EVALUATION OF LATERAL IMPLANTATION OF PLACENTA AND ITS ASSOCIATION WITH THE DEVELOPMENT OF PREECLAMPSIA AND ITS OUTCOME

By

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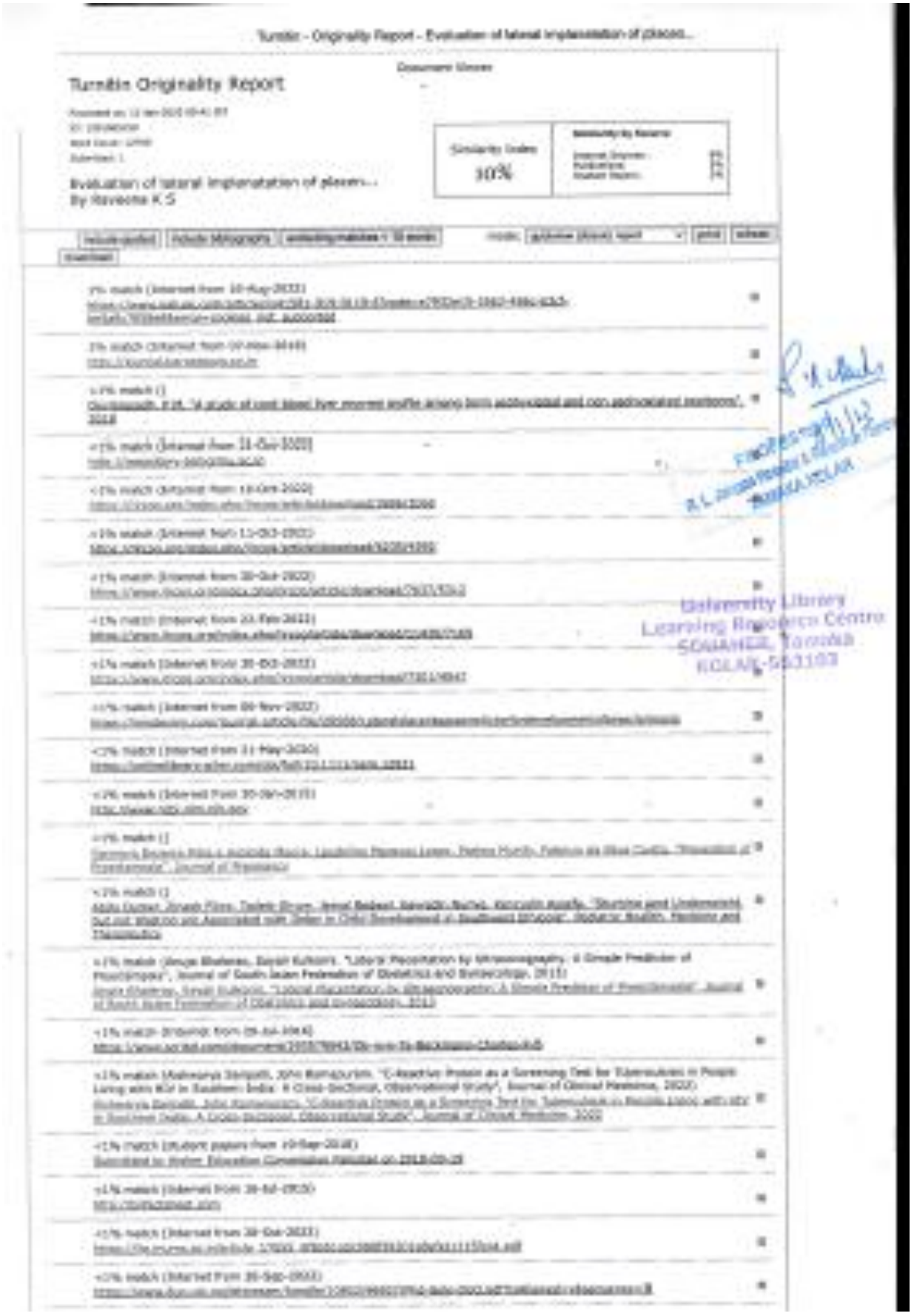

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DR.RAVEENA K S

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ABBREVIATIONS

Glossary	Abbreviations
HDP	Hypertensive Disorder of Pregnancy
PIH	Pregnancy-Induced Hypertension
ISSHP	International Society for the Study of Hypertension in Pregnancy
sENG	Soluble endoglin
IVF	In Vitro Fertilization
IUGR	Intra Uterine Growth Restriction
LMP	Last Menstruation Cycle
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
PPROM	Preterm premature rupture of membranes
DIC	Disseminated intravascular coagulation
Sflt-1	Soluble FMS like tyrosine kinase
SNP	Single nucleotide polymorphism
SGA	Small for gestational age
LBW	Low birth weight
PE	preeclampsia
USG	ultrasonography
HELLP	Hemolysis elevated liver enzymes low platelet count

ABSTRACT

BACKGROUND: The leading cause of maternal and neonatal morbidity and death is preeclampsia. A condition affecting trophoblastic tissue is preeclampsia. One of the early signs in patients who will later experience pregnancy-induced hypertension is a placental abnormality. Preeclampsia and eclampsia handle over 15% of all maternal fatalities globally. Aside from contributing to pregnancy consequences, as preeclampsia is a multiorgan disease, HDP elevates the chance of various postpartum issues. In order to prevent such a disorder, timely screening is necessary and examination of the substantiate segments of population are required to discover the disease at an initial phase. The most important component in preventing this is right prediction.

AIM : The aim of the study was to evaluate whether a placenta that appeared laterally on ultrasound was associated with the onset of preeclampsia.

MATERIALS AND METHODS: Between Jan to Dec 2021, 126 antenatal women who were carrying singletons and were between 18- and 24 weeks gestational age participated in this prospective observational study in the "Obstetrics & Gynecology Department of R.L. JALAPPA Hospital and Research Centre, Kolar". Women who met the inclusion criteria underwent antenatal transabdominal ultrasound examinations between 18 and 24 weeks of pregnancy to determine the location of the placenta, depending on which they were divided into two groups namely central and lateral placenta. These women were then followed up and monitored for the development of preeclampsia and the outcome.

RESULTS: It was observed that lateral position of placenta significantly was greater in the LSCS compared to normal vaginal mode of delivery. Preeclampsia affected 26.98% of the women in the study group. Among the subjects with lateral placental location, 48.15% had

incidence of preeclampsia and in subjects with central placental location only 11.11% had preeclampsia. As a result, the occurrence of preeclampsia has risen exponentially in subjects with lateral position of placenta (P value <0.001). The Placenta had sensitivity of 48.15%, Specificity was 88.89% and total diagnostic accuracy was 71.43% in predicting Lateral Placenta Preeclampsia.

CONCLUSION: The outcomes revealed a substantial increase in the frequency of preeclampsia in participants with a lateral placenta.

INTRODUCTION

INTRODUCTION

Maternal hypertensive disorders (a hypertensive disorder of pregnancy - HDP) are the prime reason of perinatal and maternal death and morbidity globally, affecting up to 10% of all gestations. [1]

Preeclampsia, gestational hypertension, superimposed preeclampsia, and chronic hypertension are the four organized kinds of hypertensive disorders of pregnancy. [2]

"Blood pressure SBP > 140 mmHg and DBP > 90 mmHg are indicators of pregnancy-induced hypertension. SBP 140 to 149 and DBP 90 to 99 mmHg are considered mild, SBP 150 to 159 and DBP 100 to 109 mmHg are considered moderate, and SBP 150 to 159 and DBP 100 to 109 mmHg are considered severe (SBP 160 to DBP 110 mmHg)". "One of four medical conditions is referred to as pregnancy-induced hypertension (PIH): Chronic hypertension, gestational hypertension, long-term hypertension with preeclampsia, and preeclampsia are the four types of hypertension". [3]

Preeclampsia and eclampsia handle over 15% of all maternal fatalities globally. Aside from contributing to pregnancy consequences, as preeclampsia is a multiorgan disease, HDP elevates the chance of various postpartum issues, notably postpartum hypertension, coronary heart disease, intracranial haemorrhage, high cholesterol, chronic high BP and chronic high blood sugar levels. The risk factors for hypertensive disorders of pregnancy are widely perceived. High body fat, advanced maternal age, first pregnancy, history of maternal hypertensive disorders, high blood sugar level during pregnancy, pre-existing illnesses including chronic high blood sugar, family medical history, hereditary risk, consumption of alcohol during pregnancy, and additional conditions like anemia and urinary tract infections are the risk factors. [1]

Preeclampsia is a trophoblastic tissue condition. It is a complicated clinical condition involving several organ systems that continues to be the leading cause of maternal and neonatal morbidity and death. [4]

Pregnant women presenting with such difficulties should receive prompt and efficient care in order to avoid the majority of preeclampsia-related deaths. Worldwide, preeclampsia and its complications caused roughly 76,000 deaths among pregnant women each year. [5] In order to prevent such a disorder, timely screening is necessary and examination of the substantiate segments of population are required to discover the disease at an initial phase. The most important component in preventing this is right prediction.

The major part of blood to the uterus is delivered by uterine arteries, a branch of the internal iliac artery. The location of placental implantation within the uterus during pregnancy may play a significant role in determining placental blood flow. Non-invasive Doppler investigations of uterine arteries in the second trimester indicate abnormal waveforms suggestive of inadequate uterine perfusion induced by placental implantation when one artery supplies most of the intervillous flow. [6,7]The location of the placenta may be detected using a variety of methods, including X-rays and isotopic placentography. The safest, simplest, and most precise approach for determining placental position for the past 20 years has been ultrasonography. In women with aberrant waveforms, the placenta may be lateral, which has been linked to preeclampsia, IUGR, and other conditions[4,8]

NEED OF THE STUDY

An essential component supporting pregnancy is the placenta. The anomalies in the placental trophoblastic tissues handle the various gestational issues. Preeclampsia, a hypertension disorder of pregnancy caused by anomalies in the placental trophoblastic tissues, is the leading cause of maternal mortality and morbidity. Preeclampsia development and placental position have been linked in several studies.[8–10] The placental position by ultrasound at 18 to 24 weeks has been found to be one of the most cost-effective, non-invasive, and strongly predictive predictors of preeclampsia among the other predictors.[11,12] Preventive strategies for preeclampsia continue to be difficult to implement, and the hunt for the appropriate predictive test is still ongoing. The study's goal was to assess lateral placental implantation and its relationship to the development of preeclampsia and its outcome.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES

- To assess the placental laterality determined by antenatal ultrasonography.
- To establish association between placental laterality and development of preeclampsia and its outcome.

REVIEW OF LITERATURE

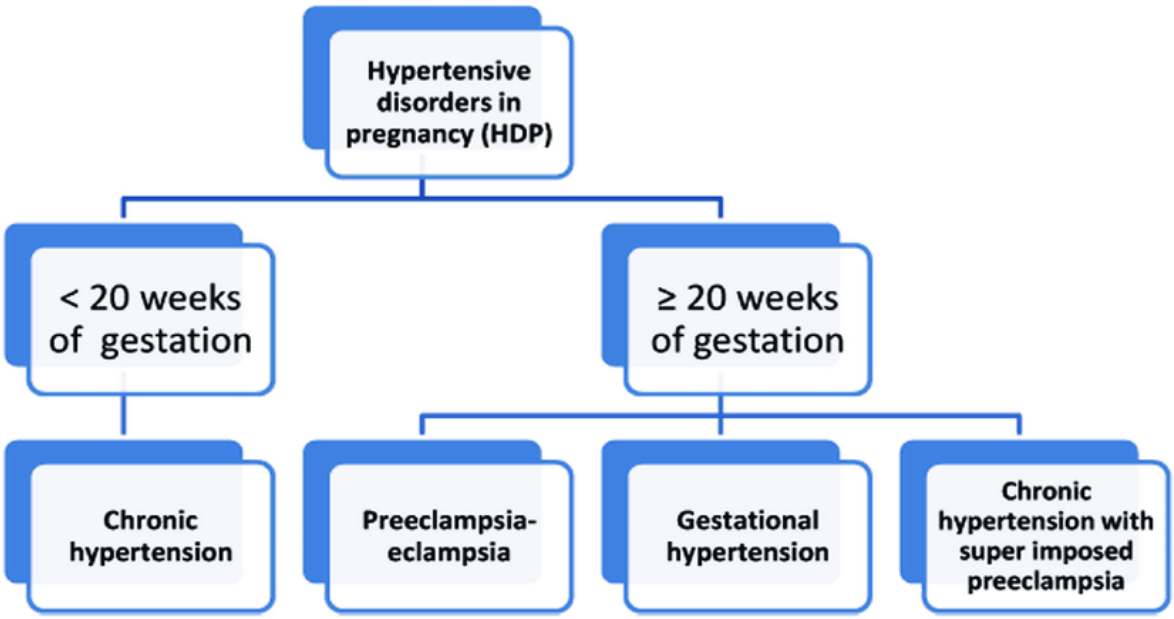
REVIEW OF LITERATURE

1. Pre-eclampsia

What is pregnancy induced hypertension, how pre-eclampsia is a part of it

Preeclampsia is characterized as a SBP >140 mmHg and a DBP > 90 mmHg. It is categorized as follows: mild (SBP 140 to 149 and DBP 90 to 99 mmHg), moderate (SBP between 150 to 159 and DBP between 100 to 109 mmHg), and severe (SBP 150 to 159 and DBP 100 to 109 mmHg) (SBP 160 to DBP 110 mmHg). Pregnancy-induced hypertension (PIH) refers to one of four medical problems: a) chronic hypertension; b) gestational hypertension; c) long standing hypertension with lay over preeclampsia and d) preeclampsia [3]

Figure 1: "Classification of hypertensive disorders in pregnancy". [13]



Definition, classification of pre-eclampsia,

In line with the "International Society for the Study of Hypertension in Pregnancy" (ISSHP), the start of high BP during gestation is demarcated as a "determinedly high systolic/diastolic blood pressure of 140/90 mm Hg as well as proteinuria of 300 mg/24 h after 20 weeks of pregnancy in women with previously normal blood pressure".[14]

Table 1: Basic Differences between early and late preeclampsia[15]

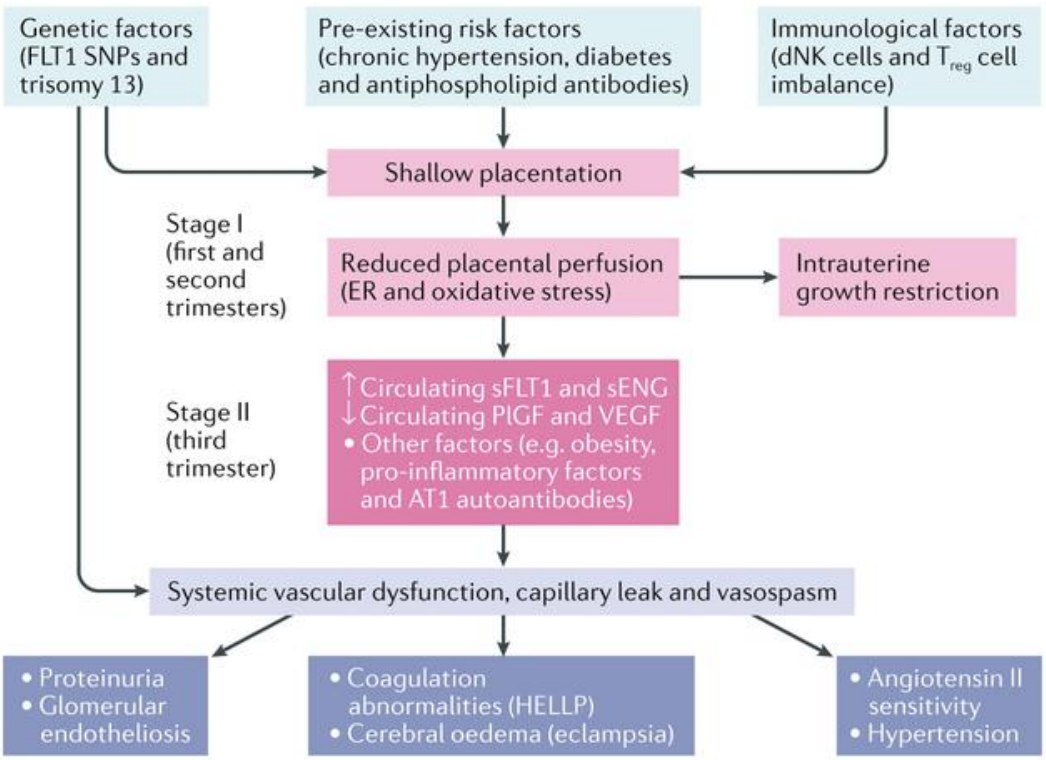
Early onset preeclampsia (≤ 34 weeks of gestation)	Late onset preeclampsia (≥ 34 weeks of gestation)
A fetal diseases that is typically associated with placental dysfunction	Maternal disorder due to underlying maternal constitutional factors
Reduction in placental volume (Figure 3)	Normal placental volume
IUGR	Normal fetal growth
Abnormal uterine and umbilical artery Doppler measurement	Normal uterine and umbilical artery Doppler evaluation
Adverse maternal and fetal outcomes	Favorable maternal and neonatal outcomes
Low birth weight	Normal birth weight

IUGR – intrauterine growth restriction.

