood pressure, mean arterial pressure (MAP), and total vascular resistance (TVR) differences with regard to control that were statistically significant. EF was also reported to be lesser than normotensive pregnant women. However, **Mostafavi A et al.**[133] in an Iranian study found no significant difference in the EF, likely as a result of the smaller sample size. Nevertheless, the authors acknowledged that further disruption in ventricular function, followed by yet another reduction in the LVEF, is possible in the context of pre- eclampsia and increased vascular resistance, and that this can continue long after delivery. In their review, **Pabon MA et al.**[134] found that, even after taking into consideration the intermittent development of coronary artery disease, women with a history of hypertensive disorders of pregnancy (HDPs) have a nearly two-fold increased risk of developing HF in the future as compared to other parous women. The HDPs predict increased HF risk with both decreased and maintained ejection fraction. This makes the finding prognostic.

Comparison of mean Diastolic parameters between 2 groups showed the mean E Wave (0.82 ± 0.45), mean A Wave (0.59 ± 0.35) in women of Group A were significantly higher than that seen on 2D Echo in the normotensive pregnant women in Group B. Although these values are lesser than that reported in other studies the observed differences are similar..In a comparable direction, **Shivnanjiah et al.**[135] found that in pre-eclamptic subjects, the mean E-wave velocity was 0.98 ± 0.14, while in normotensive women, it was 0.66 ± 0.09 ($P < 0.0001$). This suggests that there wasa greater pressure gradient across the mitral valve during early passive filling. The study was conducted in Bangalore. Atrial systole is significant since the mean peak A-wave velocity in pre-eclamptic patients was 0.70 ± 0.12 compared to 0.56 ± 0.03, $P < 0.0001$). This was similar to the

study by **Solanki R et al.**[136] from Baroda, where the E-wave velocity of the pre-eclamptic patients was 1.023 ± 0.1926 compared to the normotensives 0.675 ± 0.137, and the pre-eclamptic group had a higher A wave velocity of 0.775 ± 0.278 compared to the normotensives 0.500 ± 0.130. Preeclamptic participants' elevated E wave velocities indicate changes in the hypertrophic ventricle's passive myocardial compliance and a larger transmittal pressure gradient during early passive filling. The elevated summit A wave velocity in pre-eclamptic patients indicates that atrial systole plays a more significant function in these women's hypertrophied ventricular filling. Pregnant hypertension women had much lower transmital annular velocities (cm/s) on tissue Doppler (septal and lateral E') than normotensive women. The mean Septal e' in Group A was significantly lesser (0.119 ± 0.163) as compared to Group B (0.156 ± 0.133) and the mean difference was statistically significant at $p<0.001$. The mean Lateral e' in Group A was significantly lesser (0.121 ± 0.030) as compared to Group B (0.132 ± 0.040). Similar decreased velocities in hypertensive pregnant women as compared to the normotensive controls were reported by a study by **Muthyala T et al.**[137] from Chandigarh. Diastolic measures such LAVi, mitral E, mitral A, E/A, DT, Sep E', lateral e', E/e', and TR velocity were found to be considerably abnormal in HDP women when compared to normotensive women, according to a recent retrospective study conducted in the USA by **Laith Alhuneafat et al.**[138]. When comparing the PRE and SPE groups with controls in their unadjusted and adjusted regression models, DT and mitral annular septal and lateral e' velocities showed a graded decrease. The SPE group had the lowest annular velocities and shortest DT, both of which were indicative of a greater decrease in the LV's diastolic compliance.Comparison of Mode of Delivery between 2 groups showed that Group A had a higher occurrence of emergency LSCS (67.6%) compared to Group B (45.1%) as also preterm vaginal Delivery. Pre-eclampsia with Severe Features was most predominantly seenin

Group A (28.2%) and was the most prevalent maternal indication of delivery in our study. Thisis consistent with research from Telangana by **Kalpana P** et al.[139], which found that pregnancy-induced hypertension and eclampsia accounted for 32.5% of maternal emergency LSCS patients. **Nagar et al.**[140] discovered in an Udaipur study that out of 500 pregnant women exhibiting preeclamptic symptoms, 30% underwent an emergency Caesarean section (CS) and 12% underwent an elective CS. In a retrospective study conducted in Belgrade, **Babović et al**.[141] also observed that, compared to our research, a higher percentage of CS procedures were performed on pregnant women whose preeclampsia and FGR complicated their pregnancy—58.5% of emergency CS procedures and 41.5% of elective procedures. In addition, a study conducted in Atlanta by **Boulet SL et al**.[142] found that in high-risk pregnancies, including preeclampsia, delivery is typically induced prematurely and ended instrumentally or surgically for the benefit of the mother and/or the fetus. The results of our study are further supported by a recent Saudi study by **Wahabi H et al.**[143], which found a number of risk factors linked to emergency cardioversion (CS) in pregnant Saudi women. The study also projected that pregnant women with hypertensive disorders had a 176% increased risk of emergency CS (OR 2.76, 95% CI 1.35–5.63).One of the main causes of severe maternal morbidity (SMM) during pregnancy is hypertensive disorders of pregnancy (HDP).[21] Group A exhibited significantly more maternal complications overall, with statistically significant differences in lower respiratory tract infections, pulmonary oedema, and wound gaping as compared to Group B. Similarly, **Kaur H etal.**[144] from the UK discovered that severe pre-eclampsia is related to acute pulmonary edema mostfrequently in pregnancy; a retrospective study by **Karanth S et al.**[145] from Bangalore**,** reported that a higher chance of preeclampsia, in 14.4% of their patients and increased rates of lower segment caesarean section (LSCS) in 46.2%, were observed in those pregnant women with respiratory complications.

The results from our study, in line with these findings suggests that mothers having respiratory disorders had to be antenatally checked as LSCS chances are high. Inline with that reported in earlier literature, in our study, adverse maternal outcomes like Postpartum Haemorrhage (PPH), higher need for ICU admission, and the proportion of Intra- uterine death were higher among Group A. According to **Ramachandra Bhat PB et al.**[146], the two most common primary diagnoses at the time of ICU admission in Coastal Karnataka were significant obstetric hemorrhage (27.7%) and pregnancy-related hypertension with associated consequences (26.2%). According to **Poon LC** [147] , maternal and perinatal morbidity and mortalityare most frequently caused by hypertensive disorders of pregnancy (HDP), which account for 16% of maternal mortality in high-income nations and roughly 25% in low- and middle-income countries.

Among the foetal outcomes, low Apgar score <7 (80.3%), but increased Respiratory Fetal Distress (32.4% vs 18.3%), Fetal Growth Retardation (15.5% vs 0.0%) and NICU Admission(40.8% vs 22.5%), low mean birth weight (2.49 ± 0.56 Kgs. Vs 2.69 ± 0.50 Kgs) hadbeen reported in higher occurrence in Group A as compared to Group B.. Similar data from Ethiopian settings were observed by **Gudayu T et al.**[148], who found that the following factors were independently linked to a low Apgar score: the type of anesthesia, amniotic fluid stained with meconium, antepartum hemorrhage, time from skin incision to delivery, pregnancy-inducedhypertension, and fetal birth weight less than 2.5 kg.

Desalegn et al.[149] in another retrospective case– control study from Ethiopia, also reported that nearly a fifth of mothers whose newborns had low Apgar scores had pre-eclampsia. In a Pakistan, a study by **Samia Arif et al.**[150] likewise found that most infants in the hypertension group had an Apgar score between 6 and 8, whereas most infants in the control group had an Apgar score between 8 and 10. Overall, the Apgar score of babies delivered to normotensive moms was higher than

that of kids born to hypertensive mothers group. This difference may have resulted from fetal impairment linked to low birth weight and development restriction in hypertensive patients.In a significant nationwide cohort research carried out in the United States, which comprised 156,681 infants born to mothers with preeclampsia, **Stevens, W. et al.**[151] similarly revealed that the preeclamptic group had a higher likelihood of RDS than the non-preeclamptic group (6.6% vs. 1.9%). **Marins LR et al.**[152] in a review of evidence, also reported that foetal growth restrictionis the most frequent neonatal complication in newborns (NB) with hypertensive mothers, as seen among in our study. Further a systematic review and meta-analysis by **Maher et al**.[153] and a South African prospective cohort study by **Nathan HL et al.**[154] also supported these findings. According to a review by **Bokslag A et al.**[155], hypertensive disorders of pregnancy (HDP) and fetal growth restriction (FGR) are two of the most important obstetrical syndromes linked to morbidity and death in both the mother and the fetus. **Di Martino DD et al**.[156], in an Italian study, has shown that early shallow trophoblastic invasion, poor placental development, poor fetal nutrition, and ultimately placental oxidative stress are the causes of the severe complications that these pregnancies develop, first on the fetal side (fetal growth restriction), and then on the maternal side (hypertension). Pregnancies impacted by HDP-FGR, as predicted, had the worst obstetric outcomes, including the highest rate of cesarean sections and infant mortality, as well as the lowest gestational age at delivery. An important finding of our study that adds to the existing evidence was the comparison of Fetal outcomes among patients with Abnormal 2D ECHO findings which revealed that the Fetal Growth Retardation among abnormal 2D ECHO findings patients between 2 groups was statistically significant. This study has resemblance to that conducted in Gujarat by **Parikh PM et al.**[157], wherein patients exhibiting atypical structural echocardiographic results were shown to have a poor feto-maternal outcome. Pregnant women inthis group experienced higher

