"ROLE OF ULTRASONOGRAPHY AND COLOR DOPPLER IN ASSESSMENT OF HIGH-RISK PREGNANCIES AND ITS ACCURACY IN PREDICTING FETAL OUTCOME"

By

Dr. R. MAHIMA KALE

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR OF MEDICINE
IN
RADIODIAGNOSIS

Under the Guidance of

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2023

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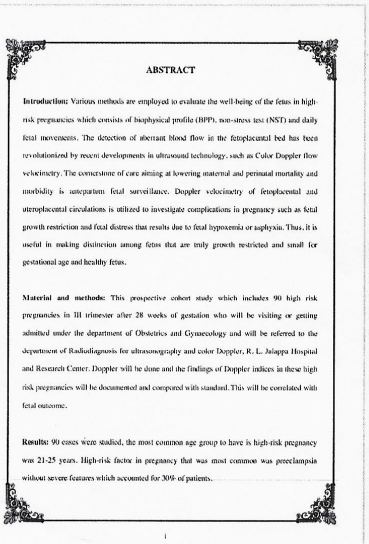

ABSTRACT

Introduction: Various methods are employed to evaluate the well-being of the fetus in high-risk pregnancies which consists of biophysical profile (BPP), non-stress test (NST) and daily fetal movements. The detection of aberrant blood flow in the fetoplacental bed has been revolutionized by recent developments in ultrasound technology, such as Color Doppler flow velocimetry. The cornerstone of care aiming at lowering maternal and perinatal mortality and morbidity is antepartum fetal surveillance. Doppler velocimetry of fetoplacental and uteroplacental circulations is utilized to investigate complications in pregnancy such as fetal growth restriction and fetal distress that results due to fetal hypoxemia or asphyxia. Thus, it is useful in making distinction among fetus that are truly growth restricted and small for gestational age and healthy fetus.

Material and methods: This prospective cohort study which includes 90 high risk pregnancies in III trimester after 28 weeks of gestation who will be visiting or getting admitted under the department of Obstetrics and Gynaecology and will be referred to the department of Radiodiagnosis for ultrasonography and color Doppler, R. L. Jalappa Hospital and Research Center. Doppler will be done and the findings of Doppler indices in these high risk pregnancies will be documented and compared with standard. This will be correlated with fetal outcome.

Results: 90 cases were studied, the most common age group to have is high-risk pregnancy was 21-25 years. High-risk factor in pregnancy that was most common was preeclampsia without severe features which accounted for 30% of patients.

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Natasha R. KUMAR, William A. GROBMAN, Nicola LANCKI, Olivia BARRY, Amelia C. CLEMENT, Lynn M. YEE. "Evaluating the maternal and perinatal sequelae of severe gestational

ACKNOWLEDGEMENT

I owe debt and gratitude to my grandparents ***HANUMANTHA RAO & L. N. SHANTHA BAI*** *and* ***Late. HANUMANTHA RAO. M & PUSHPA BAI****, and my parents* ***H. RAMESH KALE*** *and* ***H. SHOBHA KALE*** *for their moral support and constant encouragement during the study. I owe my gratitude to my uncle Late* ***Dr. H. BALAJI KALE*** *for his blessings. I would also like to thank* ***GEETHA KALE, KUBENDRA RAO, USHA. H, GANESH RAO N, ANAND G VANSE, DEEPAK G VANSE, PRAJWAL. K & SURESH RAO*** *for their constant support. I would also like to thank my friend for being a constant support in all the tough times.*

With humble gratitude and great respect, I would like to thank my teacher, mentor and guide, ***Dr. RAJESWARI,*** *Associate Professor, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar, for her able guidance, constant encouragement, immense help and valuable advices which went a long way in molding and enabling me to complete this work successfully. Without her initiative and constant encouragement this study would not have been possible. Her vast experience, knowledge, able supervision and valuable advices have served as a constant source of inspiration during the entire course of my study. I would like to express my sincere thanks and humble gratitude to my co-guide* ***Dr. S. R. SHEELA****, Professor, Department of Obstetrics and Gynaecology, Sri Devaraj Urs Medical College, Kolar, for her able guidance, constant encouragement, immense help and valuable advices. I would like to express my sincere thanks to* ***Dr. ANIL KUMAR SAKALECHA****., Professor and Head of Department of Radiodiagnosis, Sri Devaraj Urs Medical College for, valuable support, guidance and encouragement throughout the study. I would also like to thank* ***Dr. DEEPTI NAIK****, Professor, Department of Radiodiagnosis, and* ***Dr. HARINI BOPAIAH****, Asso. prof, Department of Radiodiagnosis, Sri Devaraj Urs Medical College for their wholehearted support and guidance.*

I am extremely grateful to the patients who volunteered for this study, without them this study would just be a dream.

I would like to thank ***Dr. VARSHITHA G.R, Dr. AASHISH, Dr. MONISHA V, Dr. CHAITHANYA, Dr. YASHAS ULLAS L and Dr. SURAJ H.S*** *and all my teachers of Department of Radiodiagnosis, Sri Devaraj Urs Medical College and Research Institute, Kolar, for their constant guidance and encouragement during the study period.*

I am thankful to my fellow postgraduates ***Dr. KALATHURU UHASAI, Dr. LYNN JOY, Dr. NIKHIL****,* ***Dr. ARUN, Dr. SANDEEP, Dr. MADAN, Dr. PRAVEEN, Dr. REVANTH R.B, Dr. GURU YOGENDRA, Dr. SHANTALA, Dr. POOJITHA, Dr. SURYA, Dr. SIVA, Dr. KRISHNA, Dr. MANNAN, Dr. GAURAV*** *and* ***Dr. RISHI*** *for having rendered all their co-operation and help to me during my study.*

My sincere thanks to ***Mr. SUNIL, Mrs. NASEEBA, Mrs. HAMSA*** *and rest of the computer operators.*

I am also thankful to ***Mr. RAVI,*** *and* ***Mr. SUBRAMANI*** *with other* ***technicians*** *of Department of Radiodiagnosis, R.L Jalappa Hospital & Research Centre, Tamaka, Kolar for their help.*

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LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
AC	Abdominal circumference
AEDV	Absent end diastolic velocity
AFI	Amniotic fluid index
BPD	Biparietal diameter
BPP	Biophysical profile
CDC	Center for disease control and prevention
CP Ratio	Cerebroplacental ratio.
CTG	Fetal cardiotocography
DBP	Diastolic blood pressure
DIC	Disseminated intravascular coagulation
DM	Diabetes mellitus
EDF	End-diastolic flow
EFW	Estimated fetal weight
FGR	Fetal growth restriction
FL	Femoral length
FVW	Flow velocity waveforms
GDM	Gestational diabetes mellitus
GH	Gestational hypertension
HC	Head circumference
HC/AC	Fetal head circumference and abdominal circumference
HTN	Hypertension
IUD	Intrauterine death
IUGR	Intrauterine growth restriction
IVC	Inferior vena cava
LBW	Low birth weight
LSCS	Lower segment Caesarean section
MCA	Middle cerebral artery

MI	Mechanical Index
NICU	Neonatal intensive care unit
NPV	Negative predictive value
NST	Non-stress test
PAS	Placenta accreta spectrum
PE	Preeclampsia
PI	Pulsatility index
PIH	Pregnancy induced hypertension
PPV	Positive predictive value
PRF	Pulse repetition frequency
PSV	Peak systolic velocity
REDV	Reversed end diastolic velocity
RI	Resistance index
SBP	Systolic blood pressure
SD Ratio	Systolic to diastolic ratio
SLE	Systemic lupus erythematosus
SVC	Superior vena cava
TI	Thermal Index
UA	Umbilical artery
US	Ultrasound
USG	Ultrasonography
UTA	Uterine artery

ABSTRACT

Introduction: Various methods are employed to evaluate well-being of fetus in high- risk pregnancies which consists of biophysical profile (BPP), non-stress test (NST) and daily fetal movements. Detection of aberrant blood flow in fetoplacental bed has been revolutionized by recent developments in ultrasound technology, such as Color Doppler flow velocimetry. The cornerstone of care is lowering maternal & perinatal mortality and morbidity is antepartum fetal surveillance. Doppler velocimetry of fetoplacental and uteroplacental circulations is utilized to investigate complications like fetal growth restriction (FGR) and fetal distress.

Material and methods: This prospective cohort study which includes 90 high-risk pregnancies in III trimester after 28 weeks of gestation on whom ultrasonography and Doppler was performed and the findings of Doppler indices will be documented and compared with standard. This was correlated with fetal outcomes.

Results: Among 90 cases, common high-risk factor in pregnancy was preeclampsia without severe features (30%). Wave pattens were acquired from middle cerebral artery (MCA), umbilical arterty (UA) and uterine artery (UTA). Cerebroplacental (CP Ratio) & UA pulsatility (PI) had better sensitivity & positive predictive value (PPV). Diagnostic accuracy of CP ratio (Accuracy=81.11%) was more in predicting adverse outcomes than other parameters.

Conclusion: CP Ratio & UA PI had better sensitivity & PPV in identifying unfavorable outcomes. Diagnostic accuracy of CP ratio was highest in predicting adverse outcomes. Study's findings support use of colour Doppler imaging in high-risk pregnancies will help in early identification of adverse fetal outcomes and aids in early intervention.

Key words: Colour Doppler, High-risk Pregnancies, fetal outcome, UA, MCA.

INTRODUCTION

INTRODUCTION:

The incidence of pre-eclampsia is 8-10% in pregnant women, according to National health portal of India. In India, a research found that frequency of hypertensive disorders during pregnancy was 7.8%, with preeclampsia (PE) occurring in 5.4% of the population.[1] Various methods are utilized to evaluate well-being of fetus in high risk pregnancies which include non-stress test (NST), biophysical profile (BPP) and daily fetal movements. The above tests are all less desirable since they lack high level of positive predictive value (PPV), sensitivity and specificity.

The identification of abnormal blood flow in fetoplacental bed has been revolutionized by recent advances in ultrasound (USG) such as Color Doppler flow velocimetry. Early detection of these abnormalities are helpful in determining optimal time for delivery & early diagnosis of intra-uterine growth restriction (IUGR) which reduces fetal mortality and morbidity.

This technique demonstrates blood flow in uterine arteries (UTA), umbilical artery (UA) and middle cerebral artery (MCA). This is a non-invasive technique to study uteroplacental and fetoplacental circulations. It is simple, safe and reproducible. Fetal hypoxia can be assessed with the abnormal wave patterns obtained from these vessels. Therefore, current study is important for accurate assessment of well-being of fetus in high-risk pregnancies in order to improve fetal outcome.

Fetal growth restriction (FGR) and fetal distress that results due to fetal hypoxemia or asphyxia can be investigated using Doppler velocimetry of uteroplacental and fetoplacental circulations.[2,3]

Thus, it is useful in distinguishing a healthy fetus from a fetus that is truly growth restricted from small for gestational age.[4]

NEED FOR THE STUDY:

Pregnancy is constantly monitored by laboratory investigations, clinical examination and radiographic examinations at regular intervals. High-risk pregnancies are those that pose a potential health or life risk to mother and/or fetus.[5] Diabetes, Advanced maternal age, autoimmune diseases, prior miscarriages, infectious diseases and/or substance abuse are some of the examples of such factors. Close follow-up by medical personnel is essential to reduce risks to the mother and fetus.[6] Gestational hypertension (GH) or pregnancy-induced hypertension (PIH) are significant causes of high-risk pregnancies.[7] Oligohydramnios can be either idiopathic or can co-exist with other conditions. Numerous adverse fetal & maternal outcomes can occur due to diabetes mellitus (DM) and anemia.[8] Various methods are utilized to evaluate the wellbeing of the fetus in high risk pregnancies which include NST, BPP and daily fetal movements. The above tests are all less desirable since they lack a high level of PPV, sensitivity and specificity in assessment of fetal well-being.