Pathogenesis of preeclampsia

Atypical placentation and the emergence of the maternal syndrome are the two phases of pre-eclampsia pathophysiology.

Figure 2: The pathogenesis of pre-eclampsia.



Placental dysfunction (stage I) can be caused by genetic, maternal, and immunological factors, which primes to the production of Preeclampsia is brought on by inflammatory mediators and antiangiogenic features, such as accessible sFLT1 and soluble endoglin (sENG) (stage II). Golgi apparatus, placental growth factor, SNP, regulatory T cell, and endothelial vascular growth factor.[16]

Atypical placentation

The placenta is assumed to be the origin of pre-eclampsia, and the term placental syndrome might be used to characterize its early phases. Pre-eclampsia occurs in hydatidiform moles, suggesting that the placenta is needed for the syndrome to occur rather than the foetus. Atherosclerosis, sclerotic restriction of arterial vessels, fibrin buildup, and infarcts are some of the pathological characteristics of pre-eclamptic placentas. All of these clinical abnormalities are associated with reduced blood supply to the placenta and ischaemia, and they appear to be connected to the severity of pre-eclampsia. Additionally, hypertrophic decidual vasculopathy— a substantial increase in cell size of the media in the decidual arteries —has been documented.[17]

Pre-eclampsia is a condition that affects humans and doesn't appear to affect any other species. The disparately high ratio of the human fetus's brain to body weight, which requires 60% of the mother's nutrients interchanged during the 28 to 40 weeks of gestation against just 20% in other animals, is thought to be the cause of this specificity. The maternal vessels undergo structural changes and adaptations during normal placentation to make room for the necessary flow of blood to the growing fetus. The uterine radial arteries split into 2 or additional branches that either stop in the myometrium or decidua (basal artery) or enter the intervillous region. The entrance into

the “intervillous space” influences the “cytotrophoblasts” that penetrate the “spiral arteries”.[16]

These spiral arteries eventually show a lack of elastic and muscular tissue, a discontinuous endothelial coating, and often have mural thrombi. To suit the placenta's high blood flow demands and to dominate the vasomotor regulation of the parental arteries, it is put forward that the spiral arteries must shift from tiny muscle arteries to giant tortuous channels. The foetus suffers from hypoxia and ischaemia if this remodelling of spiral arteries doesn’t occur. For well over a century, scientists have been studying the features of pre-eclamptic placentas. When related to pregnant female who do not have protein, generally albumin, in their urine, women with "toxaemia, albuminuria, and eclampsia" had a higher frequency of placental infarcts in 1914, according to Young.[16]

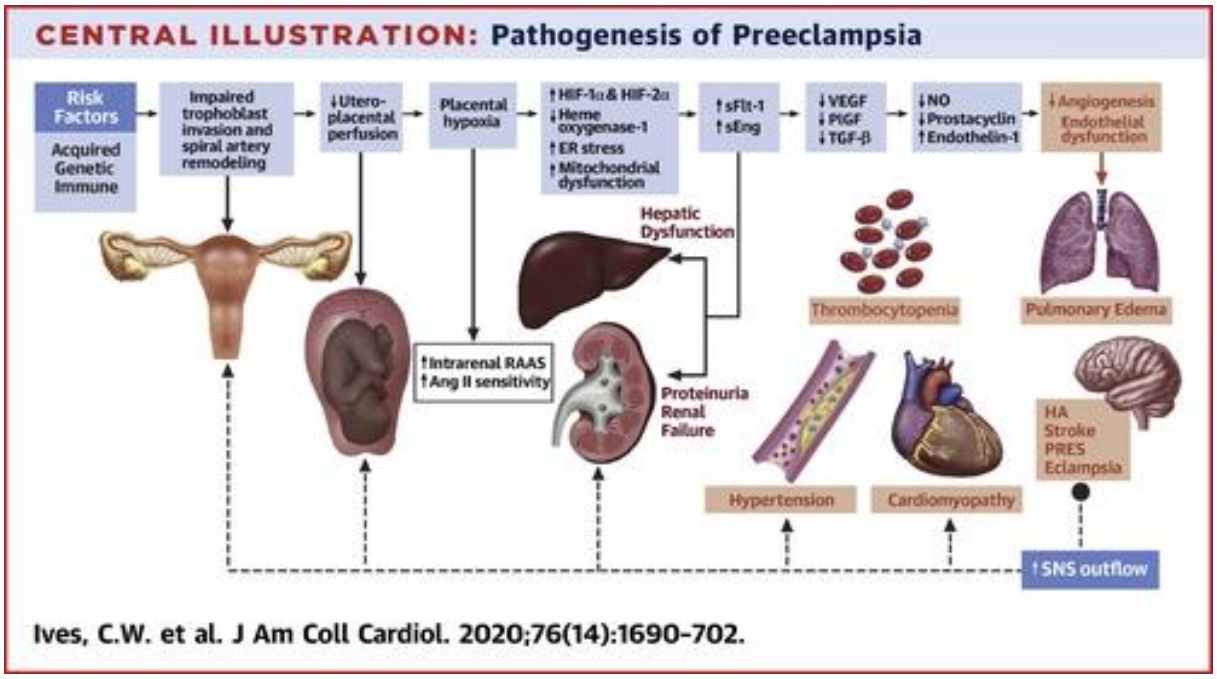
The infarcts indicated ischaemia and placental hypoperfusion. In the 1960s, a number of organisations attempted to explain the alterations in placentation among pre-eclamptic and normal blood pressure pregnancies. Endothelium and media of the basal and spiral vessels proliferated, and there were many mural thrombi of the vasculature in the placental bed biopsies of pregnant females with multiple hypertensive diseases of pregnancy and chronic hypertension. Foam cells were seen in specimens from pre-eclamptic and eclamptic uterine beds, indicating acute atherosis, and acute fibrinoid degradation of the vascular wall. These characteristics were very different from those found in the samples. In pre-eclamptic placental beds, lipophage infiltration and total thrombotic obstruction of arteries were also frequent.[16]

The revelation that in preeclampsia, the functional alterations of the “spiral arteries” were limited to the decidua but expanded nearer to the centre into the myometrium in pregnancy provided additional evidence in favor of the ischaemic placenta theory.

Furthermore, in their collection of maternal-fetal interface biopsy samples, pre-eclamptic samples had spiral arteries with an average diameter of only 200 mm, as opposed to 500 mm in placentae from healthy pregnancies. The proximal portions of the spiral arteries are widened and enlarged by the exterior penetration of the decidua, which causes hypoperfusion of the uterus and greater blood circulation rates to the inter - membrane area.[18]

In the pre-eclamptic maternal-fetal interface, lipophage infiltration and total thrombotic obstruction of arteries were also frequent. These conclusions were supported by a study that revealed a significant flaw in the remodelling of the myometrial spiral arteries, which was more common when there was a link between pre-eclampsia and severe foetal growth restriction. [19] There is ongoing discussion regarding the molecular mechanisms underlying spiral artery remodelling. Studies have shown that pre-eclampsia inhibits the shift of cytotrophoblasts from an epithelium to an “endothelial phenotype” during normal placentation, a process termed as pseudo-vasculogenesis or "vascular mimicry."[20]

Figure 3: pathogenesis of preeclampsia



Stress

Stress from oxidation. Additionally, present at the placental bed, oxidative stress is considered to have a substantial role in both healthy and unfavorable placental growth. It is believed that early in gestation, oxidative stress and apoptosis play a role in the proper closure of the outer villi, which are where placental blood flow starts. An imbalance between pro- and anti-oxidant pathways seems to arise in pre-eclampsia.[20] This imbalance may be produced by inappropriate spiral artery remodelling, which occurs in afflicted pregnancies and is hypothesised to induce recurrent ischaemia-reperfusion damage due to the retention of spiral artery contractile segments in the myometrium. [16,22] After ischaemia and reperfusion, in vitro studies on human placental tissue revealed elevated levels of reactive oxygen species, which are consistent with this hypothesis. [22]

Maternal syndrome

Pre-eclampsia symptoms extend beyond the placenta and include numerous effects on the mother as well. [23]These effects are collectively known as the maternal syndrome (stage II). Pre-eclampsia and eclampsia pathologic lesions are distinguished histologically by severe endothelial lesions in several organ beds.[24]

Epidemiology

Preeclampsia is thought to cause 50,000 female deaths annually worldwide. Depending on the population studied and how preeclampsia is defined, the incidence ranges from 2-10%. The incidence was reported as 2.8% from an Israeli study, 5.8% from Scotland, 14.1% from Australia, and 5% from Seattle. It affects 5 to 8% of expectant mothers globally and can lead to the most serious issues for both the mother and the child.[24]

In affluent countries, hypertensive illnesses account for 16% of all maternal mortality and up to 26% of postpartum deaths in Caribbean and Latin America, 9% of postpartum deaths in Africa, and the rest of the world..[25] Eclampsia instead of preeclampsia is the main cause of death in regions with a high prevalence of maternal mortality.[26] The incidence of preeclampsia during labor and birth grew by 25% between 1987 and 2004, while the rate of eclampsia fell by 22%, according to the US National Review Survey. However, the changes were just not substanstial.[27] Severe morbidities such renal failure, strokes, cardiac arrest or arrest, pulmonary impairment, coagulopathy, and hepatic cirrhosis are all possible results of preeclampsia and eclampsia.[28,29]

Etiology, risk factors[30]

Positive risk factors

- Pre-eclampsia family history
- No born children
- Multiple pregnancy
- Women >35 years of age
- IVF "In vitro fertilization"
- Comorbidities in the mother, such as SLE, hypertension, obesity, long standing renal ailment, and DM
- A history of intrauterine foetal growth restriction or placental abruption
- Trisomy 13- patau syndrome
- Molar pregnancies

Negative risk factors

- Parental smoking
- Long-term sexual cohabitation [23,31]

Clinical presentation

Figure 4: Pre-eclampsia symptoms and signs by organ system. [32]

Systems	Signs/Symptoms
Central Nervous system	Headaches Visual disturbances Seizures (eclampsia)
Renal system	Proteinuria Oliguria Abnormal kidney tests Hypertension
Vascular system	Severe hypertension
Cardiorespiratory system	Chest pain Dyspnea Low oxygen saturation Pulmonary edema
Hepatic system	Abnormal liver function Epigastric pain Nausea
Hematologic system	Hemorrhage Coagulation impairment Intravascular disseminated coagulation Shock

Diagnosis

Proteinuria and newly raised blood pressure after 20 weeks of pregnancy are diagnostic markers of preeclampsia. Edema and blood pressure increases beyond the patient's average level are not considered diagnostic markers. Severe preeclampsia is characterised by higher blood pressure(more than 160/100mmhg) and more severe proteinuria. Other symptoms of severe preeclampsia include oliguria, cognitive or visual deficits, pulmonary edoema, or cyanosis. [33]

Diagnostic Criteria for Preeclampsia

The actual definition of pre-eclampsia included proteinuria of 300 mg in a 24-hour obtaining, 0.3 g/g by “urinary protein:creatinine ratio”, or +1 by urinary test strip if

quantifiable methods are not available. Additionally, it included a rise in SBP to 140 mmHg or a rise in DBP to 90 mmHg on 2 separate cases in a subjects who had earlier been normotensive. Proteinuria was ruled out as a diagnostic factor in cases with other end-organ disease, such as thrombocytopenia, impaired liver performance, novel kidney problems, oedema, or newly manifested brain or visual issues. "Pre-eclampsia severe symptoms are characterised by at least one of the following traits: 2 metrics of 160/110 mmHg, a platelet count of 100,000/ml, unusually high liver function test results that are twice the regular concentration, symptoms of hard right upper dimension or epigastric pain, kidney problems indicated by a creatinine level greater than 1.1 mg/dl (97.2 mol/l)".[34]

Preeclampsia

Blood pressure in a woman with prior normal range must be 140 mm Hg or greater systolic or 90 millimeters of mercury or greater diastolic after 20 weeks of pregnancy on two occasions 6 hours apart.

Proteinuria is defined as 0.3 g or > protein in a 24-hr urine sample (normally 1+ or more on a urine dipstick test).

Severe Preeclampsia

In a woman on bed rest, blood pressure should be 160 mm Hg or > systolic or 110 mm Hg or > diastolic on two occasions at least 6 hrs apart.

Proteinuria is characterised by the presence of 3+ or more on dipstick analysis of two random urine samples taken at least 4 hours apart, or by the existence of five g or over of protein in a 24-h urine collection.

Oliguria (a urinary output of < than 500 mL in 24hrs), abnormalities of the brain or vision, pulmonary edoema or cyanosis, pain in the right upper quadrant or the

epigastrium, impaired liver parameters, thrombocytopenia, and intrauterine restriction of growth are other symptoms.

Eclampsia: presence of seizures, with all other possible causes of seizures being ruled out in a women with preeclampsia.

Chronic hypertension: when the blood pressure is more than 140/90mmhg which is diagnosed before 20weeks of gestation or even prior to conception.

Superimposed preeclampsia on chronic hypertension: In a women with chronic hypertension, development of proteinuria newly prior to 20 weeks.

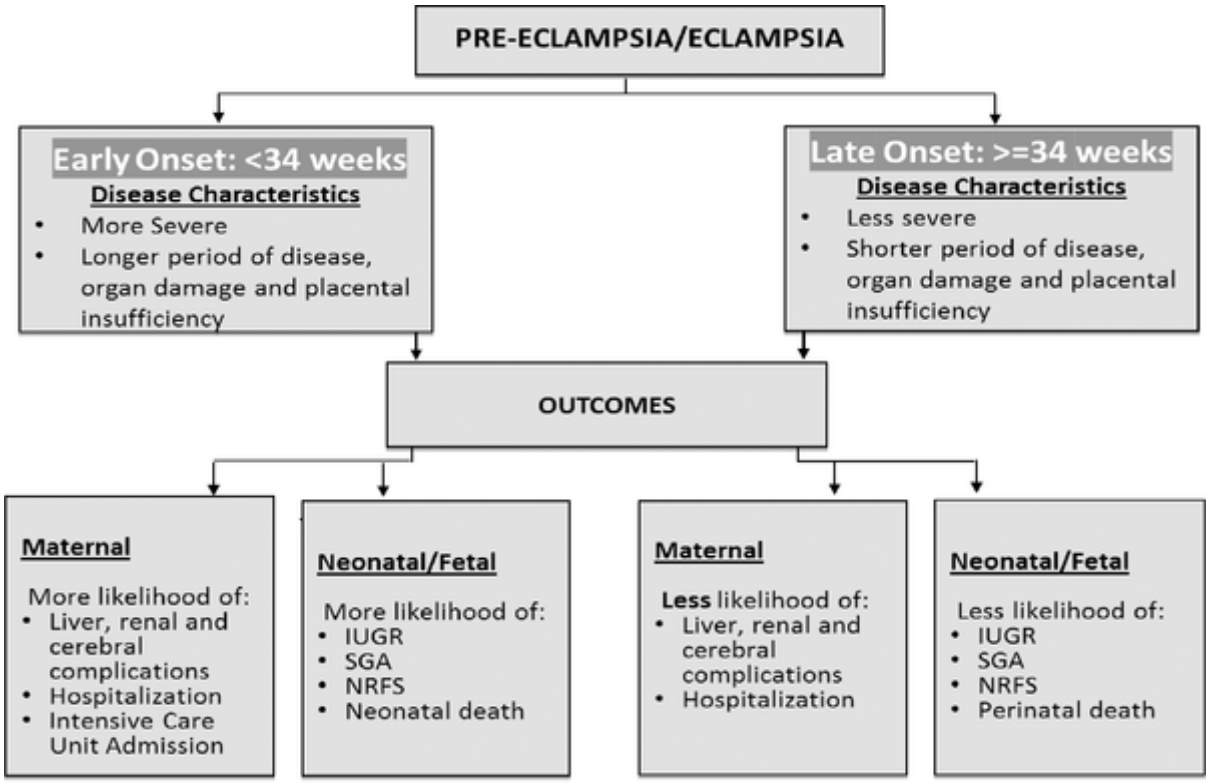
Morbidity, mortality associated complications, maternal and neonatal outcome

Preeclampsia, impacts 5% to 7% of all pregnant women but results in the morbidity and mortality are greater, which causing more than 7 lakh maternal fatalities and 5 lakh foetal mortality worldwide each year. [35]

Figure 5: Complications of pre-eclampsia per organ system [36]

Central nervous system	Eclampsia
	Cerebral haemorrhage
	Cerebral edema
	Retinal edema
	Cortical blindness
Renal system	Renal tubular necrosis
	Renal cortical necrosis
	Pulmonary edema
	Laryngeal edema
Hepatic system	Disseminated intravascular coagulation
	Hemolysis
Liver	Hemolysis, elevated liver enzymes, low platelets (HELLP) Syndrome
	Jaundice
Placenta	Placental abruption
	Placental infarction
Baby	Growth restriction
	Preterm delivery
	Death

Figure 6: Maternal and fetal outcomes associated with preeclampsia [37]



IUGR, Intrauterine growth restriction; SGA, Small-for-Gestational-Age; NRFS, Non-reassuring Fetal Status

Prevention

Research on innovative therapeutics for either the prevention or management of preeclampsia has been sparked by recent developments in understanding the pathophysiology of the condition and the desire to lessen its long- and short-term morbidities. H2 blockers, antithrombin, relaxin, glutamyl reductase inhibitors (statins), usage of apheresis, and other substances fall under this category. Studies have shown that, the most effective medicines for preventing preeclampsia, such as aspirin, atorvastatin, metformin, and proton pump inhibitors. [41]

Utilizing treatment to prevent PE problems is a key component of tertiary prevention. Magnesium sulphate, for instance, is the medication of choice for lowering the incidence of eclampsia.[42]

All these preventive measures are directed towards reducing the vasospasm , platelet aggregation and endothelial damage.