rates of morbid occurrences and deterioration. Decreased uterine artery blood flow as a result of increased peripheral vascular resistance during pregnancy is the reason for the higher incidence of FGR and fetal demise, which were 53.1% and 18.7%, respectively. Fetomaternal outcomes were better in women with only disturbed heart function than with those with both structural and functional abnormalities. Of the three groups, those with a normal echocardiographic report have the best prognosis. Further supporting our study findings is another Indian study by **Verma S et al.,**[158] which found that 13 (8.1%) women in the HDP group had mild systolic dysfunction, 8 (20%) had severe pre-eclampsia, and 5 (33.3%) had eclampsia. These women also reported significantly lower values of left ventricular ejection fraction and higher values of N-terminal pro B-type natriuretic peptide (NT-proBNP) levels in HDP women who experienced maternal complications and had unfavorable neonatal outcomes. Women with HDPs had higher rates of maternal and neonatal morbidity, according to our research. This is consistent with other research.

According to a comprehensive epidemiological study conducted in the United States by **WaarenS et al**.[159] preeclampsia raised the likelihood of an unfavorable event for mothers and from 7.8% to15.4% for infants. The overall evidence from the current research can shape earlier detection of HDP for better maternal and fetal outcomes in India.

SUMMARY

The Hypertensive (Group A) and Normotensive (Group B) Pregnant Women were in the agerange of 18-37, with a gestational age of 31-42 weeks. Among the 142 patients examined, (Group A) 71 women had hypertensive disorders ofpregnancy; of them 34 women (48%) were primigravida and 37 women (52%) were multigravida. Majority of the Hypertensive Pregnant Women in this study had PE with Severe Features (54.9%). In 3rd Trimester, H/o Anemia was prevalent in similar rates in both Group A and Group B (2.8%in both groups). Pedal oedema was found in more than a third of Hypertensive women (Group A); mostly GradeI oedema (15.5%) and a small percentage (4.2%) having abdominal wall oedema but these findings were not observed among normotensive pregnant women. The mean Heart rate (94.20 ± 9.22), mean Systolic BP (145.41 ± 13.23), mean Diastolic BP (94.65 ± 7.34), the mean MAP (112.00 ± 8.10) in Group A was significantly higher than in Group B. The mean percentage of Ejection Fraction in Group A was significantly lesser (61.52 ± 5.13) as compared to Group B (65.59 ± 4.49). Comparison of mean Diastolic parameters between 2 groups showed the mean E Wave (0.82 ± 0.45), mean A Wave (0.59 ± 0.35) in women of Group A were significantly higher than that seenon 2D Echo in the normotensive pregnant women in Group B. The mean Septal e' in Group A was significantly lesser (0.119 ± 0.163) as compared to Group B(0.156 ± 0.133) and the mean difference was statistically significant at $p<0.001$. The mean Lateral e' in Group A was significantly lesser (0.121 ± 0.030) as compared to Group B (0.132 ± 0.040). Comparison of Mode of Delivery between 2 groups showed that Group A had a higher occurrence of emergency LSCS (67.6%) compared to Group B (45.1%) as also preterm vaginal Delivery. Pre-eclampsia with Severe Features was most predominantly

seen in Group A (28.2%) and was the most prevalent maternal indication of delivery in our study. Group A exhibited significantly more maternal complications overall, with statistically significant differences in lower respiratory tract infections, pulmonary oedema, and wound gaping as compared to Group B. Maternal Outcome like Postpartum Haemorrhage (PPH), higher need for ICU admission, the proportion of Intra-uterine death were higher among Group A. Respiratory Fetal Distress, higher neonatal ICU admissions, Fetal growth retardation and low birth weight and Maternal Outcome like higher need for ICU admission

Among the foetal outcomes, reduced cry after birth (80.3%), but increased Respiratory Fetal Distress (32.4% vs 18.3%), Fetal Growth Retardation (15.5% vs 0.0%) and NICU Admission (40.8% vs 22.5%), low mean birth weight (2.49 ± 0.56 Kgs. Vs 2.69 ± 0.50 Kgs) had been reported in higher occurrence in Group A as compared to Group B. Comparison of Fetal outcomes among patients with Abnormal 2D ECHO findings revealed that the Fetal Growth Retardation among abnormal 2D ECHO findings patients between 2 groups was statistically significant.

CONCLUSIONS

Identifying predictors for adverse cardiovascular and obstetric outcomes in pregnant women is a major public health priority. There is insufficient information about the role of echocardiography in pregnant women without known structural heart disease and whether abnormal echocardiographic findings predict worse obstetric outcomes in this population. The findings in women with Hypertensive disorders of Pregnancy (Group A) demonstrated higher prevalence of Abnormal 2D ECHO findings as compared to normotensive pregnant women.

This study also showed significantly higher poor feto-maternal outcome in patients with abnormal echocardiographic findings among Group A as compared to normotensive women without abnormal 2D ECHO findings, which highlights the need for appropriate antenatal surveillance, and postnatal management of these high risk patients.

RECOMMENDATIONS

The results of our study will aid in the early detection of structural and functional cardiovascularabnormalities and management of high risk pregnant women who have high blood pressure. Hence lowering the possibility of complications leading to fetomaternal morbidity and mortality.

LIMITATIONS

1. Non availability of 2D Speckle Tracking Echocardiography software technology has limited the study impact into evaluating high risk patients .
2. Cardiac MRI is non-invasive better investigative tool which is expensive and all patients are not affortable .
3. Element of bias: subjective bias, machinery errors in measurement of echocardiogram tissue doppler parameters.
4. Less sample size: sample size of the study being less makes it necessary to take more samples into consideration to take into account the significance of 2Decho as one of the HDPSs investigation procedures.

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ANNEXURE- I

CONSENT

FORM

B.L.D.E. (DEEMED TO BE UNIVERSITY) S.H.R.I. B.M..PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, BIJAPUR-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned,_____, D/O W/O_____, aged ___years, ordinarily resident of_____do hereby state/declare that Dr MADDERLA SOWMYA of Shri. B. M. Patil Medical College Hospital and Research Centre have examinedme thoroughly on_____at_____(place) and it has been explained to me in my own language that I am undergoing Echocardiography as a screening tool for cardiac disease.Further, Dr. Madderla sowmya has informed me that he/she is conducting a dissertation/research titled "**A PROSPECTIVE COMPARATIVE STUDY OF SIGNIFICANCE OF TWO-DIMENSIONAL ECHOCARDIOGRAPHY IN HYPERTENSIVE DISORDERS OF PREGNANCY(H.D.P.) AND NORMAL PREGNANCY AND ITS EFFECT ON FETOMATERNAL OUTCOME.**" under the guidance of Dr.Shreedevi Kori requesting my participation in the study. The Doctor has also informed me that during the conduct of this procedure, adverse results may be encountered. Among the above complications, most of them are treatable but are not anticipated; hence there is a chance of aggravation of my condition, and in rare

circumstances, it may prove fataldespite the anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this study would help in the evaluation of the results of the study, which is a useful reference to the treatment of other similar cases shortly, and also,I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made, photographs video graphs taken upon me by the investigator will be kept secret and not assessed

by a person other than my legal hirer or me except for academic purposes. The Doctor did informme that though my participation is purely voluntary, based on the information given by me, I canask for any clarification during the course of treatment/study related to diagnosis, the procedure of treatment, result of treatment, or prognosis. At the same time, I have been informed that I canwithdraw from my participation in this study at any time if I want, or the investigator can terminate me from the study at any time study but not the procedure of treatment and follow-up unless I request to be discharged. After understanding the nature of dissertation or research, diagnosis made, mode of treatment, I the undersigned Smt. under my full conscious

state of mind agree to participate in the said research/dissertation.