Doppler ultrasound's introduction to medicine has offered great advantage. Fitzgerald DE and Drumm JE published the first fetal Doppler ultrasound study in 1977.[9] Doppler signal, focused on fetal blood moving within UA, produced the waveforms by varying the ultrasound frequency. These flow velocity waveforms (FVW) from fetoplacental circulation are dependent on contraction force of fetal cardiac, peripheral or downstream resistance, density of blood and elasticity of vessel wall.[10] Indices of impedance are moderated as elevated fetal heart rates and breathing hence proposed that FVW's is obtained with mother in semi-recumbent position during inactivity of fetus.[11]

In high-risk pregnancies, particularly those with PE, gestational diabetes mellitus (GDM) and IUGR, Doppler ultrasound (US) is used to study blood flow to assess fetal inaccessibility in the UA and MCA of fetus. A non-invasive way for obtaining a qualitative and quantitative evaluation of maternal and fetal circulation has been made possible by development of Doppler US technology.[12] Color Doppler flow velocimetry which is recent advance in US technology has revolutionized diagnosis of abnormal blood flow in fetoplacental bed, and early detection of these abnormal patterns are helpful in determining optimal time for delivery to lower perinatal mortality. Further benefit of colour Doppler flow velocimetry is the ability to detect IUGR early, which can lower the fetal morbidity and mortality.[10,13] Doppler velocimetry of UA in unselected and low-risk pregnancies is not advised, according to a research.[14] Doppler technology has made it possible to examine fetal circulation non-invasively, as abnormal fetal circulation is thought to be a significant contributor to FGR, both as a cause and an indicator.[15]

This method makes it easier to delineate small intracranial vessels resulting in quicker and accurate examinations. With abnormal wave patterns obtained from this artery, fetal hypoxia can be evaluated. Therefore, this study was required to accurately assess fetal well-being in all high-risk pregnancies in order to improve fetal outcome and to incorporate this procedure a part of the protocol for assessment of fetal well-being in these patients. Given the aforementioned information, current study's objectives was to assess role of colour Doppler USG in high-risk pregnancies and to correlate results of fetal biometry and estimated fetal weight (EFW) in high-risk pregnant women.

AIMS & OBJECTIVES

AIM AND OBJCETIVES:

1. To determine the role of Doppler indices in high risk pregnancies and its accuracy in predicting fetal outcome.
2. To determine the fetal biometry and estimated fetal weight in high risk pregnancies.

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

When a pregnancy is high-risk, either mother or fetus is at an increased risk of adverse outcomes compared to uncomplicated pregnancies. There are currently no clear rules for differentiating “high-risk” and “low-risk” pregnancies, however some conditions that have demonstrated to increase the chance of adverse outcomes in mother or fetus.[16]

These conditions can be classified into three primary categories: health problems pre-existing in mother before she becomes pregnant, health problems in mother that occur during pregnancy and certain conditions with fetus.

The Center for disease control and prevention (CDC) projected that there were ~ 65,000 "high-risk" pregnancies in the United States in 2012.[5]

CAUSES

Maternal factors:

If the mother has certain pre-existing health conditions pregnancy may be deemed "high-risk”. These include the following:

1. **Age**

Advanced age – Refers to pregnancies in women over age of 35. While first-time mothers in this age group may experience normal pregnancies, but studies indicate that these women are at high-risk of having: first trimester miscarriage, chromosomal abnormalities in fetus and FGR, ectopic pregnancy, placenta previa & abruption, GDM, PE and cesarean delivery.[17] Such complications could lead to preterm birth and can also raise the risk of perinatal mortality.

An increased risk of fetal chromosomal abnormalities such as Down Syndrome (Trisomy 21) and Trisomy 13 are associated with advanced age.[17] There is increased risk of first trimester miscarriage in some of these chromosomal abnormalities.

Although the mechanism by which advanced age increases risk of FGR is not fully known, research has stated that it may be due to placenta dysfunction.

Younger age – According to CDC (2010), 3.4% of births in United States occurred among women between age 15 and 19. Compared to mothers between ages of 20 and 35, these adolescents are more susceptible to anemia, preterm delivery, low birth weight (LBW) and PE. Incidence of sexually transmitted diseases are high in adolescents.[18]

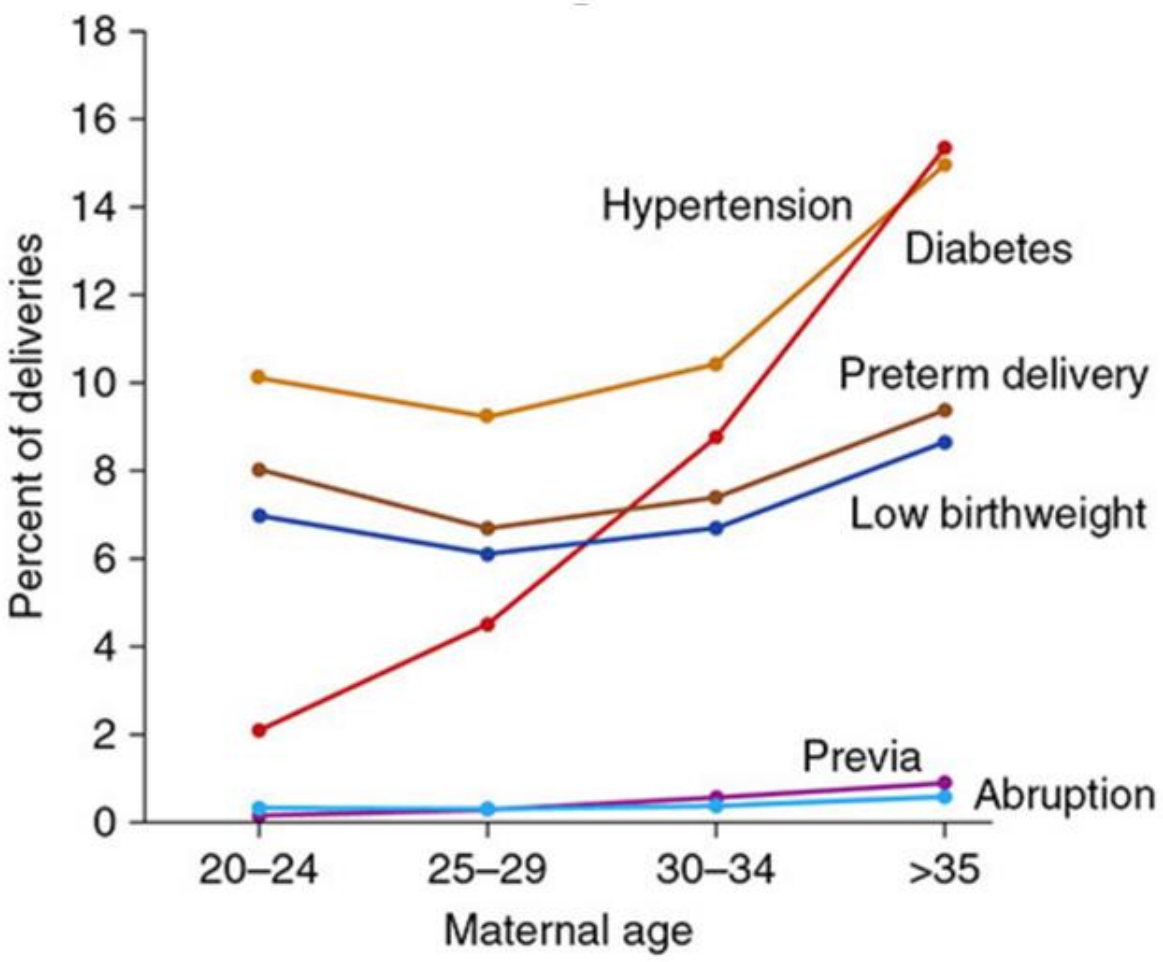


FIGURE 1: This line graph illustrates the incidence of selected pregnancy complications in relation to maternal age among women.

2. **Chronic high blood pressure:–**

According to the CDC, there are 166.9 cases of chronic hypertension (HTN) for every 100,000 hospital births in the United States.

HTN is risk factor because it leads to increased risk of PE, FGR and preterm birth.[19,20] The mechanism by which HTN increases the risk of these outcomes is not yet fully understood. However, it is believed that HTN causes decreased blood flow to placenta.[20]

Reduced blood flow to the fetus may result in restricted growth and cause other changes that increases the risk of PR, FGR, and pre-term birth.[20]

3. **Pre-existing diabetes mellitus:–**

Uncontrolled pre-existing DM during pregnancy raises the chance of spontaneous miscarriages in the first few weeks of pregnancy as well as congenital deformities such congenital heart problems and neural tube defects.[21,22]

Although the exact process by which hyperglycemia causes congenital abnormalities is still being researched, one possible factor is the increased oxidative stress that arises from hyperglycemia. The risk of preterm birth and high birth weight, often known as macrosomia is increased by preexisting DM. Due to shoulder dystocia during vaginal delivery the risk of brachial plexus injury is high in macrosomia.[21,22]

4. **Cardiac or heart disease** -

The volume of circulating blood increases during pregnancy.[23] This increased blood volume in women with cardiac disease has the potential to worsen/exacerbate existing heart disease.[24]

PE is common and may provoke after-load failure. Findings from the Registry on Pregnancy and Cardiac Disease indicate that women with preexisting heart disease who develop PE have 30 % risk of developing heart failure during pregnancy.[25] Heart failure in pregnancy is most frequently caused by chronic HTN with superimposed preeclampsia in numerous population.

5. **Autoimmune disease -**

Systemic lupus erythematosus (SLE): The incidence of lupus was almost 1 case per 900 births in a meta-analysis of over 9 million pregnancies. During pregnancy, lupus gets better in a third of women, remains the same in a third, and worsens in remaining third. Thus, clinical condition can worsen or flare without warning in any pregnancy. Compared to women who are unaffected, women with SLE have reported higher risks of having adverse pregnancy outcomes.[26]

Outcome	Relative Risk (CI)
Preeclampsia/Eclampsia	3.4 (3.2–3.6)
Cesarean delivery	1.4 (1.1–1.7)
Stillbirth	16.5 (2.9–92.1)
Fetal loss	7.6 (4.8–11.9)
Preterm birth	2.3 (1.8–3.1)
SGA infants	2.5 (1.4–4.5)
LBW infants	4.8 (3.7–6.3)
NICU admission	2.8 (2.3–3.4)

CI = confidence interval; LBW = low birth weight; NICU = neonatal intensive care unit; SGA = small for gestational age.

TABLE 1: Risk of adverse pregnancy outcomes in women with Systemic lupus erythematosus

Fetal-related factors

Some pregnancies can be classified as high risk due to particular disorders that can occur in the developing fetus or fetuses. To reduce the likelihood of morbidity and mortality in these cases, more care must be taken during pregnancy to treat these factors while the fetus is still in the womb. Fetal related factors that can result high risk pregnancy include:

- Congenital Malformations
- Multiple Pregnancies
- FGR: “either an EFW <10th percentile for gestational age or an AC <10th percentile for gestational age”

Pregnancy-related factors

A pregnancy may also be considered high-risk if the mother develops a medical condition or complications occur during pregnancy.

1. Conditions developed during pregnancy:

PREGNANCY INDUCED HYPERTENSION (PIH):[27]

Definition and classification:

PIH causes complications in 6-10% of pregnancies. Systolic blood pressure (SBP) >140 mmHg and diastolic blood pressure (DBP) >90 mmHg are the criteria for this diagnosis of this condition.

Classification:

01	Mild	SBP of 140-149 and DBP of 90-99 mmHg
02	Moderate	SBP of 150-159 and DBP of 100-109 mmHg
03	Severe	SBP of ≥160 and DBP of ≥110 mmHg

Preeclampsia (PE):

PE is a syndrome characterized by a significant rise in a pregnant woman's blood pressure after the 20th week of pregnancy. Mother's kidneys, liver, and brain are affected. If condition is left untreated, it may be fatal to the mother and/or the fetus and cause long-term health issues.[28]

Eclampsia: Convulsion in pregnant lady with preeclampsia that is not attributable to another cause. Seizures may appear before, during or after labor and is generalized.[29]

Condition	Criteria Required
Gestational hypertension	BP >140/90 mm Hg after 20 weeks in previously normotensive women
Preeclampsia: Hypertension plus	
Proteinuria	≥300 mg/24h, or Urine protein: creatinine ratio ≥0.3, or Dipstick 1+ persistent[a]
	or
Thrombocytopenia	Platelets <100,000/μL
Renal insufficiency	Creatinine >1.1 mg/dL or doubling of baseline[b]
Liver involvement	Serum transaminase levels[c] twice normal
Cerebral symptoms	Headache, visual disturbances, convulsions
Pulmonary edema	—

[a]Recommended only if sole available test.
[b]No prior renal disease.
[c]Aspartate transaminase (AST) or alanine transaminase (ALT).
BP = blood pressure.