Such measures are still not characterized as being definitive, the only possible preventive measure is to detect the development of preeclampsia early by looking for the signs and symptoms.

Management

Preeclampsia prevention, early detection, and therapy are the three pillars of effective preeclampsia care. [38]Preeclampsia is thought to be highly risky for women (such as those who have concomitant renal illness, chronic hypertension, or autoimmune antiphospholipid antibody syndrome) should be sent for pre-pregnancy counselling to determine variable risk factors). This treatment may involve nutritional advice like salt restriction, smoking cessation counselling, medication modifications to treat existing medical conditions including kidney disease, and the cessation of medications like warfarin and ACE inhibitors that might have teratogenic consequences. The baseline values for renal function, platelet function, blood pressure, and renal function are significant. Preeclampsia risk may be lowered by a favourable prenatal condition. Low-dose aspirin should be used from week 12 of gestation to week 36 of gestation by women who are thought to be at high risk. In females with low calcium diets, calcium fortification (1 g/day) has indeed been linked to a significantly lower incidence of preeclampsia.[39,40]

2. Lateral implantation of placenta

Normal implantation is essential for a healthy pregnancy. In a nutshell, the human blastocyst usually implants in the upper region of the uterus about 6-7 days following

conception, when the implantation in the endometrium takes place. Beginning with the blastocyst's attachment to the endometrium, it progresses to endometrial invasion. The innermost 1/3 of the myometrium and the mother spiral vessels are invaded by the trophoblast just on anchoring villi via the endometrium. Placental abnormalities, maternal vascular malperfusion brought on by insufficient remodelling of the paternal spiral arteries, atypical placentation, and morbidly attached placenta are a few of the problems brought on by implantation defects.[43] [44]

Pathogenesis

The pathophysiology appears to be caused by an anomalous interface between the decidua and the extravillous trophoblast, which prevents the maternal tissues from controlling the trophoblast from invading. [45] [46] [47] [48] Many explanations have been proposed to account for this phenomenon. One of these is a paucity of anti-invasive elements that are antagonism to matrix metalloproteinases or stimulators of tissue antagonists of metalloproteinases, which are secreted by the decidua, in regions with reduced decidualization. A discrepancy in the autocrine regulation between the trophoblasts that really are invading and the undeveloped decidualized endometrium is yet another possible cause.[49] [50]

Risk Factors and Etiology,

The most prevalent cause of aberrant placentation is abnormal decidualization, which can be resulting from trauma or decidua shortage, as in situations of placenta previa implanting in the lower uterine section or a caesarean scar. [50]

Any elements that can end up harming or scarring the endometrium are considered risk factors. The most significant risk factors are history of spontaneous or induced abortion,

previous caesarean birth or uterine surgery, including prior dilatation and curettage, and myomectomies, as well as prior pregnancies with aberrant placentation. [51] [52] Atypical placentation has been linked to endometriosis and aided conception in primigravida. [50]

Placental laterality in USG – diagnostic accuracy,

Numerous clinical, biochemical, and biophysical techniques have been suggested for assessing preeclampsia early or mid-pregnancy. Placental perfusion and vascular confrontation (mean BP in the 2nd trimester, cold pressor test, axonometric hand grasp exercise, turn over test, IV injection of angiotensin II, 24-hr ambulatory blood monitoring), fetoplacental unit endocrinology (α-fetoprotein, hcg , follicle stimulating hormone), renal function (serum uric acid or microalbuminuria). Among the several preeclampsia warning signs, ultrasound of the placenta from 18 to 24 weeks is reasonably inexpensive, non-invasive, and has a high predictive value. The placental position as assessed by ultrasonography between weeks 18 and 20 of pregnancy has been shown to be a reliable screening technique.[53]

3. Relationship between placenta laterality and preeclampsia development and outcome

A condition affecting trophoblastic tissues is pre- eclampsia. Pre-eclampsia-related quantitative investigation of trophoblast invasion had revealed limited trophoblastic cell invasion. Pre-eclampsia only manifests itself when the placenta is present. The placenta serves as a crucial pathway for metabolic, endocrine, and other bodily activities between the mother and the foetus. The placenta's blood supply is not distributed equally. As a result, the placental's placement within the uterus because of the implantation site is probably a key factor in influencing placental blood flow and,

ultimately, pregnancy success. Both uterine arteries in humans have many branches, and each of them supports the uterus on its respective side.[54]

The 2 uterine arteries are connected by anastomoses, however there is little proof that they are advantageous. Both uterine arteries had similar confrontation in people with a centrally located placenta, and the uteroplacental blood circulation requirements were satisfied by equitable split from both uterine arteries. One uterine artery typically meets the majority of the maternal blood flow requirements when the placenta is positioned laterally, with some assistance from the second uterine artery through collateral circulation.

Different subjects may have different levels of collateral circulation, and a lack of involvement may promote the development of IUGR, pre-eclampsia, or both. A healthy placenta is critical for this "cytotrophoblastic" invasion, and pre-eclampsia causes the cytotrophoblasts to fail to establish a vascular adhesions phenotype.

Pre-eclampsia, intra uterine growth restriction (IUGR), or both have a significantly positive correlation with placental position, uterine artery resistance, and unfavourable outcomes. The placement of the placenta as assessed by USG between 18 and 24 weeks is therefore a non-invasive, economical, and secure prognosticator of pre-eclampsia.[55]

Association between placental laterality and maternal, neonatal outcome

The placenta is an important organ that joins the growing fetus to the uterine wall. The placenta performs the fetus's metabolic, endocrine, respiratory, and excretory activities. Maternal and fetal circulations are present in the placenta. Placental abnormalities can cause adverse maternal and fetal outcomes such as pregnancy induced hypertension, pregnancy induced diabetes, malposition, malpresentation, preterm birth, SGA, IUGR, LBW, intrauterine demise, abortion, and so on. The various divisions for placental

location (inside 2 cm of the internal os) include central, unilateral (left lateral, right lateral), fundal, and low lying. [56]

Sumathi, N et al [8] aimed to assess the connection between preeclampsia incidence and placental location. to research the prevalence of preeclampsia in both patients with laterally and centrally implanted placentas. to ascertain whether ultrasound-measured placental laterality can be employed to identify the onset of preeclampsia. Out of a total of 250 women, 123 had lateral placentas, and 98 (79.6%) of those women experienced preeclampsia. 22 (17.3%) of the 127 women with central placentas out of the remaining 127 developed preeclampsia. Therefore, it was found that there was a significant laterally placed placenta increases the overall risk of preeclampsia. The value of P is <0.001.

Yousuf S, et al. [12]conducted a cohort research on 201 participants. Preeclampsia was diagnosed in 37 (52%) of the 71 (24.5%) women with laterally positioned placentas, while it occurred in 14 (10.8%) of the 130 (75.5%) women with centrally located placentas. Preeclampsia was diagnosed in 35 (92%) of the 38 participants with lateral placentas with Doppler abnormalities, compared to 2 (6%) of the 33 subjects with lateral placentas alone. With a laterally placed placenta, the overall risk of having preeclampsia was 9.27(OR).

Ambastha V, et al.[55] performed a prospective study on 250 participants. Preeclampsia occurred in 8 (5.41%) of the 148 women with central placentas and 40 (39.22%) of the 102 women with lateral placentas. As a result, out of the 48 women who had PE, 40 (83.33%) had lateral placentas while only 8 (16.67%) had central placenta. According to the study's findings, women with placentas that are laterally positioned as indicated by USG are five times more likely to experience PE. Therefore, placental laterality is a straightforward, trustworthy, and affordable predictive screening test for pre-eclampsia.

Keshavarz E, et al.[57] conducted a case control study. According to this study, the best strategy to forecast the chance of evolving pre-eclampsia is to do a routine screening ultrasonography test that can readily assess the placental position during the middle trimester of pregnancy.

Gupta A, et al.[5] conducted a study on 200 antenatal female. They found 84 having lateral placentas and 116 having central placentas. Preeclampsia was developed in 55 (65.5%) of the 84 women with lateral placentas and in 28 (24.1%) of the 116 (58%) women with central placentas. According to this study, preeclampsia was significant to occur in females whose placentas were detected by ultrasound at a later stage of pregnancy—between 18 and 24 weeks.

Granfors M, et al. [58] conducted a cohort research. Lateral placental locations were associated with a variety of adversative pregnancy consequences in comparison to posterior placental placement, the most important of which were mechanical placenta removal in natural births and extremely preterm delivery. Additionally, preeclampsia and severe postpartum bleeding were linked to the position of the placenta on the lateral side. Fundal and lateral placental p

lacements have a number of negative pregnancy, birth, and infant outcomes compared to posterior placental location.

Bhalerao A, et al.[4] studied 281 female with low risk factors and 182 females who had high risk factors. In all, 71 pregnant women were afflicted with preeclampsia, and 52 of them (73.23%) had lateralized placentas. This connection appears to be important. The placental location and delivery method had no relationship. Cesarean delivery rates were 26.78% while normal delirium rates were 73.21%. 48 (10.36%) of the caesareans had placentas that were positioned laterally, while 76 (16.41%) had placentas that were

positioned centrally. Based on the findings of this study, preeclampsia can be predicted using ultrasonography in pregnant women between 20 and 24 wks of pregnancy . It is a simple, noninvasive, helpful, and affordable method.

Pawar SM, et al. [59] conducted a study on 75 antenatal patients. There was a strong association between the placenta's position and the success of the pregnancy. According to this study, the placement of the placenta, which can be identified with standard ultrasonography, is significantly correlated with the success of a pregnancy.

Faizi S, et al. [60] performed a observational study on 620 females. In comparison to other placental implantation locations, the lateral position of the placenta is linked to unfavorable prenatal outcomes such pre-eclampsia, antepartum hemorrhage, and IUGR.

Pillay R, et al. [61] conducted a research on 100 subjects. Only 7 (10%) of the women in the group with the central placenta had hypertension at the time of follow-up, as opposed to 21 women in the group with the lateral placenta. Around 75% of the subjects who established hypertension had placentas in lateral position .

Soleimani Z, et al. [62] conducted a study on 1000 gestational women subjects. In 44%, 42.1%, 8.2%, and 5.7% of cases, the placental site placement was anterior, posterior, lateral, and fundal, respectively. The lowest and greatest mean birth weights were 2999.3±643.9 and 3269.7±1776.9 gr in patients with lateral and posterior placental site locations. Preterm births made up 4.88% of the babies in the lateral group, which was substantially more than the other groups. In comparison to other groups, the lateral group had an IUGR birth rate of 4.88% .

Jaiswal J, et al. [63] performed an observational randomized study on 130 women. The placenta was implanted centrally in 71.5% of cases, while the lateral form was seen in

28.4% of cases. The ratio was nearly the same for primi and multigravida. There were 28/130 cases (21.5%) of hypertensive disorders in pregnant women in the cohort under study. When compared to the central placenta, which had a prevalence of 9.7%, the lateral placenta had a high occurrence of PIH (51.3%).

Rai A, et al. [6] conducted a study on 106 females. There were 37 lateral placenta and 69 central placenta in the patients. Preeclampsia affected 17 patients in all, 12 of whom had placentas that had been implanted laterally. This study showed a substantial risk of preeclampsia in patients with laterally placed placentas, pointing to the need for a preventive strategy and the best possible care of preeclampsia.

Chhabra S, et al.[64] found that that risks can be identified by studying the position and extent of the placenta in the initial stages of pregnancy.

Kakkar, T et al[65], Ultrasound's capacity to identify placental laterality and anticipate the start of preeclampsia. Out of the 150 women, 56% had lateral, and majority had preeclampsia. The remaining 44% had centrally placed placentas, as did 24 (36.3%) of those. With a p value of, the v2 test indicated that the difference was statistically significant (0.00002). They concluded from the preceding study a placenta at lateral position as assessed by USG at 18-24 wks of gestation are more likely to get preeclampsia.

Patil AS, et al. [66] performed a prospective study on 108 women. In comparison to centrally positioned placentas, lateral placentas displayed higher SBP and DBP at every GA. However, only statistically significant findings are obtained for gestational ages 36–37. In the current study, lateral placentas were found in 61.1% of preeclampsia patients, whereas central placentas were found in 37% of patients. In comparison to a placenta that was positioned in the middle, a placenta that was positioned laterally showed greater Systolic and Diastolic blood pressure, which is statistically significant.

Elbehissi OM, et al.[67] A prospective observational cohort research was carried out. The placenta was centrally situated in 64.9% of the instances studied, and laterally located in 35.1% of the cases. In comparison to central placentas, lateral placentas exhibited a lower rate of intrauterine growth restriction but a higher risk of preterm labour and premature membrane rupture. According to the study, the progress of pregnancy induced hypertension and other undesirable consequences did not differ considerably depending on where the placenta was located in the body.

Ghadei, R et al[68] study is to ascertain whether placental laterality and preeclampsia are related. At 18 to 24 weeks of pregnancy, 300 singleton gestation women who do not have any other obstetric or medical conditions are subjected to ultrasounds, and they are then monitored for preeclampsia development until delivery. Of the 76 lateral placentas in 300 women, 28 (36.84%) went on to develop preeclampsia. A statistically significant association between preeclampsia and centrally located placenta was found in 27 (12.05%) of 224 women who had the condition. A lateral placenta is linked to a four times greater peril of preeclampsia. Consequently, preeclampsia can be predicted using this.

Alpesh Patel et al[9], aimed to determine whether preeclampsia and the uterine artery resistance index are related. In this study, 200 non-high risk primigravida were included, and all underwent ultrasonography between weeks 18 and 22 of pregnancy to determine whether the placenta was central or lateral. Undergoing color Doppler, all patients with lateral placentas had changes to the uterine artery resistance index recorded. Preeclampsia was observed in all patients up until delivery and noted. Preeclampsia was discovered in 14 out of 40 patients with lateral placenta.

Kore, S et al[10], study set out to determine if placental laterality, as assessed by ultrasonography between 20 and 24 weeks, may be used to predict the onset of

preeclampsia. This research comprised 200 low risk singleton pregnant women engaged in an antenatal programme at a tertiary teaching institution. Ultrasound was utilised to find the placenta between 20 and 24 weeks. There were two groups of women: group B had a lateral placenta, whereas group A had a central placenta.There were 161 (Group-A) central placentas and 39 (Group-B) unilateral placentas among 200 expectant women (Group-B). Preeclampsia affected 32 women in all.