Date: Signature of the patient:

Place: Signature of Doctor:

ANNEXURE -II

PROFORMA

A PROSPECTIVE COMPARATIVE STUDY OF THE SIGNIFICANCE OF TWO-DIMENSIONAL ECHOCARDIOGRAPHY IN HYPERTENSIVE DISORDERS OF PREGNANCY(HDP) AND NORMAL PREGNANCY AND ITS EFFECT ONFETOMATERNAL OUTCOME

NAME:

A.G.E.:

IP NUMBER:

ADDRESS AND PHONE

NUMBER:CHIEF

COMPLAINTS: OBSTETRIC

HISTORY:

MARITAL HISTORY:

LAST MENSTRUAL PERIOD:

EXPECTED DATE OF

DELIVERY:PERIOD OF

GESTATION:

A.N.C.:

1ST TRIMESTER:

2ND TRIMESTER:

3RD TRIMESTER:

RELATED DRUG

HISTORY:PAST

HISTORY: PERSONAL

HISTORY:

GENERAL PHYSICAL EXAMINATION:

HEIGHT: WEIGHT: B.M.I.:

TEMPERATURE: PULSE: BLOOD PRESSURE:

PALLOR: BREAST:

ICTERUS: SPINE:

CYANOSIS: THYROID:

CLUBBING:

LYMPHADENOPATHY:

EDEMA:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

PER ABDOMEN:

P/S:

P/V:

CLINICAL PARAMETERS

Heart rate						
Systolic bp						
Diastolic bp						
Mean arterial pressure						

systolic dysfunction parameters: Ejection fraction						
LV ESV						
LV EDV						
Diastolic dysfunction Doppler parameters: E wave						
A wave						
E/A ratio						

Septal e.						
Lateral e’						

	Left ventricular ejection fraction	Stoke volume	Left ventricular mass
Normal pregnant women			
Gestational hypertension			
Mild Preeclampia			
Severe Preeclampsia			
Imminent eclampsia			

Maternal Obstetric outcome:

Time of delivery:

Final outcome: normal delivery/ instrumental delivery/ LSCS

If, then indication:

Maternal complications:

Congestive cardiac failiure: yes no

Atrial fibrillation: yes no

Hypertensive crisis: yes no

ICU admission : yes no

If yes details

Maternal mortality : yes or no

Fetal outcome:

Baby birth weight Baby cry after birth: yes no

APGAR

Need for NICU admission yes /

noNeonatal mortality : yes / no

MASTERCHART

Sl	Groups	NAME	Obstetric History	Gestation	HTN Group	3rd Trimester	Gen. Physical Exam	HR	SBP	DBP	MAP	EF (%)	LV EST	LV EDT	Stroke Vol	LV Mass	E Wave	A Wave	E/A Ratio	Septal E'	Lateral E'	2D ECHO	Mode of Delivery	Indications	Maternal	IUD	PPH	ICU Ad	Days Di	Cr q Af	Resp	MSL	FGR	IUD	NICU	Neona
1	Group A	RENUKADAI KANTEPPA HORATAGI	Primigravida	36	PE with Severe Features	Uneventful	Pedal Oedema Grade I	90	150	100	117	65.0	22.0	64.0	47.0	126.02	1.10	1.12	0.90	0.08	0.11	Normal	Full Term Vaginal	PE with Sev	Nil	No	No	No	2.9	No	Yes	No	Yes	No	Yes	No
2	Group A	JYOTHI HUSANAPPA KAMBALE	Primigravida	38	PE with Severe Features	H/o High BP	Pedal Oedema Grade II	84	170	110	130	60.0	19.0	56.0	38.0	94.54	1.50	0.67	2.24	0.07	0.14	Normal	Emergency LSCS	PE with Sev	Nil	No	No	Yes	1.5	Yes	Yes	Yes	Yes	No	Yes	No
3	Group A	AFREEN J SARAFULE	Primigravida	35	PE with Severe Features	H/o High BP	Pedal Oedema Grade II	110	160	90	113	35.0	26.0	50.0	41.0	110.20	1.10	0.71	1.55	0.06	0.12	Abnormal	Emergency LSCS	PE with Sev	Pulmonary edema	No	Yes	Yes	2.1	No	Yes	No	No	No	Yes	No
4	Group A	JYOTHI RAVI SUDHAKAR	Multigravida	35	PE with Severe Features	Uneventful	Normal	80	170	90	117	60.0	31.0	74.0	66.0	120.02	1.50	0.70	1.86	0.06	0.13	Abnormal	Emergency LSCS	H/o Previous	Nil	No	No	No	2.0	Yes	Yes	No	No	No	Yes	No
5	Group A	PARVEEN BABU GOUS SHAIKH	Primigravida	38	PE with Severe Features	Uneventful	Abdominal Wall Oedema	82	140	100	113	60.0	23.0	63.0	46.0	112.60	0.55	0.40	1.38	0.00	0.13	Normal	Emergency LSCS	Cephalopelvic	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
6	Group A	SAVITA SIDDARAM HULIMANI	Multigravida	41	PE with Severe Features	Uneventful	Pedal Oedema Grade I	100	160	90	113	60.0	24.0	58.0	34.0	112.54	0.11	0.11	1.00	0.12	0.14	Normal	Emergency LSCS	Non-progress	Tachycardia	No	No	No	2.0	Yes	No	No	No	No	No	No
7	Group A	PRIYANKA SATISH HOSAMANI	Primigravida	34	PE with Severe Features	H/o High BP	Normal	90	140	80	110	65.0	26.0	72.0	47.0	132.10	0.12	0.13	0.92	0.10	0.13	Normal	Emergency LSCS	Severe Oligo	Nil	No	No	No	2.2	Yes	Yes	No	No	No	Yes	No
8	Group A	MEENAKSHI HANAMANT WALIKAR	Multigravida	36	PE without Severe Features	Uneventful	Pallor	126	134	90	105	57.0	23.0	76.0	51.0	152.00	1.07	0.83	1.19	0.00	0.03	Abnormal	Emergency LSCS	H/o Previous	Nil	No	No	No	1.0	Yes	No	No	No	No	Yes	No
9	Group A	SHAINAZ	Multigravida	40	PE with Severe Features	Uneventful	Normal	92	170	110	130	50.0	26.0	64.0	52.0	132.10	1.06	0.83	1.28	0.07	0.12	Normal	Emergency LSCS	H/o Previous	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
10	Group A	SALMA ASIF DANGADIWALI	Primigravida	31	Imminent Eclampsia	Uneventful	Normal	86	130	100	110	65.0	25.0	74.0	53.0	132.10	0.92	1.13	0.81	0.03	0.03	Abnormal	Emergency LSCS	Abnormal	Pulmonary edema	No	No	Yes	1.3	Yes	Yes	No	Yes	No	Yes	No
11	Group A	SHAYEEN MULLA	Multigravida	38	Gestational HTN	Uneventful	Normal	90	120	90	100	70.0	30.0	102.0	72.0	112.60	0.13	0.17	0.76	0.11	0.17	Normal	Emergency LSCS	H/o Previous	Pulmonary edema	No	No	No	2.0	Yes	No	No	No	No	No	No
12	Group A	LAXMI SIDDANAGOUDA	Primigravida	35	PE with Severe Features	Pedal Oedema	Pedal Oedema Grade II	80	140	90	107	65.0	25.0	74.0	46.0	142.20	0.13	0.45	0.29	0.06	0.10	Normal	Emergency LSCS	PE with Sev	PPH	No	Yes	Yes	2.6	Yes	No	No	No	No	No	No