TABLE 2: Showing the criteria to diagnose gestational hypertension and preeclampsia.

HELLP Syndrome: Seen in PE patients, they present with pain in right upper quadrant, hemolysis, elevated liver enzymes and low platelets. [30]

Gestational diabetes mellitus (GDM): Any degree of glucose intolerance with onset or initial detection during pregnancy is defined as GDM. Many women can have healthy pregnancies if they control their diabetes and adhere to their doctor's recommended diet and treatment plan. Uncontrolled GDM increases risk for adverse fetal outcomes such as preterm labor and delivery, PE and other HTN related conditions in pregnancy. Additionally, some research indicates that GDM is linked to long-term effects like development of type 2 diabetes in mother and obesity in infant.[31-33]

Fetal complications in GDM [34]

1. Macrosomia: Most constant complication in GDM.
2. Preterm birth: Studies have shown that GDM increases the risk of preterm deliveries.
3. Hypoglycemia: Neonatal hypoglycemia, elevated cord C-peptide levels reflecting foetal insulin production, and macrosomia have long been associated.
4. Hyperbilirubinemia.

5. Infants of diabetic mothers have normovolemic polycythemia.
6. Perinatal asphyxia.

2. Timing of pregnancy:

a. Preterm birth (infants born before 37 weeks of gestation)[35,36]

i. PROM (Prelabor Rupture of Membranes)[37-39]

b. Post-term pregnancy (infants born after 42 weeks of gestation)

3. Placenta: Is a structure within uterus that helps mother and fetus exchange nutrition, oxygen, and waste products. Pregnancy is more challenging and requires careful delivery strategy when this connection between mother and fetus is abnormally positioned.

- **Placental abruption**: Also known as abruptio placentae, is separation of placenta from its implantation site before delivery.

Ananth and colleagues (2016)[40] have defined severe placental abruption as displaying one or more of the following:

(1) Maternal consequences, such as hysterectomy, renal failure, shock, disseminated intravascular coagulation (DIC), or death;

(2) Fetal complications: Non-reassuring fetal status, growth restriction, or mortality;

(3) Neonatal adverse outcomes: Death, preterm delivery, or growth restriction.

- Placenta accreta:

Placenta accreta spectrum (PAS) describes aberrant placentation likely abnormally implanted, invasive, or adhered placenta. By measuring the extent of trophoblastic proliferation, PAS variants are categorised.[41]

4. Infections –

Various infections may be passed from the mother to the fetus, increasing risk of adverse pregnancy outcomes. During pregnancy, placenta could transmit an infection the mother already has to the fetus. Additionally, during delivery through the vaginal canal or breastfeeding, a newborn infant is directly exposed to infections. Pregnancy-related fetal infections may result in spontaneous abortion or affect normal growth and development of fetus.[42] Group B streptococcus,[43] Bacterial vaginosis,[44] yeast infections,[45] and Zika virus[46] infections are notably associated with pregnancy. Even though some of these diseases are rare severe infant morbidity and mortality, especially if they spread throughout the fetal nervous system. According to preliminary research, COVID-19 maternal infection during pregnancy may raise the risk of undesirable consequences including preeclampsia.[47]

5. Twin-to-twin transfusion syndrome[48]

Most newborns in high-income nations develop healthily inside womb. If mother has a medical condition like high blood pressure, diabetes, heart /renal conditions, or if placenta does not develop normally, all this could have an impact on the growth of fetus. Doppler ultrasound can identify changes in the circulation of fetal blood. If measures like early delivery are used to treat babies with growth issues, serious illness and death may be avoided. Doppler ultrasonography, however, might lead to more interventions like caesarean sections.

ANATOMY OF FETO-PLACENTAL & UTEROPLACENTAL CIRCULATION

FETO-PLACENTAL CIRCULATION[49]

The two umbilical arteries carry deoxygenated venous-like fetal blood to placenta. As cord joins placenta, these umbilical vessels branch repeatedly beneath the amnion as they run across the chorionic plate. Branching continues within villi to ultimately form capillary networks in terminal villous branches. Blood with significantly higher oxygen content returns from the placenta via a single umbilical vein to fetus. Branches of umbilical vessels that traverse along the chorionic plate are called placental surface vessels or chorionic vessels. They respond to vasoactive substances, but anatomically, morphologically, histologically, and functionally, they are unique. Chorionic arteries always cross over chorionic veins. Vessels are most readily recognized by this anatomical relationship, but they are difficult to distinguish by histological criteria.

Truncal arteries are perforating branches of the surface arteries and pass through the chorionic plate. Each truncal artery supplies one main stem villus and thus one cotyledon. As the artery penetrates the chorionic plate, its wall loses smooth muscle, and its caliber increases. The loss of muscle continues as the truncal arteries and veins branch into their smaller rami.

End-diastolic flow (EDF) is not seen in UA at end of fetal cardiac cycle before 10 weeks of gestation. After 10 weeks, EDF appears and is maintained throughout normal pregnancy. To evaluate fetal well-being the flow patterns are studied with Doppler sonography.

MATERNAL CIRCULATION [49]

Mechanisms of placental blood flow must allow blood to leave maternal circulation; flow into an amorphous space lined by syncytiotrophoblast; and return through maternal veins without producing arteriovenous-like shunts that would prevent adequate exchange between maternal blood and fetal villi. For this, maternal blood enters through the basal plate and is driven high up toward the chorionic plate by arterial pressure before laterally dispersing. After bathing external microvillous surface, maternal blood drains back through venous orifices in the basal plate and enters uterine veins. Thus, maternal blood traverses placenta randomly without preformed channels. Trophoblast invasion of spiral arteries creates low-resistance vessels that can accommodate massive increase in uterine perfusion during gestation.

Generally, spiral arteries are perpendicular to, but veins are parallel to, uterine wall. This arrangement aids closure of veins during a uterine contraction and prevents the exit of maternal blood from the intervillous space. Number of arterial openings into intervillous space is gradually reduced by cytotrophoblastic invasion to approximately 120 entry sites at term. These discharge blood in spurts to bath the adjacent villi. After the 30th week, a prominent venous plexus lies between decidua basalis and myometrium and helps develop cleavage plane needed for placental separation after delivery.

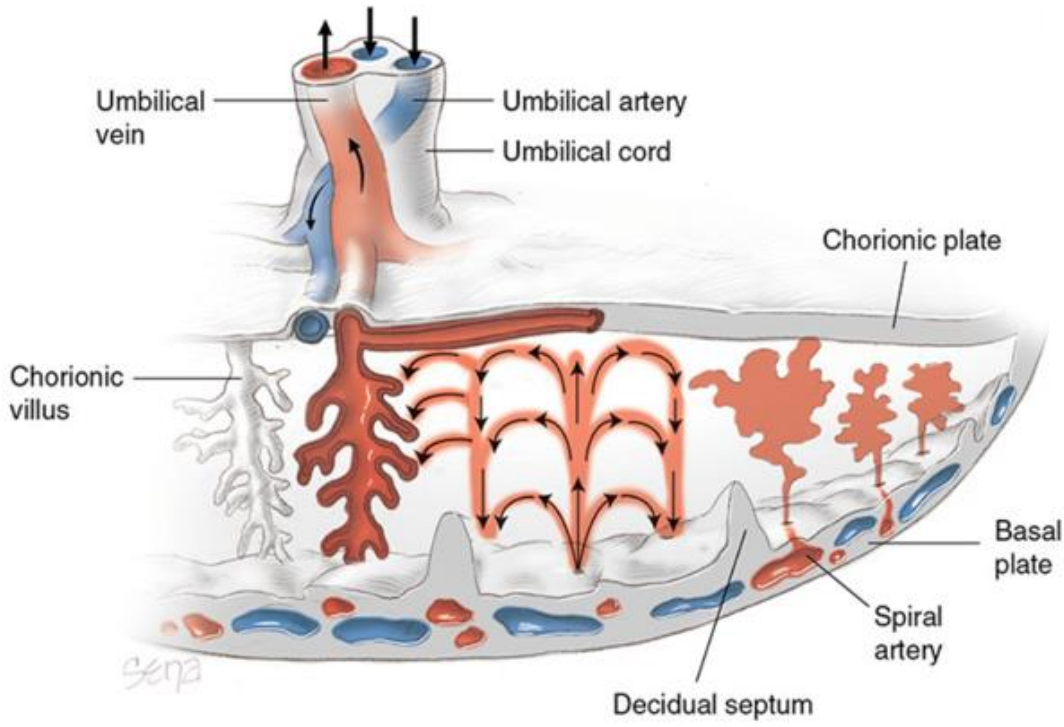


FIGURE 2: Schematic drawing of a section through a full-term placenta. Maternal blood flows into the intervillous spaces in funnel-shaped spurts, and umbilical arteries carry deoxygenated fetal blood to the placenta. Exchanges between the maternal and fetal systems occur as maternal blood flows around the villi. The umbilical vein carries oxygenated blood back to the fetus. Inflowing arterial blood pushes maternal venous blood into the endometrial veins, which are scattered over the entire surface of the decidua basalis. Placental lobes are separated from each other by placental (decidual) septa. [55]

FETAL CIRCULATION:[50]

This special circulation functions until birth, when it undergoes a significant modifications, and is very different from that of an adult. For example, fetal blood is oxygenated by the placenta and does not need to enter pulmonary vasculature. Thus, most of right ventricular output bypasses the lungs. Additionally, fatal heart's chambers operate in parallel rather than in series. This essentially supplies highly oxygenated blood from the dominant right ventricle to brain and heart than the rest of the body.

single umbilical vein supplies nutrients and oxygen from placenta to fetus for its growth and maturation. Vein subsequently divides into portal sinus and ductus venosus. Main branch of umbilical vein, ductus venosus, travels via liver and directly enters inferior vena cava (IVC). It delivers well-oxygenated blood directly to heart instead of supplying oxygen to intervening tissues. On other hand, portal sinus predominantly supplies blood to hepatic veins on left side of liver, where oxygen is extracted. IVC receives deoxygenated blood from liver & also from lower body. Blood reaching fetal cardia from IVC, consists of admixture of arterial blood that flows directly through ductus venosus and less well-oxygenated blood that returns from most of veins below level of diaphragm. Thus, oxygen concentration of blood exiting placenta is higher than that of blood delivered to heart from IVC.