A retrospective analysis by Devarajan, K et al[69] looked at the relationship between placental position and infant weight. A cohort of 796 singleton births occurred in a row and were under 37 weeks gestation. The position of the placenta had no effect on new-born weight or other perinatal outcomes.

LACUNAE OF LITERATURE

The position of the placenta can be used to predict pregnancy from the routine antenatal scan. Early detection of pre -eclampsia reduces maternal morbidity and mortality by preventing terrible maternal complications. An easy, painless, and economical predictive screening test for the onset of preeclampsia is ultrasonography. It is advised being vigilant for careful obstetric management if a lateral placenta is found on ultrasound in order to achieve a more favourable outcome, avoid some terrible complications, and lower mother and perinatal complications and death. Despite vast studies, there is no viable, practical way for predicting the onset of preeclampsia. Placental placement by ultrasound at 18 to 24 weeks of pregnancy appears to be a practical strategy in underdeveloped countries with limited resources for categorising females as greater-risk cases for the onset of preeclampsia and minimising morbidity and death. There, has been least studies in India, to investigate the clinical importance of placental laterality and its link to the development of preeclampsia.

MATERIALS &

METHODS

Source of data:

This study was conducted over a period of 18months on the in patients and out patients coming to department of Obstetrics and Gynaecology at "R.L. JALAPPA Hospital and Research centre attached to SDUMC, Kolar".

Study population: Informed consent was taken. They were followed till delivery and were divided into 2 groups based on ultrasound findings of the placental position.

Group I -women who have laterally located placenta

Group II-women who have centrally located placenta

* Based on the initial day of the last menstruation cycle (LMP), the gestational age was calculated, and in the early second trimester, an ultrasound was used to confirm it. The weeks of gestation as stated by the ultrasound examination was taken into consideration if there is a disparity between them of more than ten days. Additionally, the weeks of gestation as established by the ultrasound was taken into consideration when the woman was unclear about her LMP.

Study design: Study design was a cohort study

Study period: Jan to Dec 2021

Method of collection of data:

All subjects provided written informed consent. All the data were collected in Proformas as attached in Annexure -1.

The data that was collected are: age, parity and gravidity of the patient, gestational age at admission and at delivery and

Ultrasonography

Inclusion Criteria:

This study included all females with a singleton pregnancy who attended the antenatal clinic, including outpatient and ward admissions, between 18 and 24 weeks of pregnancy and did not have any high-risk characteristics.

Exclusion Criteria: Pregnant women having chronic hypertension, renal disease, twin pregnancy, diabetes mellitus

Sample size: According to Kakkar tania et al[9] proportion of patients with lateral placenta who developed preeclampsia was 66.6%

Proportion of patients with central placenta, who developed preeclampsia was 36.3% Assuming the alpha error -0.05% (95% confidence limit) and power of 90%

The sample size is calculated to be -126

Ethical considerations: The organizational ethics committee got the go-ahead. All research participants provided written informed consent, and only those who did were counted in the analysis. Before getting agreement, it was mentioned to the participants the risks and advantages of the study as well as the voluntary nature of participation. Participants in the research were kept in the strictest of confidence.

Methodology: At the time of their stages of pregnancy and admission, every subject had to be taken thorough history taken as well as a complete physical, systemic, and antenatal examination. The placement of the placenta was confirmed by ultrasound between 18 and 24 weeks in all the gestational women who were thereafter followed up till delivery. These women were divided into two groups placed on the placental location confirmed by USG. Central placentas are those that,

regardless of their front, posterior, or fundal placements, are distributed evenly between the right and left sides of the uterine wall. The placenta was categorised as unilaterally right or unilaterally left when at least 75% of the total placental mass was found on one side of the midline. All pregnant women received follow-up care during their prenatal visits, and the progression of preeclampsia symptoms was monitored throughout the whole pregnancy. Fetal dopper was also done for those patients who developed increased BP readings during the course. Preeclampsia was characterised by the ACOG as new guidelines, high blood pressure (BP of 140 mmHg systolic and/or 90 mmHg diastolic) when it appeared in a pregnant woman after 20 weeks of pregnancy (defined as urinary excretion of 0.3 g protein in 24 h). Preeclampsia with lateral placental predominance determined the outcome of the pregnancy. More data were examined using dependable statistical techniques.

A. Maternal pulse rate is one aspect of the general evaluation.

B. Blood pressure

C. Contraction of uterus

D. Heartbeat of the foetus.

Following investigations to be done on patients

Routine blood investigations like

CBC

SEROLOGY:HIV, HEPATITIS B

BLEEDING TIME CLOTTING TIME

LFT

RFT

URINE ALBUMIN

URIC ACID, LDH

ULTRASONOGRAPHY OF THE ABDOMEN AND PELVIS IS PERFORMED**.**

STATISTICAL METHODS

Preeclampsia, Eclampsia, IUGR, PPROM, DIC, HELLP, Abruptio & Fetal outcome were regarded result parameters, with Placenta serving as an explanatory variable. Age Group, Gravida, Parity, Live births, Abortion, 1st visit BP, 2nd visit BP, 3rd visit BP & Mode of delivery etc., were study relevant variable.

On quantitative data, using standard deviation and mean as well as on categorical variables, using frequency and percentage, descriptive analysis was carried out. Data too was graphically illustrated using suitable diagrams such as a bar diagram, a pie diagram, and a cluster bar chart.

To compare categorical outcomes across study groups, the Chi - square test test was employed. The placenta was pondered to be the gold standard and preeclampsia as a screening test. The screening test's sensitivity, specificity, predictive values, and diagnostic accuracy, as well as their 95% confidence intervals, were given.

Statistical significance was defined as a P value of 0.05. CoGuide software was used to evaluate the data:

OBSERVATIONS AND RESULTS

RESULTS

A total of 126 subjects were included in the final analysis.

Table 2: Age Group Descriptive Analysis in the Sample Group (N=126)

Age Group	Frequency	Percentage
<20 years	22	17.46%
20-25 years	68	53.97%
26-30 years	27	21.43%
>30 years	9	7.14%

Among the study population, 22 (17.46%) participants were aged less than 20 years, 68 (53.97%) were aged between 20 to 25 years, 27 (21.43%) were aged between 26 to 30 years and 9 (7.14%) were aged between greater than 30 years. (Table 2 & figure 7)

Figure 7: Age Distribution in the Study Subjects as a Bar Graph (N=126)

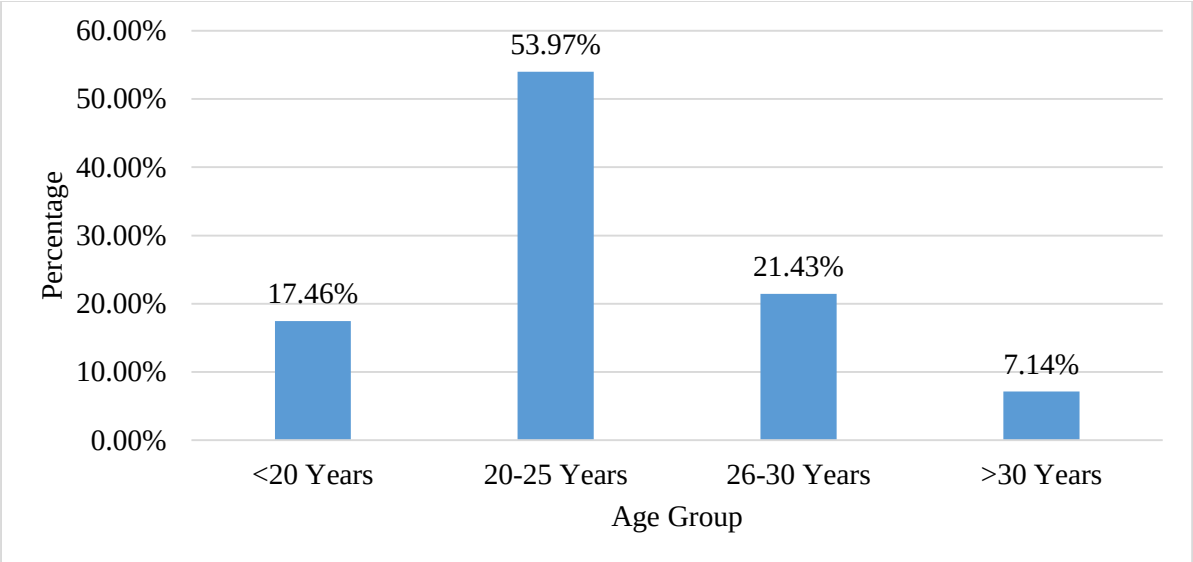


Table 3: comparison of "Preeclampsia" in the study subjects by Age Group (N=126)

Age Group	Preeclampsia		Chi square value	P value
	Yes	No		
<20 Years (N = 22)	6 (27.27%)	16 (72.73%)	0.21	0.9760
20-25 Years (N = 68)	18 (26.47%)	50 (73.53%)		
26-30 Years (N = 27)	7 (25.93%)	20 (74.07%)		
>30 Years (N = 9)	3 (33.33%)	6 (66.67%)		

With a P-value of 0.9760, it is determined that it was statistically insignificant difference in preeclampsia rates between age groups, with the bulk of 18 individuals (26.47%) falling within the 20 to 25 age range. (Figure 8 and Table 3)

Figure 8: Age differences among preeclampsia in the study participants are shown in a cluster bar chart. (N=126)

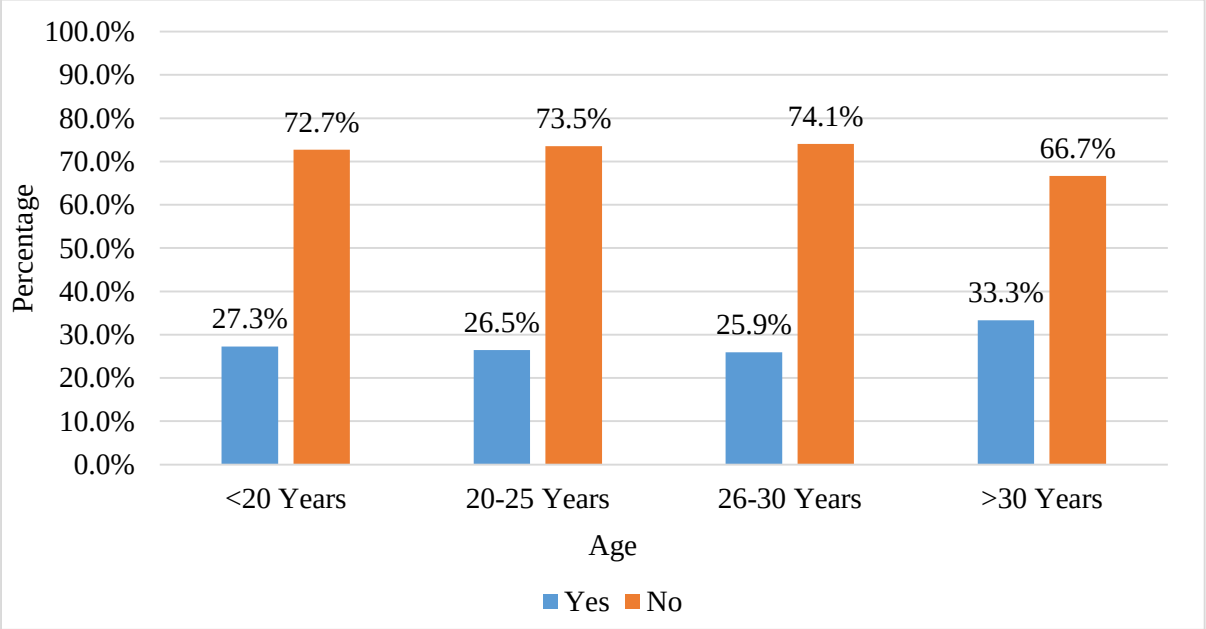


Table 4: Descriptive analysis of Parity in the study population (N=126)

Parity	Frequency	Percentage
PRIMI	89	70.63%
MULTI	37	29.37%
• G2	30	81.08%
• G3	7	18.92%

Among the study population, 89 (70.63%) participants were PRIMI and 37 (29.37%) were MULTI. (Table 4 & figure 9)

Figure 9: Pie Chart showing Population Parity in the Investigation (N=126)

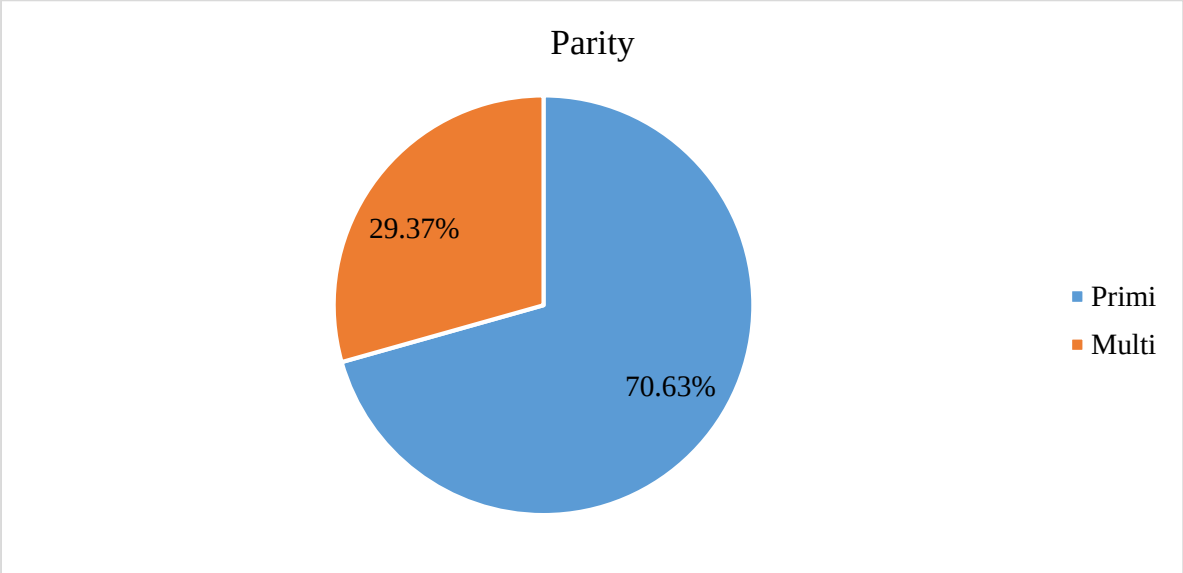


Table 5: Comparison of Parity between Placenta in the study population (N=126)

Parity	Placenta		Chi square value	P value
	Central	Lateral		
PRIMI (N = 89)	54 (60.67%)	35 (39.33%)	1.54	0.2141
MULTI (N = 37)	18 (48.65%)	19 (51.35%)		

The majority of 54 individuals (60.67%) had PRIMI parity in the central placenta, whereas 35 people (39.33%) had PRIMI parity in the lateral placenta, making the difference in placenta parity statistically irrelevant with a P-value of 0.2141. Among the Multigravida 18 of them (48.65%) had central placenta and 19(51.35%) of them had lateral placenta(Table 5 & figure 10)

Figure 10: Comparative cluster bar graph of placenta parity in the studied population (N=126)

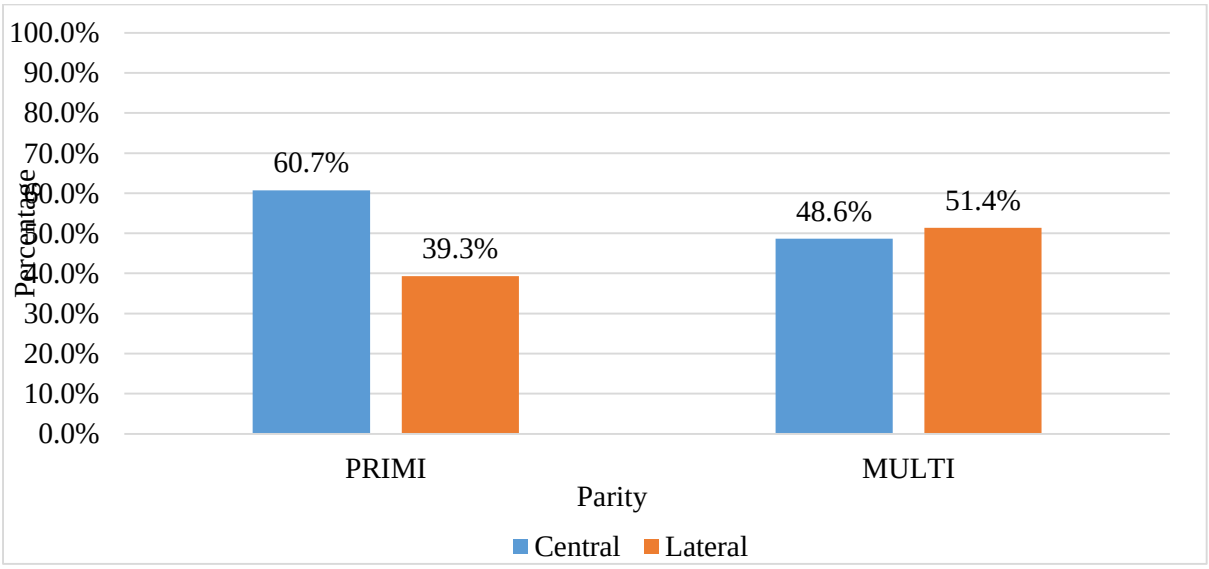


Table 6: Placenta descriptive analysis in the study sample (N=126)