13	Group A	SAKKUBAI NAIK	Primigravida	32	PE with Severe Features	H/o High BP	Pedal Oedema Grade I	80	150	100	117	57.0	37.0	87.0	50.0	154.20	0.16	0.30	0.10	0.00	0.12	Normal	Elective LSCS	PE with Sev	Nil	No	No	No	1.6	No	Yes	No	Yes	No	Yes	No
14	Group A	SIDDAMMMA PARASHURAM	Primigravida	37	PE without Severe Features	Uneventful	Normal	90	160	90	113	60.0	13.0	63.0	44.0	103.03	0.53	0.70	1.41	0.03	0.13	Normal	Emergency LSCS	Prolonged	LRTI	No	No	No	2.0	Yes	No	Yes	No	No	Yes	No
15	Group A	NITHYA MUDHOL	Primigravida	40	Imminent Eclampsia	Uneventful	Pedal Oedema Grade II	96	160	90	113	74.0	27.0	45.0	66.0	132.00	0.30	1.10	0.03	0.05	0.12	Abnormal	Emergency LSCS	Breech Pre	Nil	No	No	No	3.1	Yes	No	No	No	No	Yes	No
16	Group A	JAYASHREE LAMANI	Primigravida	40	PE with Severe Features	H/o Anemia	Normal	90	140	90	107	64.0	32.0	83.0	57.0	120.02	1.10	0.66	1.67	0.16	0.11	Normal	Emergency LSCS	Cephalopelvic	LRTI	No	No	No	2.0	Yes	No	No	No	No	No	No
17	Group A	SAVITA PUNITE	Primigravida	33	PE with Severe Features	H/o High BP	Normal	124	130	90	103	67.0	34.0	102.0	60.0	164.45	1.40	0.70	2.00	0.16	0.21	Abnormal	Full Term Vaginal	Nil	LRTI	No	Yes	Yes	3.0	Yes	No	No	No	No	No	No
18	Group A	MAHANANDA	Primigravida	33	PE with Severe Features	Uneventful	Normal	100	140	90	107	66.0	16.0	47.0	31.0	106.92	1.10	0.67	1.64	0.12	0.14	Normal	Full Term Vaginal	Nil	Abruptio Placenta	No	Yes	Yes	2.1	Yes	No	No	No	No	No	No
19	Group A	KAVERI	Primigravida	34	PE with Severe Features	Uneventful	Normal	90	160	110	127	61.0	13.0	61.0	30.0	146.00	1.50	0.71	2.11	0.00	0.11	Abnormal	Emergency LSCS	PE with Sev	Nil	No	No	No	2.2	Yes	No	No	No	No	No	No
20	Group A	SUDHA AKASH	Primigravida	33	PE without Severe Features	Uneventful	Normal	90	140	90	107	53.0	26.0	50.0	57.0	106.20	1.30	0.70	1.06	0.06	0.13	Normal	Emergency LSCS	PE with Sev	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
21	Group A	SHIVALEELA	Multigravida	33	PE with Severe Features	Uneventful	Normal	94	170	100	123	65.0	32.0	56.0	43.0	134.20	1.06	0.14	7.57	0.07	0.12	Abnormal	Preterm Vaginal	Nil	Nil	Yes	No	No	1.5	No	No	No	Yes	Yes	No	Yes
22	Group A	PRIYANKA	Primigravida	35	PE with Severe Features	Uneventful	Normal	90	130	100	110	60.0	23.0	74.0	53.0	120.40	1.07	0.13	0.29	0.03	0.03	Abnormal	Emergency LSCS	PE with Sev	LRTI	No	No	No	2.2	Yes	No	No	No	No	No	No
23	Group A	SIDDAWWA	Multigravida	33	PE without Severe Features	Uneventful	Normal	102	140	90	107	60.0	25.0	63.0	64.0	142.50	1.00	0.17	6.95	0.11	0.00	Abnormal	Emergency LSCS	H/o Previous	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
24	Group A	SHADANA	Multigravida	40	Gestational HTN	Uneventful	Normal	90	140	90	107	62.0	30.0	50.0	43.0	123.20	1.20	0.66	1.02	0.00	0.14	Normal	Emergency LSCS	H/o Previous	Nil	No	No	No	2.2	Yes	Yes	No	No	No	Yes	No
25	Group A	MAHADEVI	Primigravida	40	Imminent Eclampsia	H/o High BP	Pedal Oedema Grade I	96	170	110	130	50.0	13.0	72.0	65.0	112.40	0.93	0.30	1.10	0.05	0.13	Abnormal	Emergency LSCS	Imminent	Pulmonary edema	No	No	Yes	2.6	Yes	No	No	No	No	No	No
26	Group A	LAXMI BADIGAR	Multigravida	33	PE without Severe Features	Uneventful	Normal	80	140	100	113	57.0	27.0	80.0	63.0	142.60	1.10	1.10	1.00	0.07	0.11	Normal	Emergency LSCS	H/o Previous	Nil	No	No	No	2.1	Yes	No	No	No	No	No	No
27	Group A	SUMA AMOG SIDDA	Multigravida	37	PE with Severe Features	Uneventful	Normal	82	160	90	113	64.0	24.0	74.0	48.0	132.60	0.30	0.16	6.13	0.11	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
28	Group A	KARISHMA NANDNAPUR	Primigravida	33	Gestational HTN	Uneventful	Pedal Oedema Grade I	96	140	90	107	61.0	31.0	50.0	51.0	140.70	0.13	0.45	0.29	0.07	0.10	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	2.0	Yes	Yes	No	No	No	No	No
29	Group A	VIDYASHREE BIRADAR	Multigravida	33	Gestational HTN	Uneventful	Normal	92	140	90	107	62.0	26.0	87.0	53.0	120.30	0.16	0.66	0.24	0.06	0.03	Abnormal	Preterm Vaginal	Nil	Lactation Failure	No	No	No	2.1	Yes	No	No	No	No	Yes	No
30	Group A	KAVERI ANIL	Primigravida	35	PE with Severe Features	Uneventful	Normal	86	130	100	110	55.0	30.0	64.0	50.0	112.60	0.07	0.10	4.03	0.03	0.03	Abnormal	Preterm Vaginal	Nil	Nil	No	No	No	1.0	Yes	Yes	No	Yes	No	Yes	No
31	Group A	SUNITHA VIKAS	Multigravida	34	Imminent Eclampsia	Uneventful	Normal	90	160	100	120	55.0	21.0	76.0	47.0	126.60	0.30	0.61	1.61	0.12	0.12	Normal	Emergency LSCS	Failed Dial	Nil	No	No	No	2.2	Yes	No	Yes	No	No	Yes	No
32	Group A	SUSHMITA KUSHAL PATIL	Multigravida	37	Imminent Eclampsia	Uneventful	Abdominal Wall Oedema	92	160	110	127	62.0	30.0	80.0	50.0	215.10	0.07	0.00	0.00	0.05	0.00	Abnormal	Emergency LSCS	Eclampsia	Pulmonary edema	No	No	No	2.6	Yes	Yes	No	No	No	Yes	No
33	Group A	NIKITA	Primigravida	37	PE with Severe Features	H/o Anemia	Normal	90	120	90	100	70.0	30.0	102.0	72.0	112.60	0.13	0.17	0.76	0.11	0.17	Normal	Emergency LSCS	Failed Dial	Nil	No	No	No	2.4	Yes	No	No	No	No	Yes	No
34	Group A	GEETA DALLAPPA KUDRI	Multigravida	40	PE with Severe Features	H/o High BP	Normal	80	140	90	107	65.0	25.0	74.0	46.0	142.20	0.13	0.45	0.29	0.06	0.10	Normal	Emergency LSCS	Failed Induction	LRTI	No	Yes	Yes	2.2	Yes	No	No	No	No	No	No