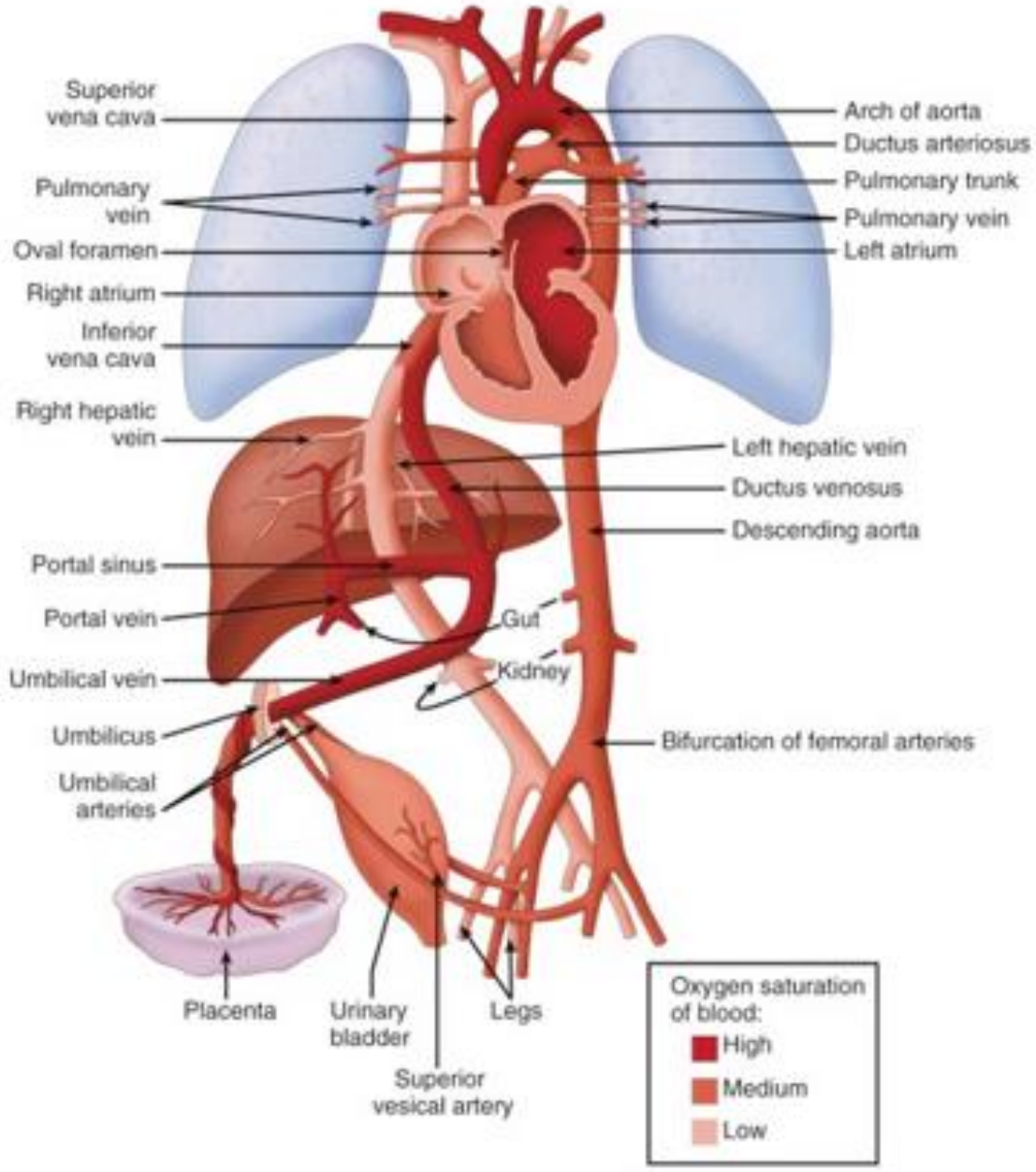


FIGURE 3: The fetal circulation demonstrating flow pathways from placenta to fetus. Shadings indicate the various oxygen saturations. The most highly oxygenated blood returns via the umbilical vein and is preferentially directed across the foramen ovale to the left atrium and left ventricle. Relatively deoxygenated blood mixes in the right atrium and moderately saturated blood is then ejected out of the right ventricle across the ductus arteriosus to the descending aorta. The umbilical arteries arise from the internal iliac arteries and deliver blood to the placenta to replenish oxygen supplies

Ventricles of fetal heart work in parallel, and hence right ventricle account for 2/3rd of total cardiac output. Well oxygenated blood enters left ventricle, which supplies heart and brain, and further less oxygenated blood fills right ventricle, which supplies rest of the body. Congenital cardiac defects may contribute to dysregulated brain development or placenta dysfunction.

These two separate circulations are maintained by right-atrium anatomy, which effectively directs entering blood to either the left atrium or the right ventricle, depending on its oxygen content. The pattern of blood flow through the IVC facilitates this division of blood according to its oxygen concentration. Well-oxygenated blood tends to course along the dorsomedial aspect of IVC and the less oxygenated blood flows along the lateral vessel wall. This aids their shunting into opposite sides of heart. Arrangement of the upper interatrial septum, known as the crista dividens, shunts the well-oxygenated blood from the medial side of the IVC through the foramen ovale into the left heart once this blood enters the right atrium. Here, it is directed to the heart and brain. After these tissues are supplied with the necessary oxygen, the resultant less oxygenated blood through the superior vena cava (SVC) enters right atrium. SVC blood flow velocity increases after 20 weeks of gestation until term pregnancy.

Less oxygenated blood that is flowing through the IVC's lateral wall reaches right atrium and is diverted to right ventricle by tricuspid valve. When SVC enters right atrium, inferiorly and anteriorly ensuring that less oxygenated blood leaving brain and upper body is directly shunted right ventricle. Similarly, coronary sinus' ostium sits just above tricuspid valve, allowing the right ventricle to receive less oxygenated blood from heart. Due to this blood flow pattern, right ventricle blood is 15 to 20% less oxygen-saturated than left ventricle blood.

Ductus arteriosus shunts ~ 90 % of blood exiting the right ventricle into descending aorta. Only around 8% of right ventricular output travels to lungs due to low resistance in ductus arteriosus and relatively high pulmonary vascular resistance and umbilical-placental vasculature (Fineman, 2014). As a result, the body receives 1/3rd of blood that passes via the ductus arteriosus. Through two hypogastric arteries, remaining right ventricular output returns back to the placenta. UA's run along the abdominal wall from the level of the bladder to the umbilical ring and into the cord. This blood is recirculated to the umbilical vein after being oxygenated in placenta.

PHYSICS OF DOPPLER ULTRASONOGRAPHY

The fetoplacental and uteroplacental circulation's blood flow can be evaluated with doppler ultrasound by non-invasively. This technique makes use of sound waves to measure blood flow through a vessel.[51] First documented in 1977, Drumm and Fitz Gerald showed how Doppler frequency shift waveform from the UA circulation might be used for fetal studies.

DOPPLER ULTRASOUND & PHYSICS OF DOPPLER

THE DOPPLER EFFECT: Christian Doppler, a professor of physics, developed the Doppler principle in 1842. To explain the apparent variations in star colors, he developed the principle.[51] According to the Doppler principle, the frequency of energy reflected from a moving boundary varies in relation to the moving boundary's velocity.[52] The frequency of the echoes produced by moving structures changes, and the degree of the shift is directly proportional to velocity of moving structure. Waveforms are used to graphically depict the Doppler shift. The waveforms show variations in the blood's flow rate across the vessels.

In systole, the velocity is higher, and in diastole, it is lower. Doppler flow is also known as Doppler velocimerty.[51] Fetal blood flow is affected by contractility of the cardia, physical characteristics of arterial walls, blood viscosity within confines of small blood vessels, and outflow impedance from arterial tree. Doppler frequency shift and blood flow velocity have a complex relationship that depends on a number parameters.

"This is expressed by the Doppler equation. [53]

$$Fd = \frac{[\ 2\ F\ v\ \cos\ q\ ]}{C}$$

Fd - Doppler frequency shift

F - Tansducer frequency

q - angle between incident ultrasonic beam and the axis of blood flow

v - blood flow velocity

c - velocity of sound in time." [53]

Flow velocity waveform (FVW) and volume blood flow are two types of information regarding blood flow that can be obtained through Doppler. FVW contains data about velocity of every blood cell in vessel being probed under USG. Downstream resistance can be measured using certain indices.

Three indices are in common use [53]–

- Resistance Index (RI)
- Pulsatility Index (PI)
- Systolic / Diastolic (S/D) ratio.

These ratios are independent of angle that the ultrasound beams are at with respect to the blood vessel. When the angle is unknown and the absolute velocity cannot be determined, this is significant. The indices are derived based on a statistically supported correlation between them and adverse clinical findings. In the belief that they represent downstream resistance, they are frequently regarded as resistance indices.

Despite not being widely accepted for screening low-risk populations, Doppler study has been shown to be effective in assessing high-risk pregnancies. It is useful in identifying fetuses with physiological compromises, according to several studies. The majority now concur that women who are vulnerable should have access to devices with pulsed Doppler capability. [53]

Table 3: Components of a Doppler ultrasound unit

Components of a Doppler ultrasound unit [51]		
Transducer	Central unit	Output
-Transmitting crystals -Receiving crystals	-Changes in frequency detected	-Visual -Oscilloscope -Strip chart -Audible sounds

DOPPLER INDICES

They are-

1. Systolic – Diastolic ratio (S/D ratio) : Ratio is calculated from the height of systolic and diastolic peaks.

2. Resistance index (RI) or Pourcelot index:

$$\frac{\text{Systolic - Diastolic}}{\text{Systolic}}$$

Difference in peak systolic velocity and end diastolic velocity is divided by systolic value

3. Pulsatility index (PI) or Gosling index:

$$\frac{\text{Systolic – Diastolic}}{\text{Mean}}$$

The difference in peak systolic velocity and end diastolic velocity is divided by mean velocity

High values of these indices indicate reduced perfusion of a specific location and obstruction to blood flow.[51]

The amount of blood flow required for the development of the fetal organs is associated with higher systolic and diastolic velocities, as well as lower RI. [54]

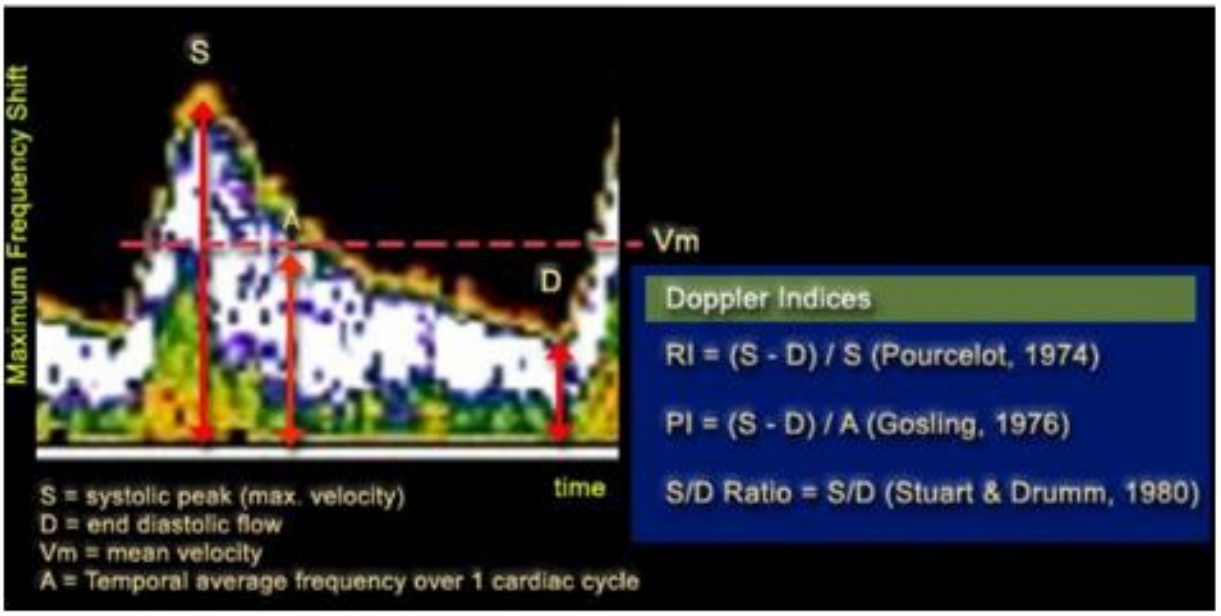


FIGURE 4: Flow velocity indices

BIO-SAFETY CONSIDERATIONS:

Since fetal harm from diagnostic insonation has never been documented, USG exposure is thought to be safe. It is now impossible to completely rule out possibility of a subtle, long-term, or cumulative harm. American Institute of Ultrasound in Medicine and FDA issued clear instructions on acoustic power output for Doppler Machine users & producers. In more than 25 years of use, no fetal dangers have been proven in low intensity range of grey scale imaging.

Doppler shift imaging has recently been developed in conjunction with grey scale imaging to localize spectral wave shape & superimpose color or intensity mapping. Due of this, FDA has restricted prenatal imaging ultrasound energy exposure to < 94 mW/ cm^2. he Mechanical Index (MI) and Thermal Index (TI) are currently the preferable measurements of ultrasonic production above intensity in mW/cm^2. [55]

MODES OF DOPPLER ULTRASOUND:

(1) CONTINUOUS WAVE DOPPLER –

Two distinct piezoelectric elements are present in the transducer, one of which transmits a high-frequency sound wave and the other of which continuously receives signals. It is simple to use and can record high frequencies at low power outputs. Information about the vessel being scanned is provided by this continuous wave Doppler in a rather non-selective manner. Continuous wave Doppler application is limited to study of superficial vessels or vessels suspended in a non-pulsatile media in measurement of blood flow velocities. This is because-

- It is unable to distinguish between signals coming from various moving components along beam.
- Blood vessel's position in relation to Doppler beam is uncertain or unstable, maintaining its position in beam path will be challenging.

(2) PULSE WAVE DOPPLER-

It has an equipment with a single crystal that sends out a signal and then waits for the return signal before sending out the next signal one. Although more expensive and requiring more power, it enables for exact targeting and visualization of the target vessel. The pulse repetition frequency (PRF) imposes a depth restriction since the ultrasound must travel to the vessel and back.