Placenta	Frequency	Percentage
Central	72	57.14%
Lateral	54	42.86%

Among the study population, 72 (57.14%) Participants were Central Placenta location and 54 (42.86%) Participants were Lateral Placenta location. (Table 6 & figure 11)

Figure 11: Bar Chart of Placenta in the study population (N=126)

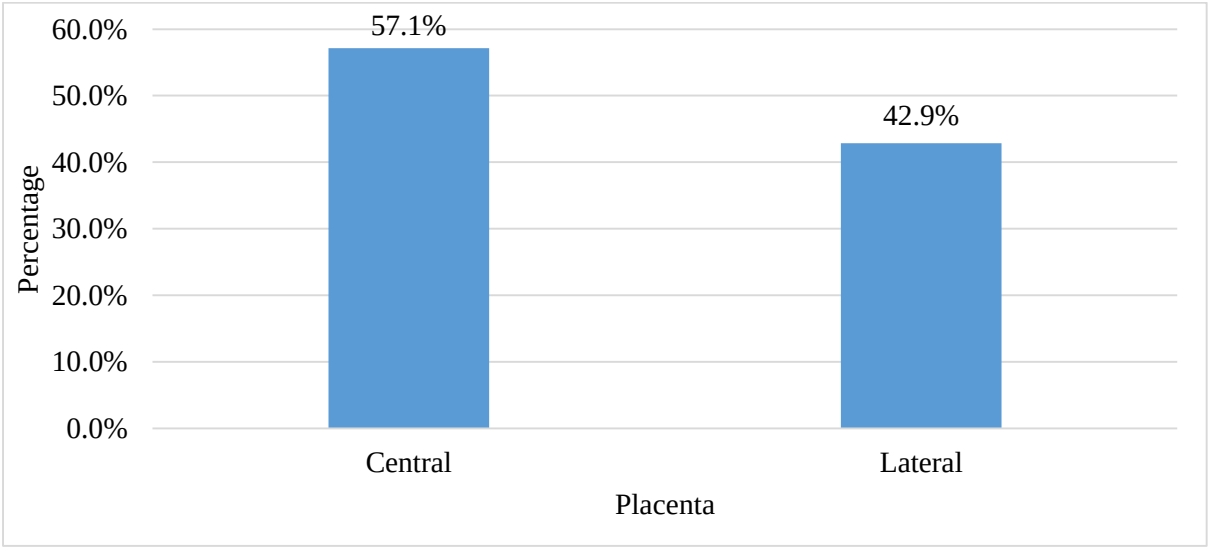


Table 7: Comparison of Parity between Preeclampsia in the study population (N=126)

Parity	Preeclampsia		Chi square value	P value
	Yes	No		
PRIMI (N = 89)	23 (25.84%)	66 (74.16%)	0.20	0.6544
MULTI (N = 37)	11 (29.73%)	26 (70.27%)		

With a P-value of 0.6544, it is determined that the difference in preeclampsia across parities is statistically insignificant. 23 patients (25.84%) of the PRIMI had lateral placenta and 66(74.16%) of the primigravida did not develop preeclampsia. Among the multigravida 11(29.73% of them developed preeclampsia and 26(70.27%) of them did not develop preeclampsia. (Figure 12 and Table 7)

Figure 12: Comparison of Preeclampsia Parity in the Study Participants in a Cluster Bar Chart (N=126)

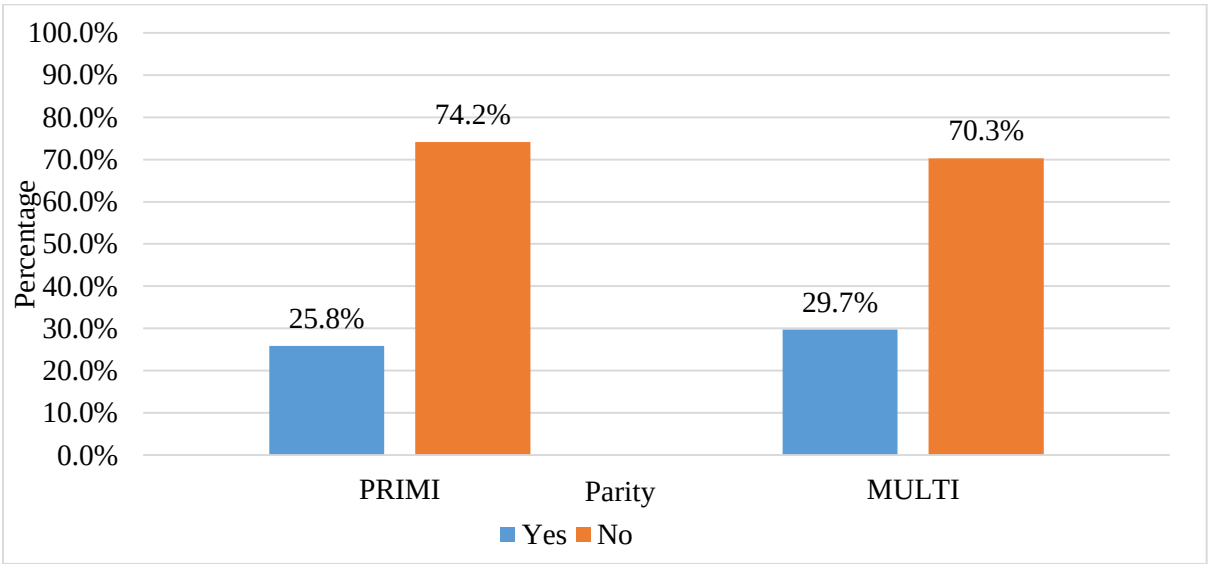


Table 8: Descriptive analysis of blood pressure parameters at different visits in the study group (N=126)

Parameter	Mean ± S. D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
1st visit						
SBP	112.06±5.98	110.00	100.00	130.00	111.02	113.11
DBP	74.68±5.47	70.00	60.00	80.00	73.73	75.64
2nd visit						
SBP	120.00±6.20	120.00	100.00	140.00	118.92	121.08
DBP	75.32±5.89	80.00	60.00	90.00	74.29	76.35
3rd visit						
SBP	129.27±16.00	130.00	100.00	170.00	126.48	132.06
DBP	83.56±12.79	80.00	60.00	110.00	81.32	85.79

The mean of 1st visit SBP was 112.06±5.98 (Range 100 to 130) and DBP was 74.68±5.47 (Range 60 to 80) in the study population. The mean of 2nd visit SBP was 120.00±6.20 (Range 100 to 140) and DBP was 75.32±5.89 (Range 60 to 90). The 3rd visit SBP was 129.27±16.00 (Range 100 to 170) and DBP was 83.56±12.79 (Range 60 to 110). (Table 8). There was a significant rise in the BP readings towards the 3rd visit.

Table 9: Comparison between Preeclampsia with Placenta in the study population (N=126)

Preeclampsia	Placenta		Chi square	P value
	Lateral (N=54)	Central (N=72)		
Yes	26 (48.15%)	8 (11.11%)	21.483	**<0.001**
No	28 (51.85%)	64 (88.89%)		

In Lateral Placenta location, 26 (48.15%) women had Preeclampsia and in Central Placenta location 8 (11.11%) had Preeclampsia. The difference in the Preeclampsia between Placenta was statistically significant with P value <0.001. (Table 9 & figure 13)

Figure 13: Cluster Bar chart of comparison of Preeclampsia between Placenta in the study population (N=126)

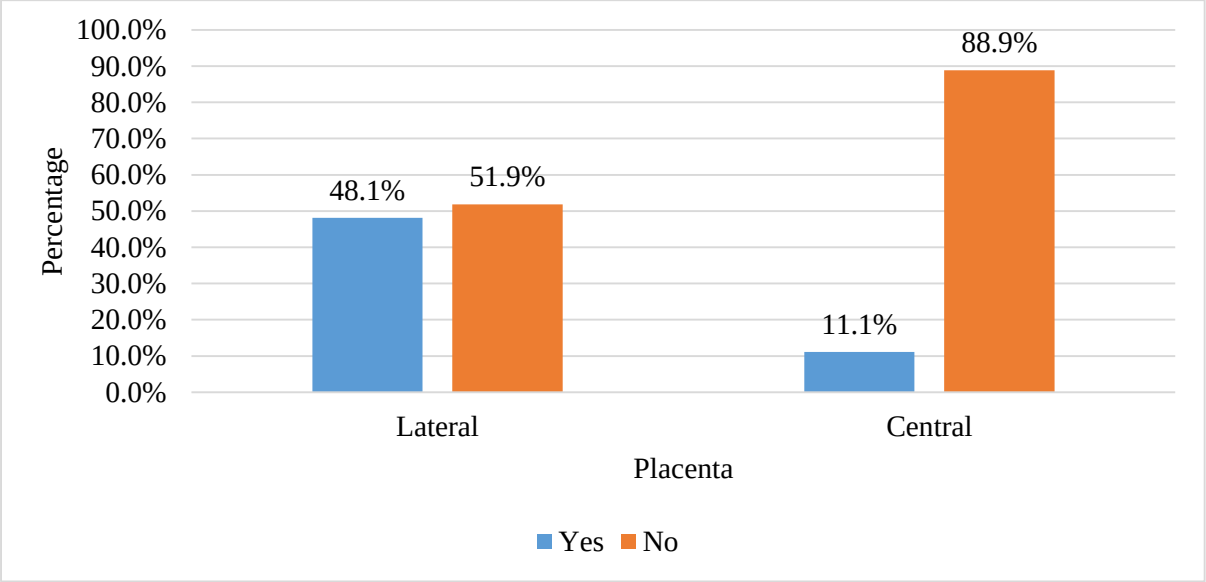


Table 10: Descriptive analysis of the study mode of delivery (N=126)

Mode of delivery	Frequency	Percentage
LSCS	55	43.65%
NVD	71	56.35%

Among the study population, the Number of women with Mode of delivery was LSCS in 55 (43.65%) and NVD in 71 (56.35%). (Table 9 & figure 13)

Figure 14: Bar Chart of Mode of delivery in the study group (N=126)

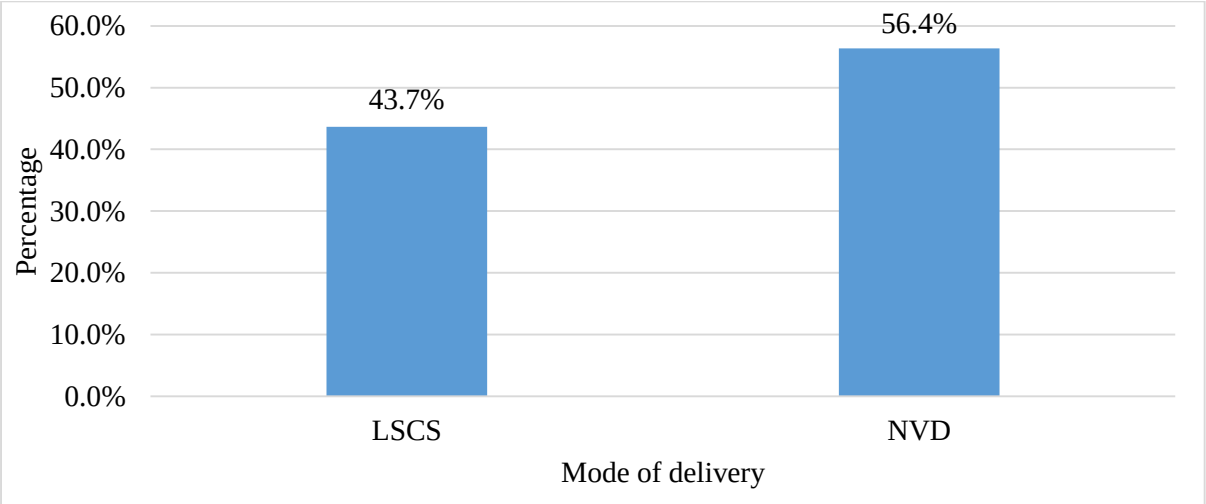


Table 11: Comparison of Mode of delivery between Preeclampsia in the study population (N=126)

Mode of delivery	Placenta		Chi square value	P value
	Central	Lateral		
LSCS (N = 55)	25 (45.45%)	30 (54.55%)	5.44	0.0196
NVD (N = 71)	47 (66.20%)	24 (33.80%)		

Out of 55 LSCS Mode of delivery, 25 (45.45%) women had Central Placenta location and 30 (54.55%) had Lateral Placenta location. Out of 71 NVD Mode of delivery, 47 (66.20%) women had Central Placenta location and 24 (33.80%) had Lateral Placenta location. With a P-value of 0.0196, it is determined that the placental difference across delivery methods is statistically relevant. (Table 11 & figure 15)

Figure 15: Preeclampsia in the study subjects is depicted in a cluster bar chart with the mode of delivery as a comparison. (N=126)

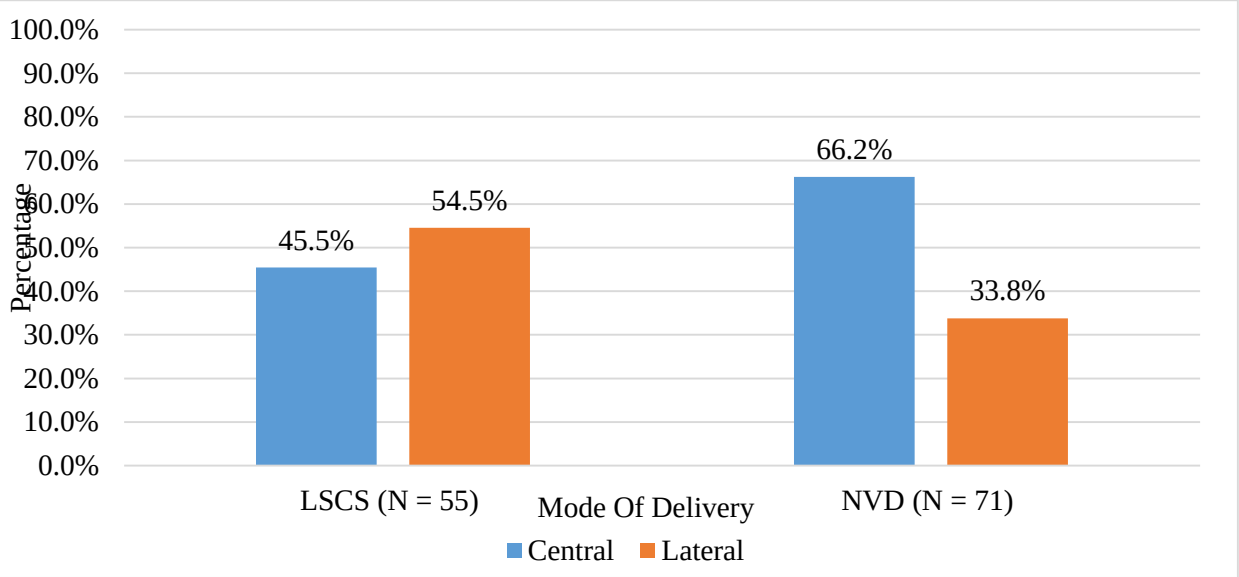


Table 12: Descriptive analysis of Preeclampsia in the study population (N=126)

Preeclampsia	Frequency	Percentage
Yes	34	26.98%
No	92	73.02%

Among the study population, the Number of women with Preeclampsia was 34 (26.98%). (Table 12& figure 16)

Figure 16: Preeclampsia in the sample group is shown as a pie chart. (N=126)