35	Group A	UJAMA ILYAS SHEIKH	Primigravida	33	PE with Severe Features	Uneventful	Normal	■■	150	100	117	57.0	57.0	87.0	50.0	154.20	0.16	###	###	0.00	0.12	Abnormal	Emergency LSCS	PE with...	Nil	No	No	No	1.7	Yes	No	No	No	No	No	No
36	Group A	DANAMMA DIDANAND SHIRAGUPPI	Primigravida	37	PE with Severe Features	Uneventful	Normal	90	160	90	113	60.0	13.0	63.0	44.0	103.03	0.33	###	###	0.03	0.13	Normal	Emergency LSCS	PE with...	Weaned ...	No	No	No	2.6	Yes	No	No	No	No	No	No
37	Group A	GEETA	Multigravida	33	Gestational HTN	Uneventful	Normal	36	160	90	113	71.0	27.0	45.0	66.0	192.00	0.30	###	###	0.05	0.12	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	2.3	Yes	No	No	No	No	No	No
38	Group A	SHOBHA RATHOD	Primigravida	40	PE with Severe Features	Uneventful	Normal	90	140	90	107	64.0	32.0	83.0	57.0	120.02	1.10	###	###	0.16	0.11	Normal	Emergency LSCS	Non Pro...	LRT I	Yes	No	No	2.2	Yes	No	No	No	No	No	No
39	Group A	BOURRAMMA ANIL BADDUR	Multigravida	37	Gestational HTN	Uneventful	Normal	124	130	90	103	67.0	34.0	####	60.0	164.45	1.40	###	###	0.16	0.21	Abnormal	Emergency LSCS	H/o Pre...	Nil	No	No	No	2.1	No	No	No	Yes	Yes	No	Yes
40	Group A	KAVERI JNANESHWAR	Primigravida	40	Gestational HTN	Uneventful	Normal	100	140	90	107	66.0	16.0	47.0	31.0	106.32	1.10	###	###	0.12	0.14	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.4	Yes	No	No	No	No	No	No
41	Group A	ANITA AWWANNA JAWADAGI	Primigravida	33	PE with Severe Features	Uneventful	Normal	90	160	110	127	61.0	19.0	61.0	50.0	146.00	1.50	###	###	0.00	0.11	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	2.3	Yes	No	No	No	No	No	No
42	Group A	CHAKKULI DATTA JHAKANE	Primigravida	42	PE with Severe Features	H/o High BP	Pedal Oedema Grade II	90	140	90	107	53.0	26.0	50.0	57.0	106.20	1.30	###	###	0.06	0.13	Normal	Emergency LSCS	PE with...	Nil	No	No	No	3.0	Yes	Yes	No	No	No	Yes	No
43	Group A	BANASHANKARI OGEPPA BATU	Multigravida	33	Chronic HTN with Superimposed...	Uneventful	Normal	34	150	100	117	65.0	32.0	56.0	43.0	134.20	1.06	###	###	0.07	0.12	Abnormal	Emergency LSCS	Bad Obs...	Nil	No	No	No	3.1	Yes	No	No	No	No	No	No
44	Group A	BHARATI SHIVANAND	Multigravida	37	PE with Severe Features	Uneventful	Normal	90	130	100	110	60.0	23.0	71.0	53.0	120.40	1.07	###	###	0.03	0.03	Abnormal	Emergency LSCS	H/o Pre...	Nil	No	No	No	2.7	Yes	No	No	No	No	No	No
45	Group A	ZOYA SAIYADAPRID	Multigravida	35	PE with Severe Features	Uneventful	Pedal Oedema Grade II	102	140	90	107	60.0	25.0	63.0	61.0	142.50	1.00	###	###	0.11	0.00	Abnormal	Emergency LSCS	Abd...	Nil	No	No	No	1.1	Yes	No	No	Yes	No	Yes	No
46	Group A	VIDYA MANSHE	Primigravida	37	PE with Severe Features	H/o High BP	Normal	90	140	90	107	62.0	30.0	50.0	43.0	123.20	1.20	###	###	0.00	0.14	Normal	Full Term Vaginal	Nil	Lactation	No	No	No	2.6	Yes	Yes	No	No	No	No	No
47	Group A	MEHBOOBI	Multigravida	31	PE with Severe Features	Uneventful	Normal	36	170	110	130	50.0	13.0	72.0	65.0	112.40	0.33	###	###	0.05	0.13	Abnormal	Preterm Vaginal	Nil	Nil	Yes	No	No	2.4	No	No	No	No	Yes	No	No
48	Group A	SUNITA SAMADHAN LAVATE	Multigravida	33	PE without Severe Features	Uneventful	Normal	■■	140	100	113	57.0	27.0	■■.0	63.0	142.60	1.10	###	###	0.07	0.11	Normal	Emergency LSCS	PE with...	Nil	No	No	No	2.3	Yes	Yes	No	Yes	No	Yes	No
49	Group A	SHILPA MALLAPA BHAGAVATI	Multigravida	35	PE with Severe Features	Uneventful	Abdominal Wall Oedema	82	160	90	113	61.0	24.0	71.0	40.0	132.60	0.30	###	###	0.11	0.11	Normal	Emergency LSCS	PE with...	Weaned ...	No	No	No	3.0	Yes	No	Yes	No	No	Yes	No
50	Group A	KASTURBAI DHARMASINH GURATHOD	Multigravida	34	PE with Severe Features	H/o High BP	Normal	36	140	90	107	61.0	31.0	50.0	51.0	140.70	0.13	###	###	0.07	0.10	Normal	Preterm Vaginal	Intr...	Tachypnoea	Yes	Yes	No	1.2	Yes	Yes	No	No	No	Yes	No
51	Group A	SANA PARVEEN KHAISAR	Multigravida	40	PE without Severe Features	Uneventful	Normal	32	140	90	107	62.0	26.0	87.0	53.0	120.30	0.16	###	###	0.06	0.03	Abnormal	Emergency LSCS	PE with...	Nil	No	No	Yes	2.0	Yes	No	No	No	No	No	No
52	Group A	DAKSHAYANI BASAVARAJ	Multigravida	30	PE without Severe Features	Uneventful	Normal	90	150	90	110	65.0	26.0	72.0	47.0	132.10	0.12	###	###	0.10	0.13	Normal	Emergency LSCS	Abd...	Nil	No	No	No	2.6	No	Yes	No	No	No	Yes	No
53	Group A	PRABHAVATI BHEEMRAY	Multigravida	30	Gestational HTN	Uneventful	Normal	■■	140	90	107	57.0	23.0	76.0	51.0	152.00	1.07	###	###	0.00	0.03	Abnormal	Emergency LSCS	H/o Pre...	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
54	Group A	POOJA HANAMANT JAMADAR	Primigravida	33	PE with Severe Features	H/o High BP	Pedal Oedema Grade I	82	140	90	130	50.0	26.0	61.0	52.0	132.10	1.06	###	###	0.07	0.12	Abnormal	Full Term Vaginal	Intr...	LRT I	Yes	No	No	2.1	No	No	No	No	Yes	No	No
55	Group A	SHAKUNTALA SANGAMESH	Multigravida	41	Gestational HTN	Uneventful	Normal	82	130	90	103	65.0	25.0	71.0	53.0	132.10	0.32	###	###	0.03	0.03	Abnormal	Emergency LSCS	Non rea...	Nil	No	No	No	2.4	No	Yes	No	No	No	Yes	No
56	Group A	SANGEETA MALLIKARJUN	Multigravida	37	Gestational HTN	Uneventful	Normal	34	140	90	107	70.0	30.0	####	72.0	142.60	0.13	###	###	0.11	0.17	Normal	Emergency LSCS	PE with...	Nil	No	No	No	2.5	No	Yes	No	No	No	No	No
57	Group A	SUNANDA BHIMAROY MARNUR	Primigravida	33	PE without Severe Features	H/o High BP	Pedal Oedema Grade I	90	160	110	127	65.0	25.0	71.0	46.0	142.20	0.13	###	###	0.06	0.10	Normal	Emergency LSCS	PE with...	Nil	No	No	No	2.6	No	Yes	No	No	No	Yes	No
58	Group A	SANTOSH KHILA BALATTI	Primigravida	30	Imminent Eclampsia	H/o High BP	Pedal Oedema Grade II	102	140	90	107	57.0	37.0	87.0	50.0	154.20	0.16	###	###	0.00	0.12	Abnormal	Emergency LSCS	Pre...	Nil	No	No	Yes	2.1	No	Yes	No	Yes	No	Yes	No
59	Group A	BIBIJAN HUSENABASHA	Primigravida	36	PE with Severe Features	H/o High BP	Normal	100	150	100	117	60.0	13.0	63.0	44.0	103.03	0.33	###	###	0.03	0.13	Normal	Full Term Vaginal	PE with...	Nil	No	No	No	2.6	No	Yes	Yes	No	No	Yes	No
60	Group A	CHANDRIKA SIDDAPPA HOLER	Multigravida	35	PE with Severe Features	H/o High BP	Pedal Oedema Grade I	90	130	100	110	71.0	27.0	45.0	66.0	192.00	0.30	###	###	0.05	0.12	Abnormal	Preterm Vaginal	PE with...	Abruption	Yes	Yes	Yes	2.0	No	No	No	No	Yes	No	Yes
61	Group A	BHAGYASHREE SANTOSH SALAKKI	Multigravida	40	PE without Severe Features	Uneventful	Normal	■■	130	90	103	62.0	30.0	50.0	43.0	123.20	1.20	###	###	0.00	0.14	Normal	Emergency LSCS	Cephal...	Nil	No	No	No	3.0	Yes	No	No	No	No	No	No
62	Group A	NANANAND A VITTAL BADIGAR	Multigravida	33	PE with Severe Features	H/o High BP	Pedal Oedema Grade I	90	140	90	107	50.0	13.0	72.0	65.0	112.40	0.33	###	###	0.05	0.13	Normal	Emergency LSCS	PE with...	Nil	No	No	No	3.2	Yes	No	No	No	No	No	No
63	Group A	BHAGYASHREE SANGANASA	Multigravida	37	PE with Severe Features	Pedal Oedema	Pedal Oedema Grade II	36	150	90	110	57.0	27.0	■■.0	63.0	142.60	1.10	###	###	0.07	0.11	Normal	Emergency LSCS	H/o Pre...	Nil	No	No	No	2.3	Yes	No	No	No	No	No	No
64	Group A	ASHWINI IRASANGAPPA	Multigravida	33	Gestational HTN	H/o PV leak	Normal	76	120	90	100	61.0	24.0	71.0	40.0	132.60	0.30	###	###	0.11	0.11	Abnormal	Emergency LSCS	Cephal...	Nil	No	No	No	3.0	Yes	No	No	No	No	No	No
65	Group A	PREMA P KAMBLE	Primigravida	30	Gestational HTN	H/o High BP	Pedal Oedema Grade I	74	140	90	107	61.0	31.0	50.0	51.0	140.70	0.13	###	###	0.07	0.10	Normal	Emergency LSCS	Secondary...	Nil	No	No	No	3.0	Yes	Yes	Yes	No	No	Yes	No
66	Group A	TAJANBI BANDENAWAZ ALMASHYAL	Multigravida	33	PE with Severe Features	H/o High BP	Pedal Oedema Grade I	102	130	90	103	62.0	26.0	87.0	53.0	120.30	0.16	###	###	0.06	0.03	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	1.3	Yes	Yes	Yes	Yes	No	Yes	No
67	Group A	RESHMA NANDYAL	Multigravida	30	PE with Severe Features	Pedal Oedema	Pedal Oedema Grade II	86	140	100	113	65.0	26.0	72.0	47.0	132.10	0.12	###	###	0.10	0.13	Normal	Full Term Vaginal	Nil	Nil	No	No	No	3.1	Yes	No	No	No	No	No	No
68	Group A	SHRUTHI SHEKAPPA KARAJAL	Primigravida	36	Gestational HTN	H/o High BP	Pedal Oedema Grade III	36	160	90	113	57.0	23.0	76.0	51.0	152.00	1.07	###	###	0.00	0.03	Abnormal	Full Term Vaginal	PE with...	Nil	No	No	No	2.5	Yes	No	No	No	No	Yes	No
69	Group A	SAVITRI SHRISHAIL AMBALI	Multigravida	30	PE with Severe Features	H/o PV leak	Normal	90	140	90	107	50.0	26.0	61.0	52.0	132.10	1.06	###	###	0.07	0.12	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
70	Group A	BHAVANI MAHANINGA HETI	Primigravida	41	Gestational HTN	H/o High BP	Pallor	34	160	110	127	65.0	25.0	71.0	53.0	132.10	0.32	###	###	0.03	0.03	Abnormal	Full Term Vaginal	Nil	Nil	No	Yes	No	3.2	Yes	No	No	No	No	No	No
	Group A	GANGABAI	Multigravida		Gestational HTN	Uneventful																Normal	Emergency LSCS	H/o...						Yes						