(3) DUPLEX DOPPLER-

Sound waves are emitted intermittently during Duplex Doppler or Colour Doppler sonography, and depth information can be retrieved depending on the time difference between the emitted and returned sound. Duplex Doppler can solve the two main practical drawbacks of continuous wave Doppler devices—poor spatial orientation and a lack of depth selectivity.

Fetal exposure is higher when using Duplex Doppler since it requires a considerably higher power output.

The device will be more expensive, less portable, more sophisticated with higher output sound intensities. Pulsed wave Doppler waveforms and the real-time image are displayed concurrently using duplex Doppler equipment.

(4) COLOUR DOPPLER-

Blood flow mean velocities are translated into a color image using color Doppler imaging, and color of image represents direction of the blood flow in relation to transducer. Using color Doppler technology, a wide field of view can be simultaneously imaged, exhibiting flowing blood in all vessels within selected area of interest. Ideal locations to capture spectral waveforms are determined by color Doppler images. The transducer flow is typically represented in red when it is directed toward it, and blue (Callen)when it is directed away, according to convention, which determines the color assigned. Low intensities are used in color Doppler sonography. Thus, using color Doppler imaging indirectly shortens the duration of the test and reduces the fetus' exposure. Assessing the effectiveness of organ perfusion is one of the most crucial goals of any hemodynamic investigation.

There are primarily two ways to use Doppler frequency shift data-

- Measurements of volumetric flow
- Analysis of Doppler waveforms

VOLUMETRIC FLOW MEASUREMENTS:

This can be calculated using the cross-sectional area and mean velocity during a cardiac cycle. Mean velocity can be calculated from mean frequency shift if insonation angle is known. Fetal left & right ventricular outputs, as well as flows in umbilical vein and descending thoracic aorta, have all been measured using this method. Estimating volumetric blood flow measurements involves various technical challenges. There are several different sources for errors to occur. The measurement of vessel diameter is a significant cause of error. The reliability of Doppler flow quantification is also significantly impacted by the angle of insonation. Because of their deep placement, tortuous course and small size; uterine, umbilical and cerebral vessels are virtually impossible to treat with this technique. Due to all of these issues, clinical applications of blood flow measurement have mostly been abandoned.

ANALYSIS OF DOPPLER WAVEFORM:

The difficulty in estimating volume flow has resulted in the development of indirect flow indices that may offer helpful data on flow without introducing too many errors. These indices do not require vessel diameter measurements and are independent of the angle of insonation. The two main waveform characteristics that serve as the foundation for this study are

1. Pulsatility, or the distinction between peak systolic and diastolic components of maximum frequency shift envelope.
2. End diastolic component is the key factor determine the pulsatility in fetal peripheral circulation.

DETERMINANTS THAT AFFECT DOPPLER MEASUREMENTS:

The velocity of the UA, UTA and MCA is influenced by a number of variables, which affects the variance of Doppler indices.

1. ***Gestational Age***: Waveform shows a progressive rise in end diastolic component of frequency shift and concurrent reduction in pulsatility with advancing gestation. The Doppler indices reflect these changes. [56]

2. ***Fetal Heart Rate***: When there is bradycardia, length of diastolic phase increases and end diastolic velocity reduces. These changes lead to increase in S/D ratio, pulsatility and resistance index. Fetal heart rate changes contribute to 15-18% of the variance of Doppler indices and correcting fetal heart rate for GA and heart rate can significantly reduce the variance. However it has not been demonstrated that such correction improves diagnostic efficacy.

3. ***Fetal Breathing***: Fetal breathing alters the maximum frequency shift envelope from one cardiac cycle to the following cycle. During breathing venous flow varies because of changes in intra-thoracic pressure. This variation may affect umbilical artery diastolic and systolic velocities by altering the placental and ventricular filling respectively. Largest variation occurs during diastolic umbilical arterial flow. It is therefore recommended that the indices should be measured only during foetal apnoea. This is applicable to MCA also.

4. ***Fetal Behavior State***: The waveforms of the aortic and internal carotid arteries change with fetal activity. The umbilical artery shows no signs of these modifications.

5. ***Uterine Contraction***: UTA systolic and diastolic flow can be changed. Therefore, it is only assessed when the uterus is relaxed. [57]

6. ***Location of Measurement***: Doppler signals are often captured from UA's middle free-floating loop. Cord's resistance is highest close to where it enters the fetal abdomen and lowest at its placental termination. In the instance of the MCA, recordings were obtained from the proximal first third of the artery. The UTA first splits off branches at the internal cervical os level before running along the lateral part of the uterine body, where it is frequently sampled. Placental locations have lower RI than non-placental sites.

7. ***Intra and Inter Observer Variation***: SD ratio & other indices have relatively little intra- and inter-observer measurement errors.

ANTEPARTUM FETAL MONITORING

Identification of fetuses at higher risk for perinatal mortality and morbidity is one of key objectives of antepartum fetal surveillance. Combination of IUGR with fetal hypoxia and asphyxia is associated with a higher-risk. Focus has been placed on Doppler sonography's capacity to recognize the growth-restricted, hypoxic, or distressed fetus. There is typical redistribution of blood inside the fetus and the placenta in the fetuses with IUGR. Doppler technology has significantly improved our capacity to evaluate the fetus's physiological state. When other tests are normal, it can be useful in spotting abnormalities in the fetal circulation and spotting the truly hypoxic fetus. In order to prevent any complications and perinatal mortality, the best timing for delivery may be determined by interval variations in Doppler measurements.
53

DOPPLER VELOCIMETRY OF FETAL & UTEROPLACENTAL CIRCULATION

UMBILICAL ARTERIAL CIRCULATION:

The component of fetal circulation that has been studied the most is this one. It is among the simplest targets to visualize. The UA diastolic flow velocity typically increases as the gestation and trophoblastic invasion advance, reflecting a gradually declining placental resistance. According to clinical studies, in healthy pregnancies, there is a strong correlation between the RI, S/D ratio and PI. In fetuses that are normally developed, placental insufficiency is main cause of IUGR, and UA Doppler can be used to diagnose it.[58,59]

Since early (7weeks) in pregnancy, Doppler ultrasound has been used to study the umbilical circulation. The diastolic blood flow component gradually appears after 12 weeks and should be present in all pregnancies by 16 weeks. Beginning at 20 weeks, the placenta grows as a result of a rise in number of functioning vascular villi. Doppler waveforms of UA indicate a consistent increase in EDV and a drop-in impedance indices as pregnancy progresses. The umbilical cord's flow velocity waveforms have a distinctive saw-tooth appearance, with continuous umbilical venous blood flowing in one direction and arterial flow in the other. Blood flow generally isn't pulsatile in the umbilical vein, hence the emergence of atrial pulsations has been interpreted as a sign that the fetus health is deteriorating.

Method: The umbilical artery and vein's distinctive waveforms are obtained by placing a transducer on the mother's belly directly above the fetus and carefully manipulating them.

Interpretation - The Doppler waveform is influenced by where the Doppler sampling site is located in the umbilical cord. Compared to the placental end of the cord, the indices are noticeably higher at the fetal end. Clinically reliable ones are those at the placental insertion or midcord. [60]

Abnormal waveforms of the UA are caused by a reduction in villi in the placenta. According to studies, higher UA impedance only becomes noticeable when at least 60% of placental vascular bed has been destroyed.

Significance of abnormal waveforms is as follows:

1. S/D ratio is considered increased if it is > 3.5 in late second & third trimesters.
2. Reversed end diastolic velocity (REDV) or Absent end diastolic velocity (AEDV) in the UA suggests elevated placental vascular impedance.
3. With extremely high individual variability, the median time between the onset of AEDV and aberrant fetal heart rate tracings is approximately one week.
4. Pregnancies with REDV (35.7%) have a much greater perinatal mortality rate than AEDV (8.9%).
5. Over 50% of AEDV-related pregnancies are worsened by maternal hypertensive problems & IUGR is reported to be 83% common with AEDV.
6. Fetal abnormalities (chromosomal defects) are more common in pregnancies with AEDV.
7. UA flow impedance may be raised or normal in post-term pregnancies that have adverse outcomes.
8. In twin pregnancies, a useful indicator of the onset of IUGR and adverse outcome is increased resistance to flow in UA.

When UA Doppler waveform measurement is incorporated into the management regimens for high-risk pregnancies, trials have demonstrated a significant reduction in perinatal death.[61] The time span between the detection of an aberrant UA Doppler waveform and the onset of fetal distress and/or intrauterine death (IUD) varies widely, ranging from days to weeks.

Due to the low likelihood of true positive cases being detected by UA Doppler at 20 weeks gestation, Doppler velocimetry cannot be utilized as a screening test.

After 30 weeks of pregnancy, the Doppler spectral discovery that has gained the most recognition is the AEDF or REDF. This discovery has been linked to perinatal mortality in 28% (and in 100% if untreated) and IUGR in 100% of cases, both of which are warning signs of impending fetal death. Nearly 8 days could pass before pathologic cardiotocographic signs are noticed when there is ADEF. REDF is a sign that preplacental blood with low oxygen content is being directed from descending aorta and pulmonary artery into brain.[62] This finding should be viewed as concerning because it has implications for obstetric diagnosis and care. [63, 64]

Numerous studies have attempted to demonstrate use of umbilical Doppler in prediction of IUGR, with the exception of the AEDF. [65-72] When compared to controls, fetuses with aberrant umbilical Doppler indices have only a 2-4 times increased risk of IUGR. Although there is a higher chance of a caesarean section for conditions like fetal distress, preterm delivery, admissions to neonatal intensive care units (NICU), the need for assisted ventilation, and perinatal mortality, umbilical Doppler has a low sensitivity in detecting IUGR, making it a poor screening tool in a less risk population.

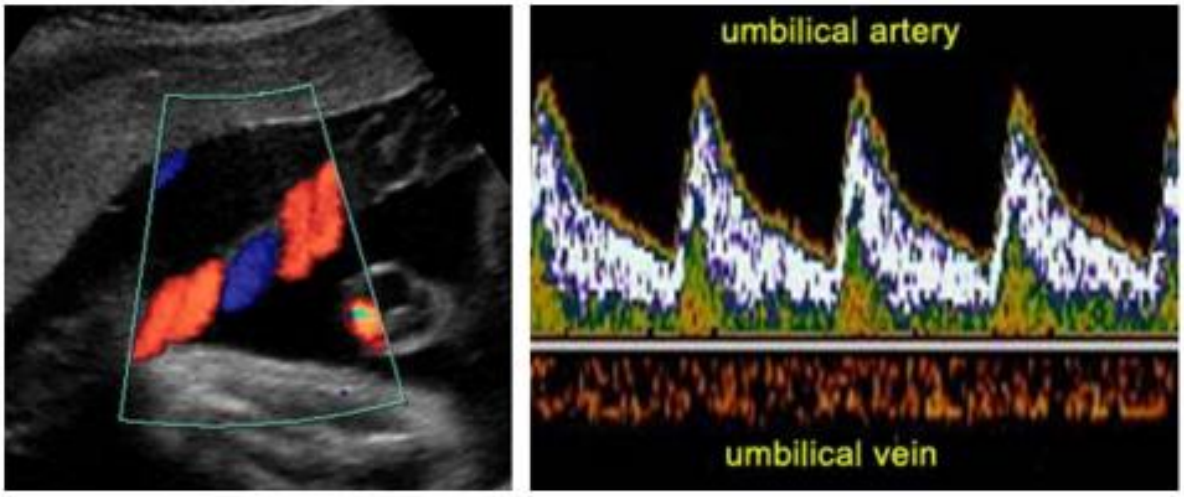


FIGURE 5: Ultrasound image with color Doppler showing the umbilical cord, red umbilical artery and blue umbilical vein (left). Normal flow velocity waveforms from the umbilical vein (bottom) and artery (top) at 32 weeks of gestation (right).