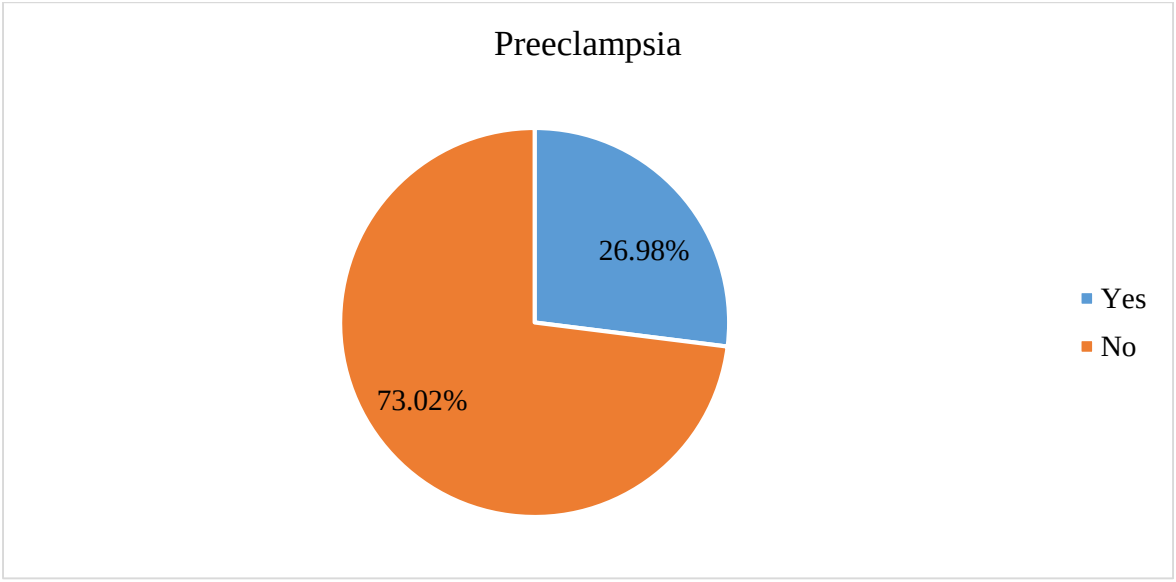


Table 13: Appearance of pre-eclampsia in weeks (N=34)

Gestational age in weeks	Frequency
26-28	1 (2.94%)
29-31	3(8.82%)
32-34	5 (14.7%)
35-37	11 (32.35%)
38-40	14 (41.19%)
>40	-

Among the pre-eclampsia cases, majority 14 (41.19%) were with 38-40 weeks of gestational age followed by 35-37 weeks with 11 (32.25%) and 32-34 weeks with 5(14.7%) cases. (Table 13)

Table 14: Distribution of patients according to severity of pre-eclampsia in the study samples (N=34)

Type of pre-eclampsia	Frequency
Mild- pre-eclampsia	30(88.24%)
Severe	4 (11.76%)

Among the study population, majority reported mild Preeclampsia with 30 (88.24%) and sever were 4(11.76%). Among the pre-eclampsia cases, majority 14 (41.19%) were with 38-40 weeks of gestational age followed by 35-37 weeks with 11 (32.25%) and 32-34 weeks with 5(14.7%) cases. (Table 14)

Table 15: Complications in the study subjects, described in detail (N=126)

Complications	Frequency	Percentage
Eclampsia		
Yes	6	4.76%
No	120	95.24%
IUGR		
Yes	11	8.73%
No	115	91.27%
Pprom		
Yes	2	1.59%
No	124	98.41%
DIC		
Yes	1	0.79%
No	125	99.21%
HELLP		
Yes	1	0.79%
No	125	99.21%
Abruptio		
Yes	2	1.59%
No	124	98.41%

Among the study population, 6 (4.76%) Participants had Eclampsia , 11 (8.73%) had IUGR, 2 (1.59%) had PPROM, 1 (0.79%) had DIC, 1 (0.79%) had HELLP and 2 (1.59%) had Abruptio complication. (Table 15)

Table 16: Comparison between Complications with Placenta in the study population (N=126)

Complications	Placenta		Chi square value	P value
	Central (N=72)	Lateral (N=54)		
Eclampsia				
Yes	2 (2.78%)	4 (7.41%)	1.46	0.4007*
No	70 (97.22%)	50 (92.59%)		
IUGR				
Yes	3 (4.17%)	8 (14.81%)	4.39	0.0539*
No	69 (95.83%)	46 (85.19%)		
Pprom				
Yes	0 (0.00%)	2 (3.70%)	-	†
No	72 (100.00%)	52 (96.30%)		
DIC				
Yes	0 (0.00%)	1 (1.85%)	-	†
No	72 (100.00%)	53 (98.15%)		
HELLP				
Yes	0 (0.00%)	1 (1.85%)	-	†
No	72 (100.00%)	53 (98.15%)		
Abruptio				
Yes	0 (0.00%)	2 (3.70%)	-	†
No	72 (100.00%)	52 (96.30%)		

=Chi Square P-Value; †= No statistical test applied due to 0 cells

Eclampsia was present in 2 (2.78%) participants in the central placenta and 4 (7.41%) participants in the lateral placenta. With a P value of 0.4007, the difference in eclampsia between placentas was not statistically significant. 3 (4.17%) participants at the central placenta position and 8 (14.81%) participants at the lateral placenta location both developed IUGR. With a P value of 0.0539, the IUGR difference between the placentas was statistically insignificant. Two (3.70%) individuals had Pprom in the lateral

placenta, one (1.85%) had DIC and HELLP, and two (3.70%) experienced abruptio complications. (Table 16)

Table 17: Descriptive analysis of Fetal outcome in the study group (N=126)

Fetal Outcome	Frequency	Percentages
IUD	2	1.59%
NICU	26	20.63%
Still birth	3	2.38%
No adverse fetal outcome	95	75.40%

In the study population, 2 (1.59%) participants had IUD, 26 (20.63%) had NICU, 3 (2.38%) had Still birth and 95 (75.40%) had no adverse fetal outcome. (Table 17& figure 17)

Figure 17: Bar Chart of Fetal outcome in the study population (N=31)

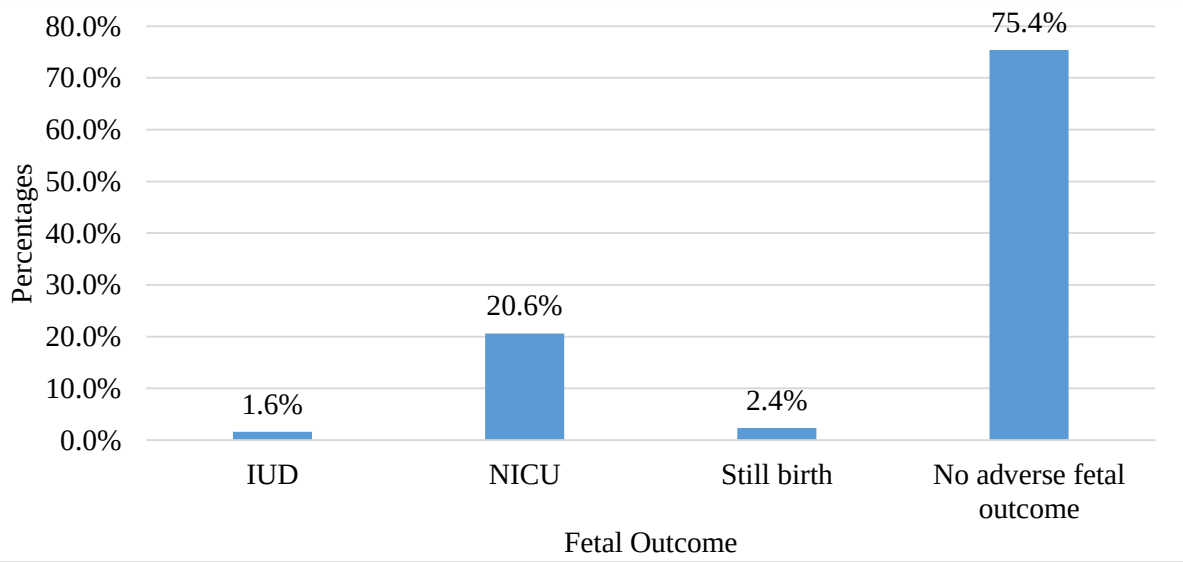


Table 18: Comparison of the study population's placenta and fetal outcome (N=126)

Fetal outcome	Placenta	
	Central (N=72)	Lateral (N=54)
IUD	0 (0%)	2 (3.7%)
NICU	9 (12.5%)	17 (31.48%)
Still birth	0 (0%)	3 (5.56%)
No adverse fetal outcome	63 (87.5%)	32 (59.26%)

No statistical test was applied due to 0 cells

In Central Placenta location, the majority of 63 (87.5%) were no adverse fetal outcome and 9 (12.5%) were NICU. In Lateral Placenta location, the majority of 32 (59.26%) were no adverse fetal outcome and 17 (31.48%) were NICU. (Table 18)

Table 19: Comparison of Placenta with Preeclampsia (N=126)

Parameter	Value	95% CI	
		Lower	Upper
Sensitivity	48.15%	34.34%	62.16%
Specificity	88.89%	79.28%	95.08%
False positive rate	11.11%	4.92%	20.72%
False negative rate	51.85%	37.84%	65.66%
Positive predictive value	76.47%	58.83%	89.25%
Negative predictive value	69.57%	59.10%	78.73%
Diagnostic accuracy	71.43%	62.70%	79.12%

The Placenta had sensitivity of 48.15% (95% CI 34.34% to 62.16%) in predicting Lateral Placenta Preeclampsia. Specificity was 88.89% (95% CI 79.28% to 95.08%), false positive rate was 11.11% (95% CI 4.92% to 20.72%), false negative rate was 51.85% (95% CI 37.84% to 65.66%), PPV was 76.47% (95% CI 58.83% to 89.25%), NPV was 69.57% (95% CI 59.10% to 78.73%), and the Total diagnostic accuracy was 71.43% (95% CI 62.70% to 79.12%) (Table 19)

DISCUSSION

DISCUSSION

The uterine arteries provide the majority of the maternal blood supply to the placenta. It is not distributed equally in laterally implanted placenta. Uterine artery resistance as measured by Doppler velocimetry is lower on the ipsilateral side compared to the contralateral in pregnancies with unilateral placentas. In the placenta's central location, the resistance is comparable on both sides. [56]

Pre-eclampsia is a multisystem syndrome that is peculiar to pregnancy and has an unidentified etiology. The pathogenesis of preeclampsia is heavily influenced by the placenta. The placenta's blood supply is not distributed equally. As a result, placental blood flow and subsequent placental positioning inside the uterus are likely crucial variables in determining pregnancy success. Pre-eclampsia cannot be predicted by a single screening test that is both reliable and affordable.[55] The current study intended to assess the ability to use placental localization might be used to indicate preeclampsia. In the current study was a prospective study with 126 subjects. The subjects were separated into two groups based on the placement of the placenta: lateral and central.

AGE

Majority (53.97%) of subjects aged between 20-25years followed by 21.43% aged between 26-30yrs, 17.46% ages less than 20yrs and 7.14% greater than 30yrs. Ambastha, V et al.[55] involved 250 subjects with majority (54%) aged between 21-25yrs, followed by 28.8% in 26-30yrs, 16-20yrs in 11.2% and greater than 30yrs in 6%. Similarly, Nandanwar, R et al[11] study, involving 900 women, found majority of 73% were aged between 21-30 yrs of age. Literature has found that maternal age increases the risk of developing preeclampsia is very high due to various comorbid conditions associated with age[70,71] As we found majority of the subjects belonged to age between

20-25yrs, the association of preeclampsia was found to be statistically unimportant across the age groups.

Parity

Most of the subjects were primiparity (70.63% n=89) and multi was 29.37% (n=37). Central location of placenta was significant more among the primiparity (60.67%) and lateral placental position among primi was found in 39.33%. In both primi and multi the pre-eclampsia was found to be insignificant as both the found similar incidence of pre- eclampsia (25.84% and 29.73% respectively). The difference in Preeclampsia between Parity was found to be insignificant, with the majority of 23 (25.84%) subjects having PRIMI Parity in the Lateral Placenta region. In Gupta, Anjali et al.[5] study the primiparity was found in 21.6% of the subjects with central location of placenta and 21.4% with lateral location, multiparity was found in 5.2% with central location and 14.3% with lateral location and the variation in parity between groups was numerically inconsequential.

Blood pressure

The mean of 1st visit SBP was 112.06±5.98 (Range 100 to 130) and DBP was 74.68±5.47 (Range 60 to 80) in the study population. The mean of 2nd visit SBP was 120.00±6.20 (Range 100 to 140) and DBP was 75.32±5.89 (Range 60 to 90). The 3rd visit SBP was 129.27±16.00 (Range 100 to 170) and DBP was 83.56±12.79 (Range 60 to 110). Hence, we found that, as that the blood pressure increased gradually from the early trimesters to later trimester of pregnancy.

Position of placenta and the outcomes.

The placenta was centrally located in 57.14% of the study population and laterally located in 42.86%. Normal vaginal delivery was found in 56.35% and C-section in 43.65% of women. Among the C-section mode of delivery (n=55), nearly 54.55% of

them had lateral placental location and 45.45% had central placental position. Among the normal vaginal delivery (n=71), majority 66.20% had central location of placenta and only 33.80% had lateral location of placenta. Hence, the study found lateral position of placenta significantly greater in the LSCS mode of delivery and this could be one of the reason for C-section. Similarly in a prospective study Ambastha, V et al.[55] found the lateral position of placenta was found in 59.2% and central was found in 40.8%. In 37% of subjects had C-section for lateral placenta delivery. In another study by Yousuf et al[12] involving 201 subjects found majority (64%) having central location and 36% with lateral location and in a study conducted in south India by Rajeshwary Pillay et al[66] in 100 subjects found 68% with central placenta and 32% has lateral location. Nair, V et al[72] found central in 83.% and lateral in 16.2%.

In the Sumathi et al.[8] study, 52 of 120 women with pre-eclampsia underwent vaginal birth, whereas 68 needed a caesarean section, with 59 (86.8%) presenting a lateral placenta.

Table 20: Comparing the position of placenta among the study population

	Central	**Lateral**
Nair, V et al[72]	**83.8%**	**16.2%**
Ambastha, V et al.[55]	40.8%	59.2%
Yousuf et al[12]	64%	36%
Rajeshwary Pillay et al[66]	68%	32%
Present study	57.14%	42.86%

Table 21: Comparing the position of the placenta to mode of delivery among the study population across various studies to present study

Ambastha, V et al.[55]	**Central**	**Lateral**	**P- value**
LSCS	(37.2%)	53.9%	<0.001 significant
NVD	62.8%	46.1%	
Present study			
LSCS	54.55%	45.45%	<0.001 significant
Central	66.20%	33.80%	

Gestational age and preeclampsia

In the current study we found majority of the preeclampsia in gestational weeks between 38 to 40 wks (41.19%) followed by 35-37 wks (32.25%), 32-34 wks (14.7%). Hence our study results found that as the gestational age progressed the risk of developing preeclampsia was greater. Kumasawa, K et al,[73] study also found that as the gestational age increased the occurrence of preeclampsia increased

Complications and placental location

Among the study population, 6 (4.76%) Participants had Eclampsia Complications, 11 (8.73%) had IUGR, 2 (1.59%) had Pprom, 1 (0.79%) had DIC, 1 (0.79%) had HELLP and 2 (1.59%) had Abruptio complication. Majority of the study population had mild preeclampsia (88.24%) and severe was observed only in 11.76%.

Occurrence of preeclampsia was seen in 26.98% of our study population. Among the subjects with lateral placental location, 48.15% had incidence of preeclampsia and in subjects with central placental location only 11.11% had preeclampsia. As an outcome, there was a substantial increase in the incidence of preeclampsia in subjects with lateral placental position (P value 0.001). According to Kakker et al.[65], out of 150 women, 84

(56%) had placentas that were positioned laterally, and of those, 56 (66.6%) experienced preeclampsia (p0.00002). In Ambastha, V et al. [55] study the the prevalence of total 39 patients develop preeclampsia, 33 (84.62%) of the subjects had a lateral placenta, whereas only 6 (15.38%) had a central placenta. 25 subjects had IUGR, with 17 (68%) having lateral placentas and only 8 (32% having central placentas). There were 19 individuals who developed gestational hypertension, with 12 (63%) having a lateral placenta and 9 (37%) having a central placenta. In a total of 250 individuals, 9 had PIH +IUGR, with 7 having lateral placentas and 2 having central placentas. In all, 250 patients were admitted to the NICU, with 37 newborns having lateral placentas and 24 having central placentas. In a total of 250 subjects, 48 had pre-eclampsia, with only 8 women having a central placenta and the remaining 40 women having a lateral placenta. [55]

Our results are consistent with those of Fung et al.[74], who did a retrospective research on 16236 subjects who had ultrasounds between 14 and 23 weeks of pregnancy. Preeclampsia was one of the adverse obstetric outcomes that was associated with non-central placental position in the second trimester [OR = 2.27; 95% CIs, 1.31-3.93]. Preeclampsia was also considerably higher in women with lateral placental placements, (4.5 vs. 1.6%; p = 0.027) according to Secken et al.[75]

In Central Placenta location, 2.78% participants had pre-eclampsia and in Lateral Placenta location, 7.41% had preeclampsia. Though the complication of pre-eclampsia was more among the lateral placenta, the difference in preeclampsia between Placenta was numerically minor. According to Singh et al.[76], with OR 2.578, lateral placentation raised the incidence of preeclampsia by 62.9%. In a prospective study involving 900 patients, Nandanwar et al.[11] discovered that lateral placental locations had a significant p value of 0.0001 and a pregnancy-induced hypertension outcome of 66.4%.