72	Group B	SUMANGALA RAJU KUMBAR	Primigravida	30	#NULL!	Unremarkable	Normal	86	100	50	53	60.0	35.0	75.0	55.0	#####	0.47	0.47	1.94	0.09	0.42	Normal	Emergency LSCS	Fetal Dis	Nil	No	No	No	2.9	Yes	Yes	No	No	No	Yes	No
73	Group B	MANANANDA CHANDRAS	Primigravida	33	#NULL!	H/o PV Leak	Normal	80	130	70	90	66.0	16.0	35.0	31.0	#####	0.14	0.14	1.10	0.14	0.14	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.1	Yes	No	No	No	No	Yes	No
74	Group B	PREETI RAVASAB PUJARI	Primigravida	36	#NULL!	Unremarkable	Normal	112	130	80	97	64.0	27.0	63.0	40.0	#####	0.00	0.11	1.90	0.04	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.0	No	No	No	No	No	No	No
75	Group B	ASHWINI MAHESH PAWAR	Primigravida	33	#NULL!	H/o PV Leak	Normal	82	110	70	83	53.0	42.0	83.0	53.0	#####	0.47	0.47	1.94	0.47	0.47	Normal	Emergency LSCS	He ar Pr	Nil	No	No	No	2.7	Yes	No	No	No	No	No	No
76	Group B	VANISHREE CHANDRAKANTH	Primigravida	34	#NULL!	H/o Anemia	Normal	90	120	80	93	60.0	35.0	75.0	52.0	#####	0.75	0.45	1.71	0.09	0.45	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.0	No	No	No	No	No	No	No
77	Group B	JYOTI P SAGARE	Primigravida	40	#NULL!	Unremarkable	Normal	92	130	80	97	62.0	23.0	55.0	51.0	#####	0.40	0.45	1.40	0.14	0.42	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
78	Group B	SUJATA	Multigravida	30	#NULL!	Unremarkable	Normal	80	126	80	95	62.0	22.0	45.0	35.0	#####	0.10	0.10	2.30	0.10	0.10	Normal	Elective LSCS	H/o Pr	Nil	No	No	No	3.0	Yes	No	Yes	No	No	No	No
79	Group B	PREETHI HATTARAKI	Primigravida	30	#NULL!	Unremarkable	Normal	92	130	80	97	70.0	20.0	26.0	43.0	#####	0.30	0.36	1.10	0.10	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.4	No	Yes	No	No	No	Yes	No
80	Group B	ASHWINI APPARAYA ADAKE	Multigravida	33	#NULL!	Unremarkable	Normal	80	110	80	90	72.0	27.0	83.0	60.0	#####	0.40	0.34	1.41	0.00	0.10	Normal	Emergency LSCS	H/o Pr	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
81	Group B	KAVITHA CHANNAMA LAPPA	Multigravida	31	#NULL!	Unremarkable	Normal	96	110	70	85	67.0	25.0	63.0	51.0	#####	0.76	0.76	1.26	0.47	0.20	Normal	Preterm Vaginal	Nil	Nil	No	No	No	1.0	Yes	Yes	No	No	No	Yes	No
82	Group B	REKHA DANDAPPA	Primigravida	33	#NULL!	Unremarkable	Normal	86	112	70	84	67.0	27.0	70.0	55.0	#####	1.30	0.66	2.11	0.15	0.15	Abnormal	Emergency LSCS	Hr eae iem	Nil	No	No	No	2.0	Yes	Yes	Yes	No	No	Yes	No
83	Group B	POOJA RAJESH HOLE	Multigravida	37	#NULL!	Unremarkable	Normal	80	110	70	85	66.0	30.0	76.0	50.0	#####	1.10	0.00	1.45	0.10	0.15	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	3.1	Yes	No	No	No	No	No	No
84	Group B	PAVITRA ANILHALLI	Primigravida	34	#NULL!	Unremarkable	Normal	80	110	80	90	60.0	27.0	80.0	55.0	#####	1.05	0.67	1.12	0.47	0.42	Normal	Preterm Vaginal	Nil	Nil	No	No	No	2.2	Yes	No	No	No	No	Yes	No
85	Group B	BHAGYASHREE ALMATTI	Multigravida	30	#NULL!	Unremarkable	Normal	86	130	80	97	73.0	20.0	36.0	73.0	#####	1.00	0.76	1.49	0.10	0.10	Normal	Emergency LSCS	Cephalop	Nil	No	No	No	3.3	Yes	No	No	No	No	No	No
86	Group B	SONALI PUJARI	Multigravida	36	#NULL!	H/o Appr ecial	Normal	94	110	70	85	64.0	31.0	75.0	55.0	#####	1.00	0.56	1.05	0.19	0.04	Abnormal	Vaginal (birth after	Int rau ter	Nil	Yes	No	No	2.0	No	No	No	No	Yes	No	Yes
87	Group B	SUPRIYA MUJAWANI	Primigravida	35	#NULL!	Unremarkable	Normal	90	130	80	97	61.0	34.0	74.0	53.0	#####	0.34	0.45	1.14	0.09	0.11	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	2.1	Yes	No	No	No	No	No	No
88	Group B	SHARANAMMA HIREMATH	Multigravida	37	#NULL!	Unremarkable	Normal	90	110	70	85	70.0	26.0	65.0	47.0	#####	0.67	0.67	1.56	0.11	0.15	Normal	Full Term Vaginal	Nil	Nil	No	No	No	3.2	Yes	No	No	No	No	No	No
89	Group B	SALANI BAJANTRI	Primigravida	35	#NULL!	H/o Anemia	Normal	92	120	70	87	72.0	22.0	80.0	56.0	#####	0.70	0.66	1.30	0.60	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.2	Yes	No	No	No	No	No	No
90	Group B	SWARNANJALI	Multigravida	36	#NULL!	Unremarkable	Normal	86	100	60	73	67.0	21.0	71.0	55.0	#####	1.10	0.36	1.10	0.09	0.09	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
91	Group B	BISMILLA	Primigravida	33	#NULL!	Unremarkable	Normal	80	110	80	90	70.0	20.0	74.0	54.0	#####	0.40	0.14	1.71	0.16	0.14	Normal	Emergency LSCS	Cephalop	Nil	No	No	No	3.0	Yes	No	No	No	No	Yes	No
92	Group B	SHARANE	Primigravida	40	#NULL!	Unremarkable	Normal	84	100	70	80	72.0	20.0	57.0	53.0	#####	0.30	0.11	1.34	0.10	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.7	Yes	No	No	No	No	No	No
93	Group B	SHILPA SANTHOSH DASHYAL	Multigravida	37	#NULL!	Unremarkable	Normal	80	110	70	85	64.0	30.0	61.0	61.0	#####	0.00	0.17	1.14	0.17	0.17	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.3	Yes	No	No	No	No	No	No
94	Group B	VIJAYALAXMI	Multigravida	33	#NULL!	Unremarkable	Normal	90	110	70	85	66.0	27.0	73.0	67.0	#####	0.67	0.11	1.26	0.00	0.15	Normal	Emergency LSCS	H/o Pr	Nil	No	No	No	3.2	Yes	No	No	No	No	No	No
95	Group B	SHWETHA	Multigravida	30	#NULL!	Unremarkable	Normal	92	120	80	93	60.0	26.0	85.0	43.0	#####	0.40	0.14	1.41	0.10	0.42	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
96	Group B	GEETA	Multigravida	35	#NULL!	Unremarkable	Normal	90	130	80	97	72.0	22.0	73.0	54.0	#####	0.30	0.45	2.10	0.47	0.11	Abnormal	Emergency LSCS	H/o Pr	Nil	No	No	No	3.4	Yes	No	No	No	No	No	No
97	Group B	BHARATHI	Primigravida	37	#NULL!	Unremarkable	Normal	80	110	80	90	70.0	23.0	40.0	57.0	#####	0.75	0.36	1.45	0.19	0.10	Normal	Emergency LSCS	Dr era k	Nil	No	No	No	3.2	Yes	Yes	No	No	No	Yes	No
98	Group B	RAJASHRI SHASHIKANT	Multigravida	30	#NULL!	Unremarkable	Normal	86	100	70	80	67.0	26.0	66.0	52.0	#####	0.40	0.56	1.35	0.09	0.11	Normal	Emergency LSCS	H/o Pr	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
99	Group B	POOJA	Multigravida	40	#NULL!	Unremarkable	Normal	90	120	80	93	60.0	22.0	71.0	53.0	#####	0.67	0.45	1.70	0.10	0.20	Normal	Elective LSCS	H/o Pr	Nil	No	No	No	2.3	Yes	No	No	No	No	No	No
100	Group B	SAVITA	Multigravida	41	#NULL!	Unremarkable	Normal	92	114	70	85	50.0	20.0	73.0	63.0	#####	0.40	0.34	1.26	0.00	0.15	Normal	Emergency LSCS	Nil	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
101	Group B	IRFANA	Multigravida	35	#NULL!	Unremarkable	Normal	90	120	82	97	61.0	23.0	64.0	43.0	#####	0.11	0.15	1.53	0.12	0.12	Normal	Emergency LSCS	Pla era la	Nil	No	No	No	3.2	Yes	No	No	No	No	No	No
102	Group B	SOWMYA GANGAVATHI	Multigravida	36	#NULL!	Unremarkable	Normal	80	120	80	93	66.0	30.0	65.0	66.0	#####	0.56	0.24	1.70	0.14	0.11	Normal	Emergency LSCS	Ha Irr mal	Nil	No	No	No	2.4	Yes	No	No	No	No	No	No
103	Group B	RASHMI	Primigravida	40	#NULL!	Unremarkable	Normal	92	120	70	87	60.0	23.0	70.0	54.0	#####	0.51	0.26	1.45	0.17	0.10	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.0	Yes	Yes	No	No	No	Yes	No
104	Group B	DEEPA	Multigravida	33	#NULL!	Unremarkable	Normal	94	110	70	85	62.0	26.0	56.0	57.0	#####	0.45	0.30	1.26	0.15	0.15	Normal	Full Term Vaginal	Nil	Nil	No	No	No	3.4	Yes	No	No	No	No	No	No
105	Group B	RADHA	Multigravida	33	#NULL!	Unremarkable	Normal	84	120	70	87	70.0	23.0	63.0	53.0	#####	0.60	0.45	1.10	0.09	0.09	Abnormal	Emergency LSCS	Sre err Oli	Nil	No	No	No	2.0	Yes	No	No	No	No	Yes	No
106	Group B	IRFANA	Multigravida	36	#NULL!	Unremarkable	Normal	96	130	70	90	60.0	32.0	73.0	56.0	#####	1.10	0.34	1.30	0.32	0.07	Abnormal	Emergency LSCS	He ar era	Nil	No	No	No	2.2	Yes	Yes	No	No	No	Yes	No
107	Group B	SUKDEVI	Multigravida	41	#NULL!	Unremarkable	Normal	94	110	80	90	60.0	27.0	74.0	61.0	#####	0.30	0.29	1.10	0.12	0.11	Normal	Emergency LSCS	H/o Pr	Nil	No	No	No	2.1	Yes	No	No	No	No	No	No
108	Group B	ROOPA BARADOL	Multigravida	33	#NULL!	Unremarkable	Normal	90	110	70	85	70.0	26.0	65.0	47.0	#####	0.67	0.67	1.56	0.11	0.15	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.2	Yes	No	No	No	No	No	No
						Unre																	Emerg	Ha												