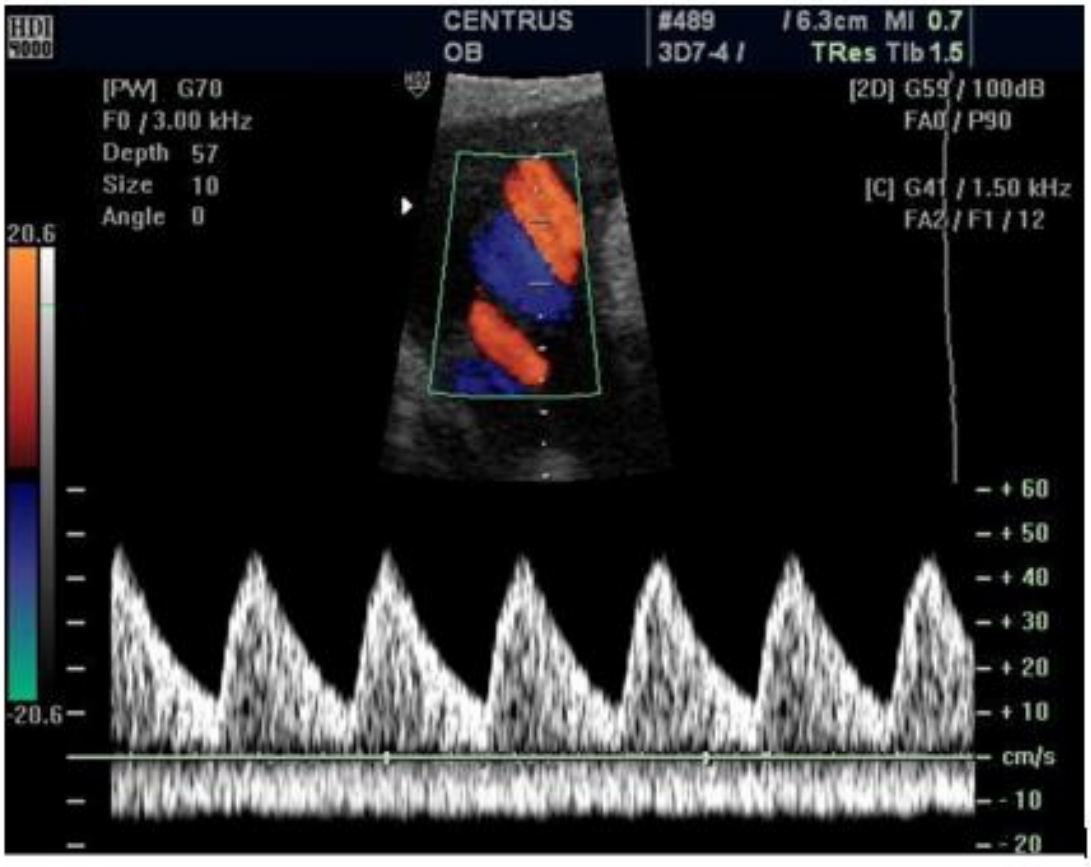


FIGURE 6: Normal flow velocity waveforms from the umbilical vein (top) and artery (bottom) at 32 weeks of gestation.

Normal pregnancy development of umbilical artery

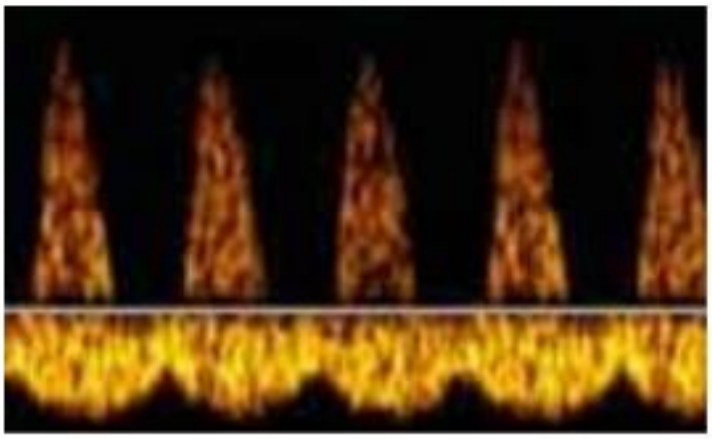


FIGURE 7: Normal impedance to flow in the umbilical arteries and normal pattern of pulsatility at the umbilical vein in 1° trimester

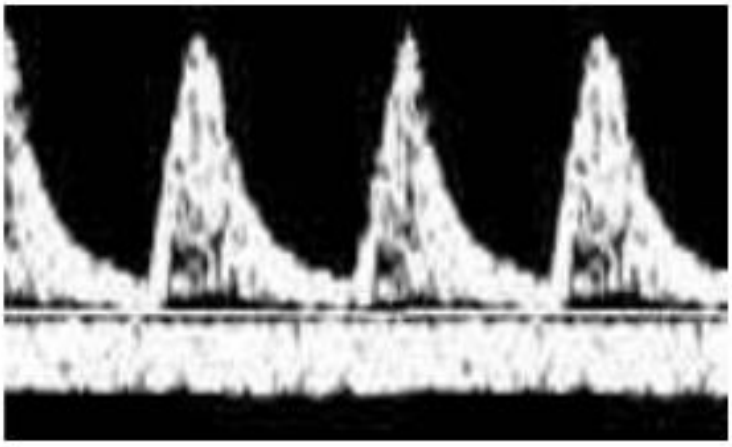


FIGURE 8: Normal impedance to flow in the umbilical arteries and umbilical vein in early 2°trimester

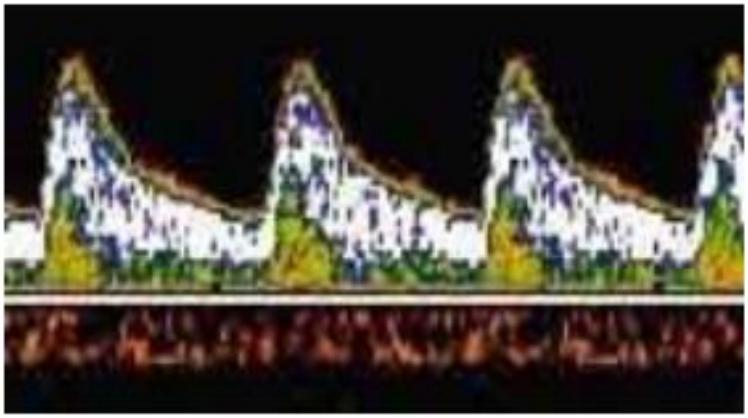


FIGURE 9: Normal impedance to flow in the umbilical arteries and umbilical vein in late 2° and 3° trimester

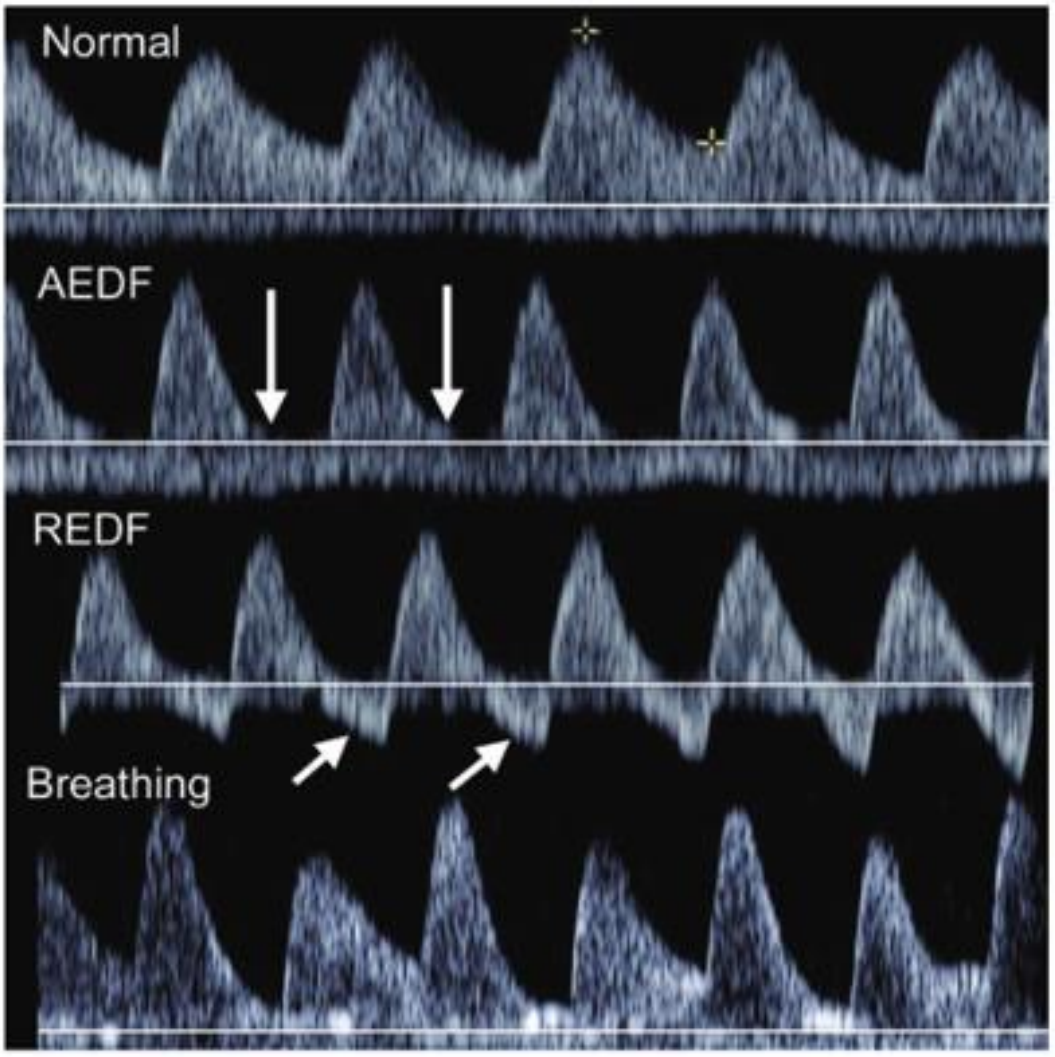


FIGURE 10: Umbilical artery waveforms. Top to bottom: Spectral US images show a normal waveform, absent end-diastolic flow (AEDF) waveform, reversed end-diastolic flow (REDF) wave-form, and breathing waveform. The normal waveform in the third trimester is low resistance, with continuous forward flow throughout diastole. The S/D ratio was 1.96 (calipers mark the peak and end-systolic velocities), and respiratory undulations are visible in the associated umbilical vein waveform. When there is absent end-diastolic flow (long arrows), the waveform smoothly tapers all the way to baseline, and there is no forward flow at the end of diastole. Respiratory variation is also depicted in umbilical vein in this example. With reversed end-diastolic flow (short arrows), flow in the umbilical artery is reversed in diastole and thus is displayed below the baseline. Not only is there lack of antegrade flow in diastole, but also the blood is actually pushed back out of the placenta owing to the high intraplacental pressure from obliteration of chorionic villi. Flow in the umbilical vein is not depicted on this spectral US image. Fetal breathing changes the intrathoracic pressure, resulting in variable systolic and diastolic flow but never absent or reversed end-diastolic flow. This breathing waveform is from a normal fetus.

MIDDLE CEREBRAL ARTERY DOPPLER:

The internal carotid artery, common carotid artery, or the anterior & middle cerebral arteries can all be examined during pregnancy to examine the cerebral blood flow.[73,74]

Pulsed wave Doppler evaluation of the fetal cerebral circulation has been described by Waldimiroff and others. Studies of fetal cerebral circulation have focused on fetal internal carotid artery as it enters Willis circle or MCA as it leaves circle.

Doppler has effective in identifying hypoxia linked to IUGR. The MCA flow velocity is typically very pulsatile, and as gestation progresses, the frequency of observable EDF rises (75% at 18 weeks and 100% at 34 weeks). All of the main cerebral arteries PI are markedly lower in fetuses with IUGR. It is believed that a hormonally induced increase in fetal peripheral vascular resistance takes place in order to preserve blood flow to brain, heart & adrenal glands. As result of these phenomena, "brain sparing effect" and "cardiac sparing effect" are produced, preserving functionality of these significant components. It is a marker of impending brain damage in the brain and may prevent prenatal hypoxia.[62]

The strongest individual predictors of a adverse fetal outcome are the MCA and UA Dopplers, respectively. However, utilizing the ratio of these two values, superior outcomes have been attained. Infants with IUGR were more likely than controls to be born prematurely, have lower birth weights, require emergency caesarean sections for fetal distress, have higher rates of prenatal and neonatal mortality, have low 5-min Apgar scores, and be admitted to a NICU due to asphyxia. Additionally, it can be used to predict very severe fetal anemia.[75-77] While an abnormal umbilical/MCA needs close fetal monitoring. Impedance to flow in the fetal MCA may be reduced in post-term pregnancies with unfavorable outcomes.[51] These arteries have limited diastolic flow in healthy foetuses, and the overall systolic diastolic ratio is typically > 4.