In our study Central Placenta location, 4.17% participants had IUGR and in Lateral Placenta location, 14.81% had IUGR. With a P value of 0.0539, the variation in IUGR between Placenta was numerically minor. Likewise, Ambastha et al. [55] and Kore et al.[10] also showed that women with lateral placentas were more likely to have IUGR babies. Furthermore, the present study found wiLateral Placenta location, 3.70% participants had Pprom, 1.85% had DIC and HELLP, 3.70% had Abruptio Complications. Similar observation was made in Gupta, Anjali et al.[5] study discovered that females with lateral placentas had a greater risk of all problems (early birth, eclampsia, HELLP syndrome, IUGR newborns, and transfer to NICU/ICU), although only pre-eclampsia and IUGR were numerically notable.

The inability of a foetus to fulfil the growth potential promised by the hereditary makeup and endogenous pregnancy variables is referred to as IUGR. Fetal development is determined by the combination of epigenetic and genetic variables acting against a background of maternal, foetal, and placental effects. IUGR endangers the foetus and the child or renders them helpless during the potential stage. The degree of vascular supply in women with a lateral placental may differ from that found in all women, and a lack of input encourages the development of IUGR. In women with a centrally located placental, both uterine arteries exhibit similar resistance. [55] Nonetheless, our investigation had substantial outcomes, studies[11,66,72] have shown significant association of laterality of placenta with IUGR.

Fetal outcome

Overall, the fetal outcome found 1.59% had IUD, 20.63% had NICU and 2.38 % had Still birth. In Central Placenta location, only 12.5% were in NICU, and among lateral, 31.48% were in NICU**.** The number of lateral placentas with NICU admissions in Nair,

V et al [72] study , was 19, and there were 62 (16.4%) central placentas with NICU hospitalizations. However, 315 central placentas and 54 (74% of lateral placentas) did not require NICU care. The results fulfilled a merely noticeable p value of 0.05. Major NICU admissions were caused by the lateral placenta. [72] According to Singh et al.[76], 16% of NICU admissions are at major risk for IUGR and preeclampsia, both of which are related to lateral placentation. The findings of Devarajan et al.[69], revealed a 5.3% lateral placenta and a 6% central placenta with NICU admission. Similarly, when compared to our research, Zia et al[77], Jaisal et al[78] identified no link between placental location and NICU admissions

Predictive value

The Placenta had sensitivity of 48.15%, specificity was 88.89%, false positive rate was 11.11% false negative rate was 51.85%, PPV was 76.47%, NPV was 69.57%, and the total diagnostic accuracy was 71.43% in predicting Lateral Placenta Preeclampsia. In Ambastha, V et al. [55] study, found sensitivity 83.33%, specificity was 69.31%, positive predictive value was 39.22% and negative predictive value was 94.59%. Pai Muralidhar V et al[79] evaluated 426 pregnant women in total, 71 of whom progressed to PE, and 74% of whom had unilaterally identified placentas. The corresponding sensitivity, specificity, PDV, and NDV values were 73%, 86%, 51%, and 94%, respectively. The statistical significance of this study was high (p value >0.001)

Table 22: Comparing the predictive value of placental position across various studies to present study

	Ambastha, V et al. [55]	**Pai Muralidhar V et al**[79]	**Present study**
Sensitivity	83.33%	**73%**	48.15%
Specificity	69.31%	**86%**	88.89%
positive predictive value	39.22%	51%	76.47%
Negative predictive value	94.59%	94%	69.57%
total diagnostic accuracy	-----	------	71.43%

Preeclampsia developed in 14 (35%) of the 40 subjects with lateral placentas, according to Alpesh et al.[9] found that color Doppler scans of 13 of the 40 people with lateral placentas revealed significant uterine artery resistance indices. They arrived at the conclusion that preeclampsia may be detected rather reliably in all people with lateral placenta and that lateral placenta may be utilised as an indicator of preeclampsia.[9]

CONCLUSION

With a sensitivity of 48.15 percent, specificity of 88.89 percent, and PPV of 76.47%, the current study indicated that lateral placental position assessed by ultrasonography around 18 and 24 weeks of pregnancy was an excellent screening technique for predicting preeclampsia.

Lateral position of placenta helps in identifying the risk or incidence of preeclampsia. Similarly, the neonatal outcome such as admission to NICU and IUGR, intrauterine death and still birth were identified with lateral position of placenta. In addition, complications such as PPROM, DIC and HELLP, Abruptio was related with lateral position of placenta.

Limitations and recommendations

1 Because this was hospital-based research with a limited sample size, the results cannot be generalized.

2 The placental location of anterior or posterior was not studied

SUMMARY

The current prospective observational study involved 126 subjects with majority of them aged between 20-25 yrs. Most of the subjects were primiparity (70.63% n=89) and multi was 29.37% (n=37). Central location of placenta was significant more among the primiparity (60.67%) and lateral placental position among primi was found in 39.33%. In both primi and multi the pre-eclampsia was found to be insignificant as both the found similar incidence of pre- eclampsia (25.84% and 29.73% respectively). Normal vaginal delivery was found in 56.35% and C-section in 43.65% of women. It was observed that lateral position of placenta significantly was greater in the LSCS compared to normal vaginal mode of delivery. Preeclampsia affected 26.98% of the women in the study group. Among the subjects with lateral placental location, 48.15% had incidence of preeclampsia and in subjects with central placental location only 11.11% had preeclampsia. As a result, there was a substantial rise in the frequency of preeclampsia in subjects with lateral position of placenta (P value <0.001). In Central Placenta location, 2.78% participants had preeclampsia and in Lateral Placenta location, 7.41% had preeclampsia. Though the complication of preeclampsia was more among the lateral placenta, the difference in preeclampsia between Placenta was numerically minor.

Majority of the preeclampsia in gestational weeks between 38 to 40 wks (41.19%) followed by 35-37 wks (32.25%), 32-34 wks (14.7%). Majority of the study population had mild preeclampsia (88.24%) and severe was observed only in 11.76%. Overall, the fetal outcome found 1.59% had IUD, 20.63% had NICU and 2.38 % had Still birth. In Central Placenta location, only 12.5% were in NICU, and among lateral, 31.48% were in NICU. The variation in IUGR between Placenta was numerically inconsequential. In Lateral Placenta location, 3.70% participants had PPROM, 1.85% had DIC and

HELLP, 3.70% had Abruptio The Placenta had sensitivity of 48.15%, Specificity was 88.89% and total diagnostic accuracy was 71.43% in predicting Lateral Placenta Preeclampsia.

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ANNEXURES

PROFORMA

NAME:

AGE:

ADDRESS:

UHID NO:

I.P NO:

DATE/ TIME OF ADMISSION:

DATE/ TIME OF DISCHARGE:

CHIEF COMPLAINTS:

OBSTETRICAL HISTORY: Booked/ Unbooked/ Referred

Married Life: Consanguinesous marriage: Yes/ No

Obstetrical Score:

MENSTRUAL HISTORY:

LMP: EDD:

POG:

cEDD:

PAST HISTORY:

PERSONAL HISTORY:

Diet:

Appetite:

Bowel and bladder habits:

Smoking/ Alcohol:

GENERAL PHYSICAL EXAMINATION:

Pallor/ Icterus/ Cyanosis/ Clubbing/ Lymphadenopathy/ Edema

Height:	Weight:	BMI:
Pulse:	BP:	
RR:	Temp:	

CNS:

CVS:

RS:

PER ABDOMEN:

BREAST:

SPINE:

THYROID

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS

Blood investigations

1. CBC
2. LFT
3. RFT
4. URINE ALBUMIN
5. URIC ACID
6. LDH
7. ULTRASONOGRAPHY OF PELVIS

INFORMED CONSENT FORM

I Mr./Mrs. _____ have been explained in my own understandable language, that I will be included in a study which is **"EVALUATION OF LATERAL IMPLANTATION OF PLACENTA AND ITS ASSOCIATION WITH DEVELOPMENT OF PREECLAMPSIA AND ITS OUTCOME. "**

I have been explained that my clinical findings, investigations, postoperative findings will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the interventions needed possible benefits and adversities due to interventions, in my own understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I have principal investigator mobile number for enquiries.

I in my sound mind give full consent to be added in the part of this study.

Signature of the patient:

Name:

Signature of the witness:

Name:

Relation to patient:

Date:

Place:

ಮಾಹಿತಿ ಕಾನ್ಸೆಂಟ್ ಫಾರ್ಮ್

ನಾನು ಶ್ರೀ / ಶ್ರೀ. _____ ಅನ್ನು ನನ್ನದೇ ಆದ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಇದು "ಜರಾಯುವಿನ ಪಾರ್ಶ್ವ ಅಳವಡಿಕೆಯ ಮೌಲ್ಯಮಾಪನ ಮತ್ತು ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯ ಅಭಿವೃದ್ಧಿ ಮತ್ತು ಅದರ ಫಲಿತಾಂಶದೊಂದಿಗೆ ಅದರ ಸಂಬಂಧ."

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಆವಿಷ್ಕಾರಗಳು, ತನಿಖೆಗಳು, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ ಸಂಶೋಧನೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದು ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ, ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಗೆ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ.

ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ, ಮಧ್ಯಸ್ಥಿಕೆಗಳ ಕಾರಣದಿಂದಾಗಿ ಸಂಭವನೀಯ ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಪ್ರತಿಕೂಲತೆಗಳ ಅಗತ್ಯವಿರುವ ಮಧ್ಯಸ್ಥಿಕೆಗಳ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಕಂಡುಬರುವ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನ ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ವಿಚಾರಣೆಗಾಗಿ ನನ್ನ ಬಳಿ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ ಇದೆ.

ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ನನ್ನ ಸಂಪೂರ್ಣ ಮನಸ್ಸಿನಲ್ಲಿ ನಾನು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ:

ಹೆಸರು:

ಸಾಕ್ಷಿಯ ಸಹಿ:

ಹೆಸರು:

ರೋಗಿಗೆ ಸಂಬಂಧ:

ದಿನಾಂಕ:

ಸ್ಥಳ:

PATIENT INFORMATION SHEET

STUDY TITLE: "EVALUATION OF LATERAL IMPLANTATION OF PLACENTA AND ITS ASSOCIATION WITH DEVELOPMENT OF PREECLAMPSIA AND ITS OUTCOME."

STUDY SITE: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

This is to inform you that, you require routine blood investigations and ultrasonography of pelvis for making treatment plan for you condition that is preeclampsia. The routine blood investigations and ultrasonography of pelvis is required for the making the diagnosis of the disease extent of the disease and for planning of the treatment. The entire finance of the tests will be of your regular treatment.

We are conducting this study to predict the placenta laterality and its association with the development of preeclampsia and its outcome. If you are willing you will be enrolled in this study and we will do routine blood investigations and ultrasonography of pelvis which are required to predict the placenta laterality and its association with the development of preeclampsia and its outcome. You will receive the standard care pre and post operatively

This study will help better understand preeclampsia, help in reducing the associated perinatal morbidity and mortality. It will also benefit other patients with preeclampsia undergoing treatment in future. You are free to opt-out of the study at any time if you

are not satisfied or apprehensive to be a part of the study. Your treatment and care will not be compromised if you refuse to be a part of the study. The study will not add any risk or financial burden to you if you are part of the study. In case of any complication during surgery patient will be treated accordingly.

Your identity and clinical details will be confidential. You will not receive any financial benefit for being part of the study. You are free to contact Dr. RAVEENA K S or any other member of the above research team for any doubt or clarification you have.

Dr. RAVEENA K S

Mobile no: 9566118673

E-mail id: raveenakethineni95@gmail.com

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ‘‘ಜರಾಯುವಿನ ಪಾರ್ಶ್ವ ಅಳವಡಿಕೆಯ ಮೌಲ್ಯಮಾಪನ ಮತ್ತು ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯ ಅಭಿವೃದ್ಧಿ ಮತ್ತು ಅದರ ಫಲಿತಾಂಶದೊಂದಿಗೆ ಅದರ ಸಂಬಂಧ’’.

ಅಧ್ಯಯನ ಸೈಟ್: ಆರ್.ಎಲ್ ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ತಮಾಕಾ, ಕೋಲಾರ.

ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯಾದ ನಿಮ್ಮ ಸ್ಥಿತಿಗೆ ಚಿಕಿತ್ಸೆಯ ಯೋಜನೆಯನ್ನು ತಯಾರಿಸಲು ನಿಮಗೆ ದಿನನಿತ್ಯದ ರಕ್ತ ತನಿಖೆ ಮತ್ತು ಸೊಂಟದ ಅಲ್ಟ್ರಾಸೊನೋಗ್ರಫಿ ಅಗತ್ಯವಿರುತ್ತದೆ ಎಂದು ನಿಮಗೆ ತಿಳಿಸುವುದು. ರೋಗದ ವ್ಯಾಪ್ತಿಯ ರೋಗನಿರ್ಣಯವನ್ನು ಮಾಡಲು ಮತ್ತು ಚಿಕಿತ್ಸೆಯ ಯೋಜನೆಗಾಗಿ ವಾಡಿಕೆಯ ರಕ್ತ ತನಿಖೆ ಮತ್ತು ಸೊಂಟದ ಅಲ್ಟ್ರಾಸೊನೋಗ್ರಫಿ ಅಗತ್ಯವಿದೆ. ಪರೀಕ್ಷೆಗಳ ಸಂಪೂರ್ಣ ಹಣಕಾಸು ನಿಮ್ಮ ನಿಯಮಿತ ಚಿಕಿತ್ಸೆಯಾಗಿರುತ್ತದೆ.

ಜರಾಯು ಪಾರ್ಶ್ವತೆ ಮತ್ತು ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯ ಬೆಳವಣಿಗೆ ಮತ್ತು ಅದರ ಫಲಿತಾಂಶದೊಂದಿಗೆ ಅದರ ಸಂಬಂಧವನ್ನು to ಹಿಸಲು ನಾವು ಈ ಅಧ್ಯಯನವನ್ನು ನಡೆಸುತ್ತಿದ್ದೇವೆ. ನೀವು ಸಿದ್ಧರಿದ್ದರೆ ನೀವು ಈ ಅಧ್ಯಯನಕ್ಕೆ ದಾಖಲಾಗುತ್ತೀರಿ ಮತ್ತು ನಾವು ವಾಡಿಕೆಯ ರಕ್ತ ತನಿಖೆ ಮತ್ತು ಸೊಂಟದ ಅಲ್ಟ್ರಾಸೊನೋಗ್ರಫಿಯನ್ನು ಮಾಡುತ್ತೇವೆ, ಇದು ಜರಾಯು ಪಾರ್ಶ್ವತೆ ಮತ್ತು ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯ ಅಭಿವೃದ್ಧಿ ಮತ್ತು

ಅದರ ಫಲಿತಾಂಶದೊಂದಿಗೆ ಅದರ ಸಂಬಂಧವನ್ನು to ಹಿಸಲು ಅಗತ್ಯವಾಗಿರುತ್ತದೆ. ನೀವು ಸ್ಟ್ಯಾಂಡರ್ಡ್ ಕೇರ್ ಪೂರ್ವ ಮತ್ತು ಪೋಸ್ಟ್ ಅನ್ನು ಆಪರೇಟಿವ್ ಆಗಿ ಸ್ವೀಕರಿಸುತ್ತೀರಿ

ಈ ಅಧ್ಯಯನವು ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯಾವನ್ನು ಚೆನ್ನಾಗಿ ಅರ್ಥಮಾಡಿಕೊಳ್ಳಲು ಸಹಾಯ ಮಾಡುತ್ತದೆ, ಸಂಬಂಧಿತ ಪೆರಿನಾಟಲ್ ಕಾಯಿಲೆ ಮತ್ತು ಮರಣವನ್ನು ಕಡಿಮೆ ಮಾಡಲು ಸಹಾಯ ಮಾಡುತ್ತದೆ. ಭವಿಷ್ಯದಲ್ಲಿ ಚಿಕಿತ್ಸೆಗೆ ಒಳಪಡುವ ಪ್ರಿಕ್ಲಾಂಪ್ಸಿಯಾದ ಇತರ ರೋಗಿಗಳಿಗೆ ಇದು ಪ್ರಯೋಜನವನ್ನು ನೀಡುತ್ತದೆ. ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ತೃಪ್ತಿ ಹೊಂದಿಲ್ಲದಿದ್ದರೆ ಅಥವಾ ಭಯಪಡದಿದ್ದರೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹೊರಗುಳಿಯಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ನಿರಾಕರಿಸಿದರೆ ನಿಮ್ಮ ಚಿಕಿತ್ಸೆ ಮತ್ತು ಕಾಳಜಿಗೆ ಧಕ್ಕೆಯಾಗುವುದಿಲ್ಲ. ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಿದ್ದರೆ ಅಧ್ಯಯನವು ನಿಮಗೆ ಯಾವುದೇ ಅಪಾಯ ಅಥವಾ ಆರ್ಥಿಕ ಹೊರೆ ಸೇರಿಸುವುದಿಲ್ಲ. ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ತೊಡಕು ಉಂಟಾದರೆ ರೋಗಿಗೆ ಅನುಗುಣವಾಗಿ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ.