110	Group B	ANNAPURNA CHIDANAND	Multigravida	34	#NULL!	Uneventful	Normal	86	100	60	79	67.0	24.0	74.0	55.0	#####	1.40	0.56	1.40	0.09	0.09	Abnormal	Preterm Vaginal	Nil	Nil	No	No	No	1.9	Yes	No	No	No	No	No	No
111	Group B	ROOPA BANNAGLI	Primigravida	30	#NULL!	Uneventful	Normal	80	110	80	90	70.0	20.0	74.0	54.0	#####	0.40	0.14	1.71	0.16	0.14	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.7	Yes	No	No	No	No	No	No
112	Group B	PRIYANKA HIREMATH	Multigravida	33	#NULL!	Uneventful	Normal	84	100	70	80	72.0	20.0	57.0	53.0	#####	0.30	0.11	1.34	0.10	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.4	No	No	No	No	No	No	Yes
113	Group B	AMBIKA BIRADAR	Multigravida	40	#NULL!	Uneventful	Normal	80	110	70	85	64.0	30.0	61.0	61.0	#####	0.00	0.17	1.14	0.17	0.17	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.5	Yes	No	No	No	No	No	No
114	Group B	PADMAVATI BOGAR	Primigravida	30	#NULL!	Uneventful	Normal	90	110	70	85	66.0	27.0	79.0	67.0	#####	0.67	0.11	1.26	0.00	0.19	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
115	Group B	MALASHREE DASHAVAN	Multigravida	40	#NULL!	Uneventful	Normal	92	120	80	93	60.0	26.0	85.0	49.0	#####	0.40	0.14	1.41	0.10	0.12	Normal	Full Term Vaginal	Nil	Nil	No	No	No	3.0	Yes	No	No	No	No	No	No
116	Group B	DEEPA	Multigravida	40	#NULL!	Uneventful	Normal	90	130	80	97	72.0	22.0	79.0	54.0	#####	0.30	0.19	2.10	0.17	0.11	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	3.2	Yes	No	No	No	No	No	No
117	Group B	JIJABAI NAIK	Primigravida	37	#NULL!	Uneventful	Normal	80	110	80	90	70.0	29.0	40.0	57.0	#####	0.75	0.56	1.45	0.19	0.10	Normal	Emergency LSCS	Non Pr	Nil	No	No	No	3.9	Yes	No	No	No	No	No	No
118	Group B	BEGUM	Primigravida	30	#NULL!	Uneventful	Normal	86	100	70	80	67.0	26.0	66.0	52.0	#####	0.40	0.56	1.95	0.09	0.11	Normal	Emergency LSCS	Cephalop	Nil	No	No	No	3.1	Yes	Yes	No	No	No	Yes	No
119	Group B	ASHWINI	Multigravida	33	#NULL!	Uneventful	Normal	90	120	80	93	60.0	22.0	74.0	59.0	#####	0.67	0.45	1.70	0.10	0.20	Normal	Emergency LSCS	H/o Pr	Nil	No	No	No	3.0	Yes	No	No	No	No	No	No
120	Group B	NEELAMMA DALWAI	Multigravida	33	#NULL!	Uneventful	Normal	92	114	70	85	50.0	20.0	79.0	69.0	#####	0.40	0.34	1.26	0.00	0.19	Normal	Emergency LSCS	Sec arr OI	Nil	No	No	No	3.2	Yes	No	No	No	No	No	No
121	Group B	LAXMI CHATRI	Primigravida	40	#NULL!	Uneventful	Normal	90	120	82	97	61.0	29.0	64.0	49.0	#####	0.11	0.19	1.59	0.12	0.12	Normal	Emergency LSCS	Sec arr OI	Nil	No	No	No	3.4	Yes	No	No	No	No	No	No
122	Group B	ANITA	Multigravida	37	#NULL!	Uneventful	Normal	80	120	80	93	66.0	30.0	65.0	66.0	#####	0.56	0.24	1.70	0.14	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
123	Group B	NEETA	Multigravida	37	#NULL!	Uneventful	Normal	92	120	70	87	60.0	29.0	70.0	54.0	#####	0.51	0.26	1.45	0.17	0.10	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.6	Yes	No	No	No	No	No	No
124	Group B	SHARADA	Primigravida	40	#NULL!	Uneventful	Normal	94	110	70	89	62.0	26.0	56.0	57.0	#####	0.49	0.30	1.26	0.15	0.19	Normal	Emergency LSCS	Sec arr OI	Nil	No	No	No	3.0	Yes	Yes	No	No	No	Yes	No
125	Group B	SUNANDA SONNAD	Multigravida	37	#NULL!	Uneventful	Normal	84	120	70	87	70.0	29.0	69.0	59.0	#####	0.60	0.19	1.10	0.09	0.09	Abnormal	Emergency LSCS	H/o Pr	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
126	Group B	KAVITA BIRADAR	Multigravida	35	#NULL!	Uneventful	Normal	96	130	70	90	60.0	32.0	79.0	56.0	#####	1.10	0.34	1.30	0.32	0.07	Abnormal	Preterm Vaginal	Nil	Nil	No	No	No	2.1	Yes	No	No	No	No	Yes	No
127	Group B	VIJAYLAXMI IWALIGAR	Primigravida	33	#NULL!	Uneventful	Normal	94	110	80	90	60.0	27.0	74.0	61.0	#####	0.30	0.29	1.10	0.12	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	3.0	Yes	Yes	No	No	No	Yes	No
128	Group B	PRATIBA WALIKAR	Multigravida	40	#NULL!	Uneventful	Normal	90	120	80	93	60.0	22.0	74.0	59.0	#####	0.67	0.45	1.70	0.10	0.20	Normal	Emergency LSCS	H/o Pr	Nil	No	No	No	3.2	Yes	No	No	No	No	No	No
129	Group B	PADMAVATI ANAND BELLUBBI	Primigravida	30	#NULL!	Uneventful	Normal	96	130	80	97	60.0	26.0	85.0	49.0	#####	0.40	0.14	1.41	0.10	0.12	Normal	Emergency LSCS	Non Pr	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
130	Group B	LAXMI MANJUNATH	Multigravida	33	#NULL!	Uneventful	Normal	84	110	70	97	72.0	22.0	79.0	54.0	#####	0.30	0.19	2.10	0.17	0.11	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	3.1	Yes	No	No	No	No	No	No
131	Group B	ARATI KASHINATH MANTYAL	Primigravida	40	#NULL!	Uneventful	Normal	80	100	70	80	70.0	29.0	40.0	57.0	#####	0.75	0.56	1.45	0.19	0.10	Normal	Emergency LSCS	Maternal	Nil	No	No	No	2.4	Yes	No	No	No	No	No	No
132	Group B	BAGAMMA MADDAPPA BIRADAR	Multigravida	30	#NULL!	H/o High BP	Normal	80	130	80	97	67.0	26.0	66.0	52.0	#####	0.40	0.56	1.95	0.09	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.9	Yes	No	No	No	No	No	No
133	Group B	POOJA MAHESH PODDAR	Multigravida	33	#NULL!	Uneventful	Normal	90	130	80	97	60.0	22.0	74.0	59.0	#####	0.67	0.45	1.70	0.10	0.20	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.0	Yes	No	No	No	No	No	No
134	Group B	ARCHANA SHRISHAIL CHAVAN	Multigravida	33	#NULL!	Uneventful	Normal	90	120	80	93	50.0	20.0	79.0	69.0	#####	0.40	0.34	1.26	0.00	0.19	Normal	Full Term Vaginal	Nil	Nil	No	No	No	3.2	Yes	No	No	No	No	No	No
135	Group B	SANANA MUDDANA GOUDA	Primigravida	33	#NULL!	Uneventful	Normal	96	110	70	89	61.0	29.0	64.0	49.0	#####	0.11	0.19	1.59	0.12	0.12	Normal	Emergency LSCS	Cephalop	Wound Gap	No	No	No	4.0	Yes	No	No	No	No	No	No
136	Group B	BASAMMA LAXMAN BADIGER	Multigravida	30	#NULL!	Uneventful	Normal	104	130	80	97	66.0	30.0	65.0	66.0	#####	0.56	0.24	1.70	0.14	0.11	Normal	Emergency LSCS	H/o Pr	Nil	No	No	No	2.4	Yes	No	No	No	No	No	No
137	Group B	NIRMALA PARSURAM MARADIMA	Multigravida	34	#NULL!	Uneventful	Normal	100	120	70	87	60.0	29.0	70.0	54.0	#####	0.51	0.26	1.45	0.17	0.10	Normal	Emergency LSCS	H/o Pr	Nil	No	No	No	2.5	Yes	No	No	No	No	No	No
138	Group B	RADHADEVI BABALU	Primigravida	41	#NULL!	Uneventful	Normal	86	130	80	97	62.0	26.0	56.0	57.0	#####	0.49	0.30	1.26	0.15	0.19	Normal	Emergency LSCS	Fetal Dis	Nil	No	No	No	2.0	Yes	Yes	Yes	No	No	Yes	No
139	Group B	LAKSHMI SHARANBASU NAGAVI	Multigravida	37	#NULL!	Grow th ease	Normal	90	120	80	93	70.0	29.0	69.0	59.0	#####	0.60	0.19	1.10	0.09	0.09	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.6	Yes	Yes	No	No	No	No	No
140	Group B	DANAMMA ILLALAMATH	Multigravida	33	#NULL!	Uneventful	Normal	92	130	80	97	60.0	32.0	79.0	56.0	#####	1.10	0.34	1.30	0.32	0.07	Abnormal	Full Term Vaginal	Nil	Nil	No	No	No	2.9	Yes	Yes	No	No	No	No	No
141	Group B	RAJESHWARI M KURANI	Multigravida	30	#NULL!	Uneventful	Normal	86	110	70	89	60.0	27.0	74.0	61.0	#####	0.30	0.29	1.10	0.12	0.11	Normal	Full Term Vaginal	Nil	Nil	No	No	No	2.2	Yes	No	No	No	No	No	No
142	Group B	GOURAMMA	Multigravida	30	#NULL!	H/o PV	Normal	86	100	70	80	60.0	22.0	74.0	59.0	#####	0.67	0.45	1.70	0.10	0.20	Normal	Emergency	H/o	Nil	No	No	No	2.9	Yes	No	No	No	No	No	No