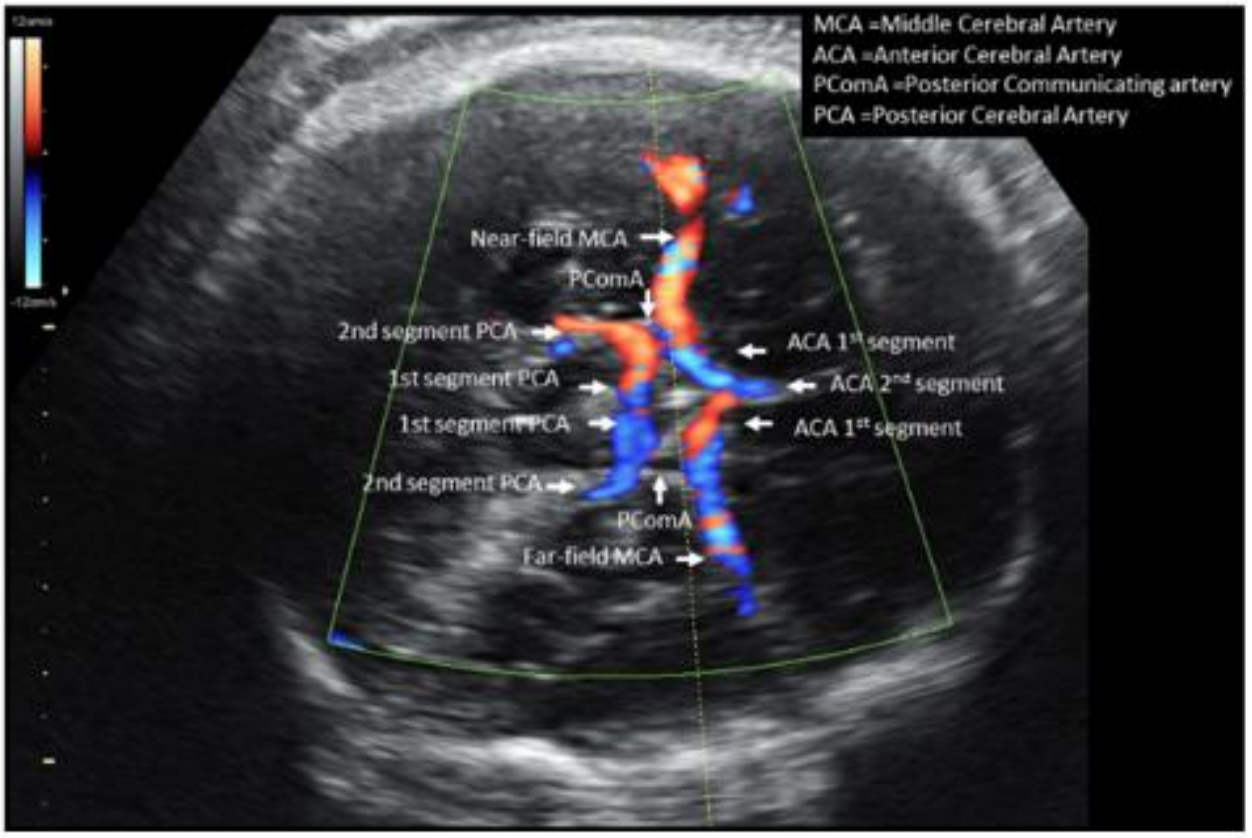


FIGURE 11: Color flow mapping of the circle of Willis

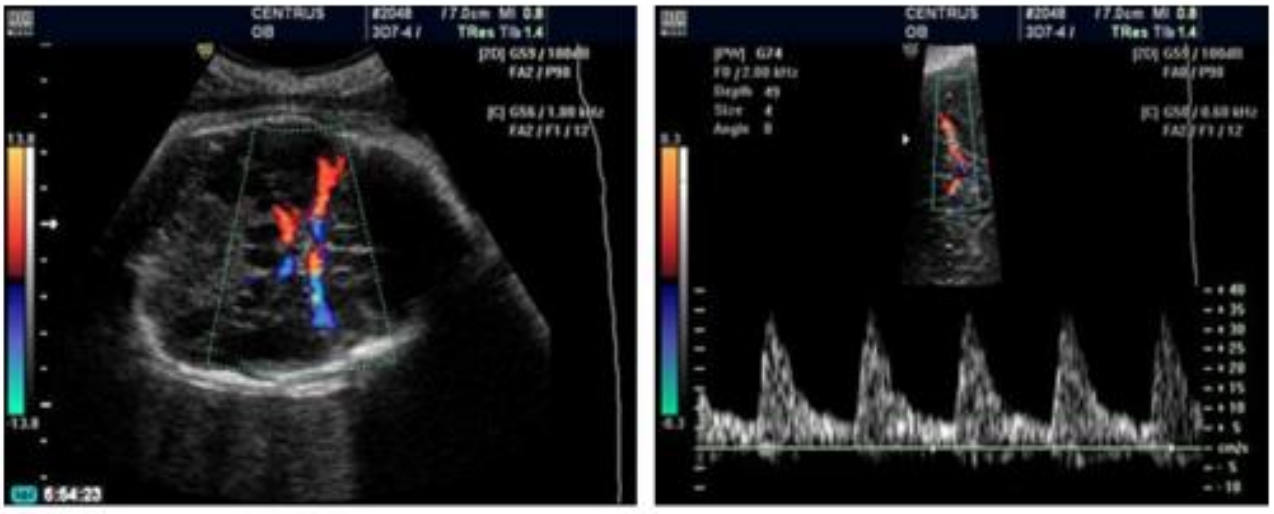


FIGURE 12: Transverse view of the fetal head with color Doppler showing the circle of Willis (left). Flow velocity waveforms from the middle cerebral artery at 32 weeks of gestation (right).

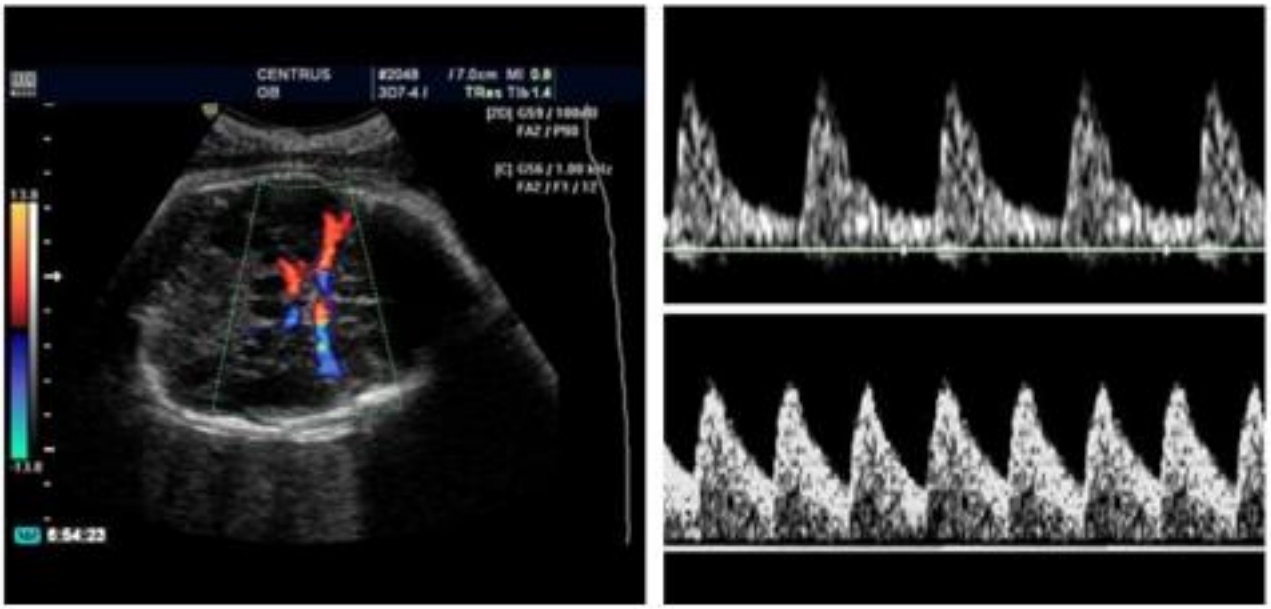


FIGURE 13: Color Doppler examination of the circle of Willis (left). Flow velocity waveforms from the middle cerebral artery in a normal fetus with low diastolic velocities (right, top) and in a growth-restricted fetus with high diastolic velocities (right, bottom).

UTERINE ARTERY DOPPLER:

First-trimester screening for early-onset PE and other unfavorable outcomes, like FGR, is done using a UTA Doppler.[78] Doppler US of the UTA may be utilized in second and third trimesters to assess pregnancies with FGR.

Branch of internal iliac artery known as the uterine artery travels anteriorly via the pelvis to junction of uterine corpus & cervix (ie, the cervicoisthmic junction), where it enters myometrium. Waveform has strong resistance, low diastolic flow and early diastolic notching in non-pregnant state. Successful placentation requires remodelling of UTA branches, which is demonstrated by a change in waveform to low resistance with continuous diastolic flow. This placental blood flow recruitment occurs quickly; notching should vanish by 13 weeks of gestation,[79] and low-resistance flow should be achieved by 20 weeks at earliest.[88] Increased resistance and the persistence of a diastolic notch through late second trimester are signs of an aberrant waveform. It is believed that a diastolic notch, which decrease in forward flow at the beginning of diastole, represents aberrant uteroplacental flow. Abnormal consequences, such as FGR, maternal preeclampsia, an increased risk of premature delivery, and fetal distress during labour, have been linked to a diastolic notch.[80-83]

Lower lateral quadrant of the abdomen is where transducer is positioned, tilted medially. The uterine artery can be seen running anteriorly and appearing to cross external iliac artery using color Doppler flow ultrasound. Aim for as close to 0° as you can given the vessel's orientation, which allows for a low angle of insonation of typically 15°–30°. Unlike the UA, umbilical vein, and MCA waveforms, the uterine artery waveform is remains unaffected by fetal activity. Samples can be taken during a mother's regular breathing cycle. Although many Doppler US characteristics can be measured in a research environment, the presence of a diastolic notch is the most significant finding.

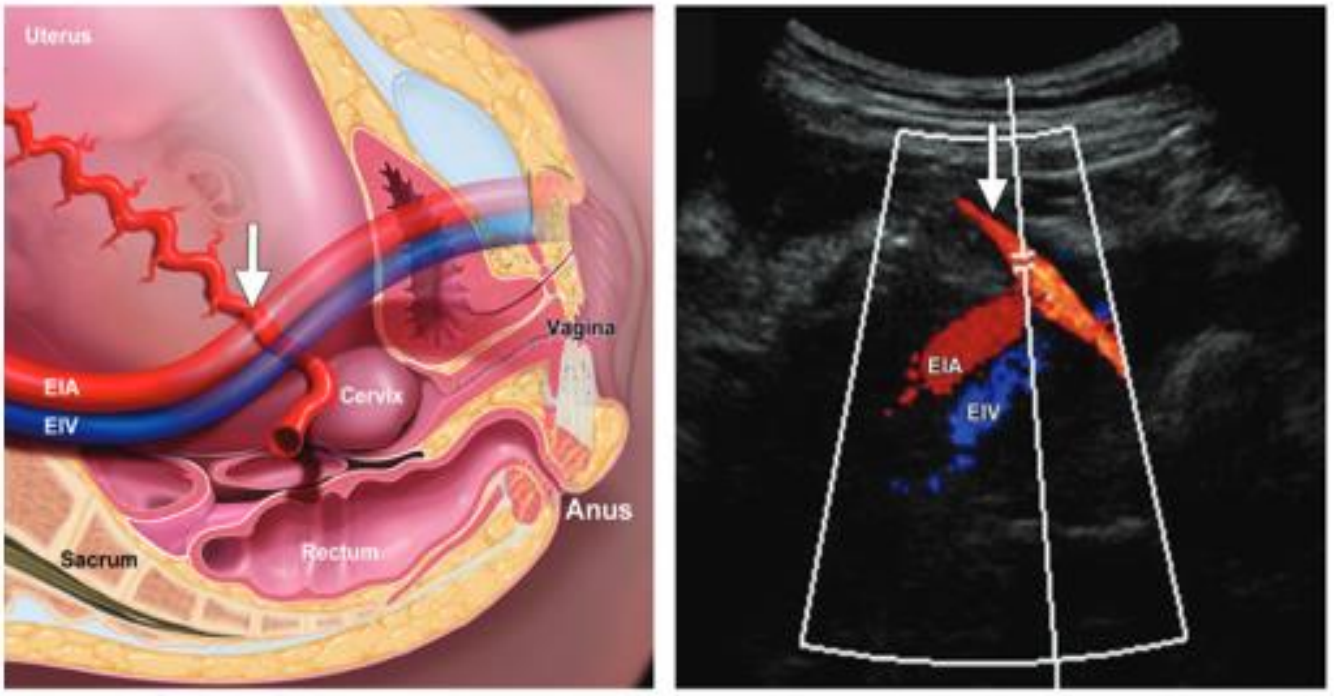


FIGURE 14: Doppler US technique for the uterine artery. EIA= external iliac artery, EIV= external iliac vein. (a)Anatomic drawing of a third-trimester pregnancy shows the location of the uterine artery (arrow) at the cervicoisthmic junction. Although the lower uterine segment elongates and the fundal height increases, the location of the cervico-isthmic junction does not change in pregnancy. (b) Color Doppler flow US image shows the sample volume placed in the uterine artery (arrow) with an angle of insonation of about 30°. The uterine artery perfuses the myometrium, so flow is toward the transducer, because the uterine artery is a branch of the internal iliac artery deep in the maternal pelvis.