ನಿಮ್ಮ ಗುರುತು ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ವಿವರಗಳು ಗೌಪ್ಯವಾಗಿರುತ್ತದೆ. ಅಧ್ಯಯನದ ಭಾಗವಾಗಿರುವುದರಿಂದ ನೀವು ಯಾವುದೇ ಆರ್ಥಿಕ ಲಾಭವನ್ನು ಪಡೆಯುವುದಿಲ್ಲ. ನೀವು ಹೊಂದಿರುವ ಯಾವುದೇ ಅನುಮಾನ ಅಥವಾ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಡಾ. ರವೀನಾ ಕೆ ಎಸ್ ಅಥವಾ ಮೇಲಿನ ಸಂಶೋಧನಾ ತಂಡದ ಯಾವುದೇ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ.

ಡಾ.ರವೀನಾ ಕೆ ಎಸ್

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MASTER CHART

Sr. No	Age	Gravida	Parity	Live births	Abortion	Placenta	1st visit SBP	1st visit DBP	2nd visit SBP	2nd visit DBP	3rd visit SBP	3rd visit DBP	Mode of delivery	Preeclampsia	Type of	Eclampsia	IUGR	Pprom	DIC	HELLP	Abruptio	Fetal outcome
1	2	1				1	120	80	130	80	126	70	1	2	3	2	2	2	2	2	2	2
2	2	2	P1	L1		1	110	70	120	70	110	70	2	2	3	2	2	2	2	2	2	4
3	2	3	P1	L1	A1	2	110	70	120	80	140	100	2	1	1	2	2	2	2	2	2	2
4	2	2	P1	L1		2	120	80	130	70	130	90	2	2	3	2	1	2	2	2	2	4
5	2	1				1	110	70	120	80	130	80	1	2	3	2	2	2	2	2	2	2
6	2	1				2	110	70	120	80	150	100	1	1	1	2	2	2	2	2	2	4
7	1	1				1	110	70	120	80	100	70	2	2	3	2	2	2	2	2	2	4
8	3	1				2	100	70	120	70	110	80	2	2	3	2	2	1	2	2	2	2
9	2	1				2	110	80	110	80	126	84	1	2	3	2	1	2	2	2	2	4
10	4	1				1	120	80	120	90	150	100	1	1	1	2	2	2	2	2	2	4
11	2	2	P1	L1		2	110	80	120	80	120	80	2	2	3	2	2	2	2	2	2	4
12	2	1				1	110	70	120	80	110	80	1	2	3	2	2	2	2	2	2	4

13	2	1				2	120	80	120	90	160	100	1	1	1	2	2	2	2	2	2	2
14	2	1				1	110	70	120	80	110	80	2	2	3	2	2	2	2	2	2	4
15	1	1				1	110	70	140	80	160	110	1	1	2	1	2	2	2	2	2	4
16	2	1				2	110	70	120	80	120	80	2	2	3	2	2	2	2	2	2	4
17	4	1				2	110	80	120	80	120	84	1	2	3	2	1	2	2	2	2	4
18	3	1				1	120	80	110	70	120	80	2	2	3	2	2	2	2	2	2	2
19	2	1				1	120	80	110	70	140	100	1	1	1	2	2	2	2	2	2	4
20	2	3	P1	L1	A1	2	110	80	120	70	120	80	2	2	3	2	2	2	2	2	2	4
21	2	1				2	110	80	120	80	110	70	1	2	3	2	1	2	2	2	2	2
22	1	1				1	110	80	120	70	110	80	2	2	3	2	2	2	2	2	2	4
23	2	1				2	120	80	130	80	150	100	1	1	1	2	2	2	2	2	2	4
24	2	1				1	110	70	120	70	120	80	2	2	3	2	2	2	2	2	2	4
25	3	2	P1	L1		1	120	80	110	70	120	70	1	2	3	2	2	2	2	2	2	4
26	4	1				2	110	70	120	80	120	70	2	2	3	2	1	2	2	2	2	4
27	2	1				1	110	70	120	80	140	90	1	2	3	2	2	2	2	2	2	4
28	2	1				2	110	70	120	80	130	80	2	2	3	2	2	2	2	2	2	4

29	2	1				1	110	70	120	80	120	80	2	2	3	2	2	2	2	2	2	4
30	2	1				1	110	70	120	70	120	80	1	2	3	2	2	2	2	2	2	4
31	2	1				1	120	80	110	70	150	110	2	1	1	1	2	2	2	2	2	2
32	2	1				2	110	70	120	80	110	70	1	2	3	2	1	2	2	2	2	2
33	1	1				1	110	80	120	80	140	100	1	1	1	2	2	2	2	2	2	4
34	2	2	P1	L1		2	110	80	120	80	110	70	1	2	3	2	2	2	2	2	2	4
35	3	1				1	130	80	120	80	126	80	2	2	3	2	2	2	2	2	2	4
36	2	1				2	110	70	120	80	150	100	1	1	1	2	2	2	2	2	2	2
37	1	1				1	110	70	120	80	140	100	2	1	1	2	2	2	2	2	2	2
38	2	1				2	100	70	110	60	100	70	1	2	3	2	1	2	2	2	2	4
39	3	1				1	110	80	120	70	100	70	1	2	3	2	2	2	2	2	2	4
40	4	1				2	100	60	100	60	100	60	1	2	3	2	1	2	2	2	2	4
41	2	1				1	100	70	110	70	120	80	2	2	3	2	2	2	2	2	2	4
42	2	1				2	110	80	120	80	140	100	2	1	1	2	2	2	2	2	2	2
43	1	1				1	110	80	120	80	120	80	1	2	3	2	2	2	2	2	2	4
44	2	2	P1	L1		2	100	80	110	80	110	80	1	2	3	2	2	2	2	2	2	4

45	3	1				1	120	80	130	80	140	100	2	1	1	2	2	2	2	2	2	2
46	2	1				2	110	70	120	80	120	80	2	2	3	2	2	2	2	2	2	4
47	2	1				1	110	70	120	80	110	70	1	2	3	2	2	2	2	2	2	4
48	2	1				1	110	70	120	70	120	80	2	2	3	2	2	2	2	2	2	4
49	2	2	P1	L1		1	110	70	120	80	110	70	1	2	3	2	2	2	2	2	2	2
50	4	2	P1	L1		2	110	70	130	80	150	100	1	1	1	2	2	2	2	2	2	2
51	2	2	P1	L1		1	110	70	120	70	110	70	2	2	3	2	2	2	2	2	2	4
52	2	1				2	110	70	120	80	160	100	1	1	1	2	2	2	2	1	2	4
53	1	1				1	120	70	110	60	120	80	1	2	3	2	2	2	2	2	2	4
54	2	2	P1	L1		2	110	80	120	80	140	90	2	2	3	2	2	2	2	2	2	4
55	3	1				1	110	80	120	80	150	100	1	1	1	2	2	2	2	2	2	4
56	1	1				2	100	70	100	60	110	70	1	2	3	2	2	2	2	2	2	4
57	2	3	P1	L1	A1	1	100	70	110	70	120	70	2	2	3	2	2	2	2	2	2	4
58	3	1				2	110	80	120	80	140	100	1	1	1	2	2	2	2	2	2	2
59	1	1				1	110	80	120	70	140	80	2	2	3	2	2	2	2	2	2	4
60	2	2	P1	L1		1	110	80	120	70	120	80	1	2	3	2	2	2	2	2	2	2

61	3	1				1	110	80	120	80	140	100	2	1	1	2	2	2	2	2	2	4
62	4	1				2	110	80	120	70	140	90	2	2	3	2	2	2	2	2	2	4
63	2	1				1	120	80	130	70	140	80	1	2	3	2	2	2	2	2	2	4
64	1	2	P1	L1		1	110	80	120	80	120	70	2	2	3	2	2	2	2	2	2	4
65	2	1				2	110	70	120	70	150	100	1	1	1	2	2	2	2	2	2	4
66	3	1				1	120	70	120	80	130	70	1	2	3	2	2	2	2	2	2	4
67	2	1				2	110	80	110	80	160	110	2	1	2	1	2	2	2	2	2	4
68	2	1				1	120	70	120	70	120	80	2	2	3	2	2	2	2	2	2	4
69	3	2	P1	L1		1	110	70	120	80	110	70	1	2	3	2	2	2	2	2	2	4
70	2	2	P1	L1		2	110	80	120	80	140	100	2	1	1	2	2	2	2	2	2	4
71	3	1				1	110	70	120	70	120	70	2	2	3	2	2	2	2	2	2	4
72	2	1				1	110	70	120	70	130	70	2	2	3	2	2	2	2	2	2	4
73	2	2	P1	L1		2	100	60	120	80	170	100	2	1	1	1	2	2	2	2	2	3
74	3	1				1	110	80	120	70	100	70	1	2	3	2	2	2	2	2	2	4
75	1	1				2	120	70	130	70	140	90	1	2	3	2	2	2	2	2	2	4
76	2	1				1	120	80	120	70	110	70	2	2	3	2	2	2	2	2	2	4

77	4	2	P1	L0		1	110	70	120	70	130	70	1	2	3	2	2	2	2	2	2	4
78	1	1				2	120	80	130	70	130	80	2	2	3	2	2	2	2	2	2	2
79	3	3	P2	L2		1	100	70	110	70	110	70	2	2	3	2	2	2	2	2	2	2
80	3	1				2	110	80	120	70	130	70	1	2	3	2	2	2	2	2	2	4
81	1	1				1	110	70	120	80	120	70	1	2	3	2	2	2	2	2	2	4
82	2	2	P1	L1		2	110	80	120	80	150	90	2	2	3	2	2	2	2	2	2	3
83	3	1				1	110	70	120	80	110	70	2	2	3	2	2	2	2	2	2	4
84	2	2	P1	L1		2	110	80	120	80	120	80	2	2	3	2	2	2	2	2	2	4
85	3	1				1	110	80	120	70	110	70	1	2	3	2	2	2	2	2	2	4
86	1	3	P2	L2		2	110	60	120	70	150	110	2	1	1	2	2	2	1	2	2	2
87	2	1				1	110	70	120	80	110	90	2	2	3	2	2	2	2	2	2	4
88	2	2	P1	L1		2	110	80	120	80	140	100	1	1	1	2	2	2	2	2	2	4
89	3	1				1	120	80	120	80	140	70	2	2	3	2	2	2	2	2	2	4
90	1	1				2	110	70	120	80	140	90	2	2	3	2	2	2	2	2	1	1
91	2	1				1	100	70	120	80	110	70	1	2	3	2	2	2	2	2	2	4
92	2	1				2	120	80	110	80	140	100	1	1	1	2	2	2	2	2	2	3

93	4	1				1	110	70	120	80	120	70	2	2	3	2	2	2	2	2	2	4
94	2	2	P1	L0		2	120	80	120	80	150	100	2	1	1	2	2	2	2	2	2	4
95	3	1				1	120	80	110	70	130	80	2	2	3	2	2	2	2	2	2	4
96	1	1				2	110	70	110	70	150	100	1	1	1	2	2	2	2	2	2	2
97	2	1				1	120	80	120	70	140	70	2	2	3	2	2	2	2	2	2	4
98	3	1				1	110	70	120	70	130	80	2	2	3	2	2	2	2	2	2	4
99	2	1				2	120	80	130	80	160	110	2	1	2	1	2	2	2	2	2	4
100	1	1				1	110	70	120	70	110	70	2	2	3	2	2	2	2	2	2	4
101	1	2	P1	L0		2	120	70	120	70	140	100	2	1	1	2	2	2	2	2	2	4
102	2	1				1	120	80	130	80	130	80	2	2	3	2	2	2	2	2	2	4
103	3	2	P1	L1		1	110	70	120	80	130	80	2	2	3	2	2	2	2	2	2	4
104	3	1				2	110	70	130	80	140	100	1	1	1	2	2	2	2	2	2	4
105	2	1				1	120	80	130	70	130	70	2	2	3	2	2	2	2	2	2	4
106	1	2	P1	L1		1	110	70	120	80	130	80	2	2	3	2	2	2	2	2	2	4
107	2	1				1	120	80	130	70	130	80	2	2	3	2	2	2	2	2	2	4
108	3	1				2	120	70	130	70	140	90	1	2	3	2	2	2	2	2	2	2

109	1	2	P1	L1		1	110	70	120	70	130	70	2	2	3	2	2	2	2	2	2	4
110	2	1				2	120	80	130	70	140	70	1	2	3	2	2	1	2	2	2	4
111	3	1				1	110	80	120	80	130	70	2	2	3	2	2	2	2	2	2	4
112	1	3	P2	L1	A1	2	110	80	130	70	140	100	1	1	1	2	2	2	2	2	2	2
113	2	2	P1	L1		1	110	80	130	80	140	80	2	2	3	2	2	2	2	2	2	4
114	3	1				2	120	80	130	80	140	100	1	1	1	2	2	2	2	2	2	2
115	2	1				1	120	80	130	80	110	70	2	2	3	2	2	2	2	2	2	4
116	2	1				2	110	70	120	70	150	90	2	2	3	2	2	2	2	2	1	1
117	4	2	P1	L1		1	100	70	120	70	120	80	2	2	3	2	1	2	2	2	2	4
118	2	2	P1	L1		1	110	80	120	70	110	70	2	2	3	2	2	2	2	2	2	4
119	2	2	P1	L1		2	120	70	120	80	140	90	1	2	3	2	2	2	2	2	2	4
120	2	1				1	110	80	120	80	130	80	2	2	3	2	2	2	2	2	2	4
121	1	1				1	120	70	120	70	130	80	2	2	3	2	2	2	2	2	2	4
122	2	1				2	110	80	110	70	140	100	1	1	1	2	2	2	2	2	2	2
123	3	3	P1	L1	A1	1	120	80	120	70	130	80	2	2	3	2	1	2	2	2	2	4
124	2	2	P1	L1		2	110	70	120	70	170	110	1	1	2	1	2	2	2	2	2	2

125	2	1				1	120	80	110	70	130	80	2	2	3	2	2	2	2	2	2	4
126	3	2	P1	L1		1	110	70	120	80	120	80	2	2	3	2	1	2	2	2	2	4

KEY OF THE MASTER CHART

Age	1=<20 Years, 2=20-25 Years, 3=26-30 Years, 4=>30 Years
Gravida	1=PRIMI, 2= SECOND GRAVIDA, 3=THIRD GRAVIDA
Placenta	1=Central, 2=Lateral
Mode of delivery	1=LSCS, 2=NVD
Preeclampsia	1=Yes, 2=No
Type of preeclampsia	1=Mild, 2=Severe, 3=NA
Eclampsia	1=Yes, 2=No
IUGR	1=Yes, 2=No
Pprom	1=Yes, 2=No
DIC	1=Yes, 2=No
HELLP	1=Yes, 2=No
Abruptio	1=Yes, 2=No
Fetal outcome	1=IUD, 2=NICU, 3=Still birth, 4=No adverse fetal outcome