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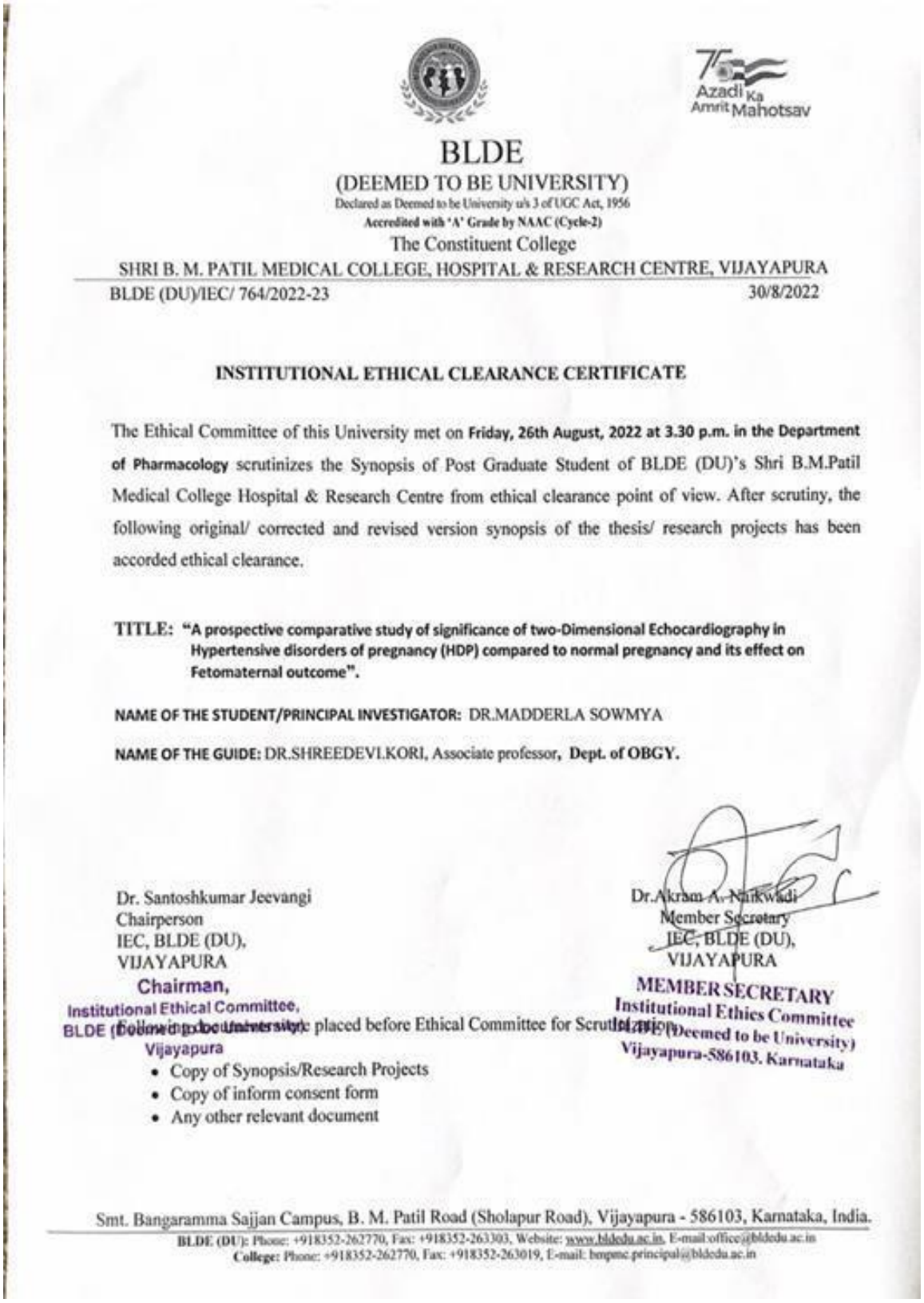

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INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Friday, 26th August, 2022 at 3.30 p.m. in the Department of Pharmacology** scrutinizes the Synopsis of Post Graduate Student of BLDE (DU)'s Shri B.M.Patil Medical College Hospital & Research Centre from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "A prospective comparative study of significance of two-Dimensional Echocardiography in Hypertensive disorders of pregnancy (HDP) compared to normal pregnancy and its effect on Fetomaternal outcome".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.MADDERLA SOWMYA

NAME OF THE GUIDE: DR.SHREEDEVI.KORI, Associate professor, **Dept. of OBGY.**

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VIJAYAPURA

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IEC, BLDE (DU),
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MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following to be placed before Ethical Committee for Scrutinization:

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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