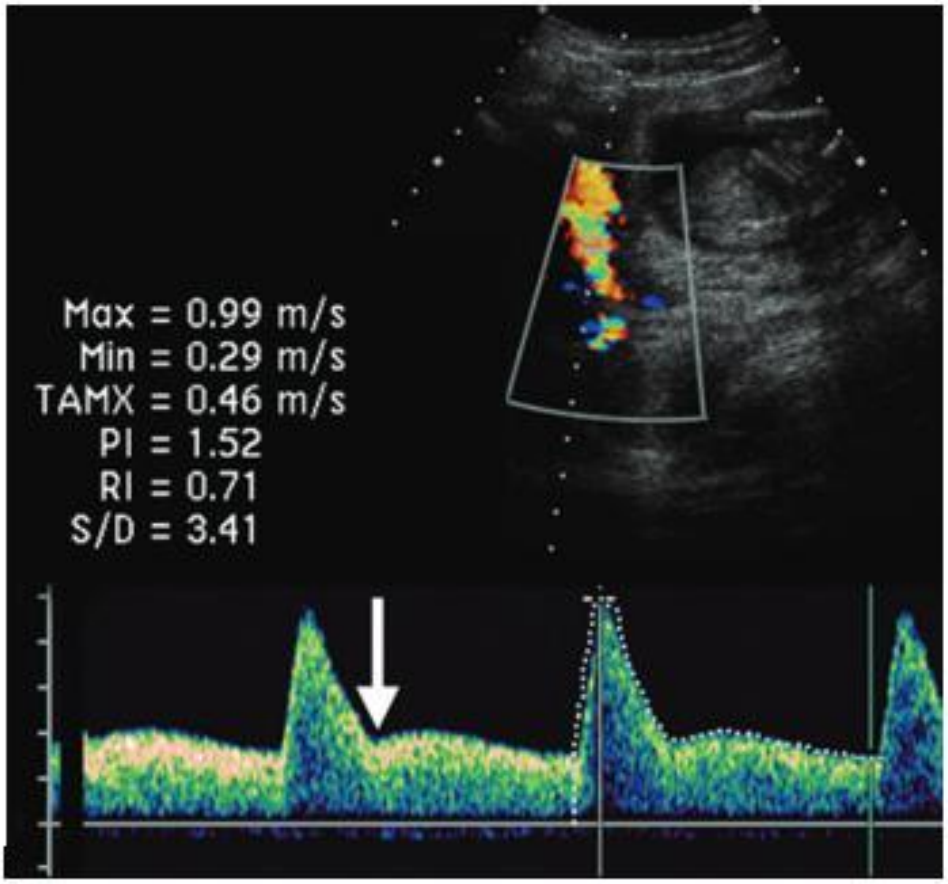


FIGURE 15: Color Doppler duplex US image shows an abnormal waveform with a mild notch (arrow) in diastole but preserved end diastolic flow. Max= maximum (same as peak systolic velocity), Min= minimum (same as end-diastolic velocity), PI= pulsatility index, RI= resistance index, TAMX= time-averaged maximum velocity

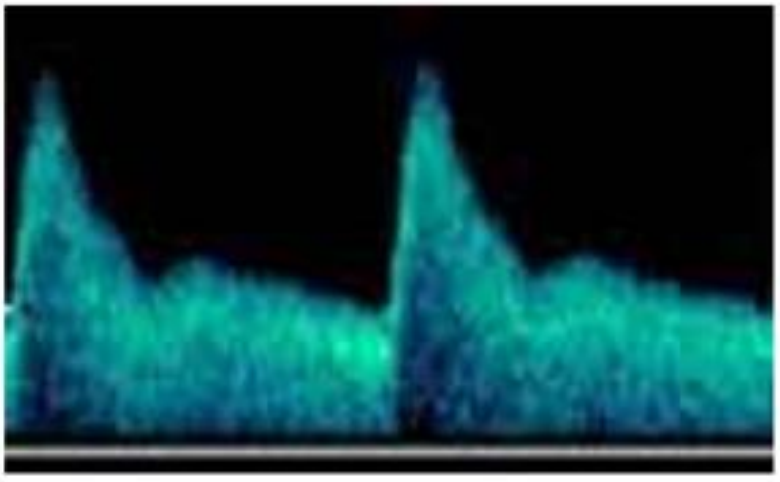


FIGURE 16: Normal impedance to flow in the uterine arteries (with the characteristic waveform of early diastolic notching)

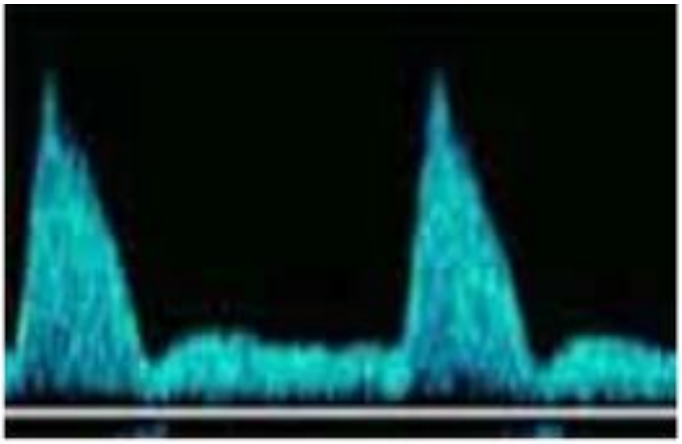


FIGURE 17: Increased impedance to flow in the uterine arteries (with the characteristic waveform of early diastolic notching)

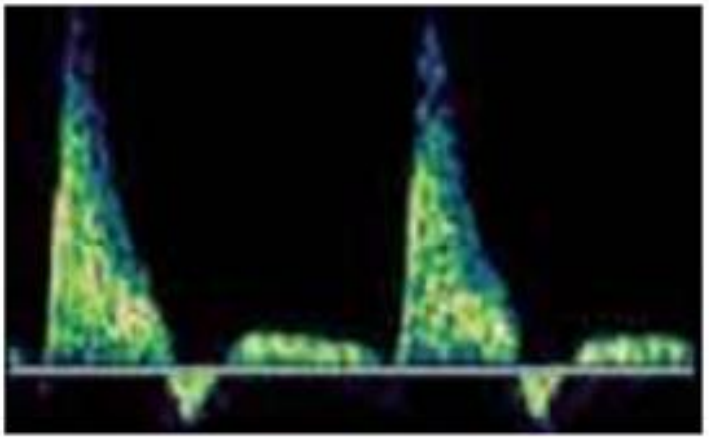


FIGURE 18: Very high resistance to flow in uterine arteries (with reverse diastolic flow

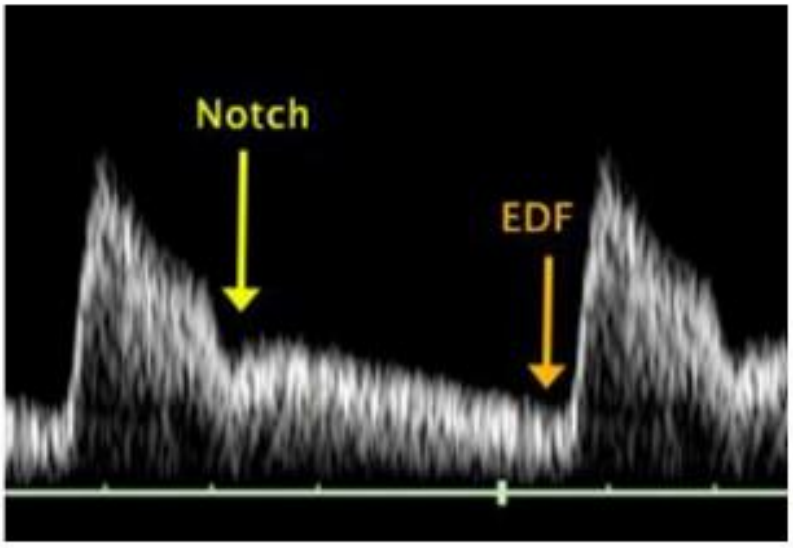


FIGURE 19: Flow velocity waveform from uterine artery at 24 weeks of gestation in a pregnancy with impaired placentation; in early diastole there is a notch (yellow arrow) and in late diastole there is decreased flow (orange arrow).

To investigate relationship & diagnostic efficacy of UA, MCA, UTA PI & cerebroplacental ratio (CP ratio) in identifying adverse outcome in singleton pregnancies at term. D'Antonio F et al. conducted a study in 2020. The Doppler results were hidden from participating practitioners. Singleton pregnancies between 36+0 and 37+6 weeks of gestation met inclusion criteria. A composite score of unfavorable outcome served as the main outcome. Data analysis techniques employed included ROC curve analysis and logistic regression. He stated that, "total of 600 consecutive singleton pregnancies starting at 36 weeks of gestation were examined. Mean UTA PI (0.8 0.2 vs 0.7 0.3, P = 0.001) was higher in pregnancies experiencing than in those not experiencing composite unfavorable outcome, but mean MCA PI (1.1 0.2 vs 1.5 0.4, P 0.001) and cerebroplacental ratio (1.4 0.4 vs 1.9 0.6, P 0.001) were lower".

The estimated fetal weight centile (P = 0.712) and UA PI, on the other hand, did not differ between two groups. However, AC was lower in fetuses that had a composite unfavorable outcome (45.4 vs. 53.2, P = 0.040). MCA PI, UTA PI, abdominal circumference centile, and gestational age at birth were all associated with adverse outcomes (odds ratio [OR] 0.1, 95% CI 0.01-.2, P = 0.001, OR 1.4, 95% CI 1.2-1.6, P = 0.001, and OR 1.6, 95% CI 1.2-2.1, P = 0.004, respectively). Despite this, Doppler has a low diagnostic accuracy in predicting a negative pregnancy outcome at term. The authors concluded that cerebroplacental ratio and MCA PI are linked to poor perinatal outcome at term. Their use as a stand-alone screening test for a bad outcome in singleton pregnancies at term is not recommended, because of their low predictive accuracy of perinatal compromise.[84]

High-risk can lead to a numerous negative outcome. In light of this, Kavitha G et al. (2019) carried out a retrospective record-based study to examine role of color doppler USG in efficient management of high-risk pregnancies at Department of Obstetrics and Gynecology. The study comprised antenatal records of women in age range of 20 to 30 years who were

carrying singletons at a gestational age of 26 weeks or longer and had one or more high-risk characteristics. Risk factors considered include DM, anemia, oligohydramnios, and PIH. UA Doppler investigation was conducted. Findings of study revealed that OUT of 140 cases, age ranged between 20 to 25 years was one in which high-risk pregnancies were most prevalent. PIH, which was responsible for 50% of cases and was frequent high-risk factor in pregnancy. 40 cases out of 140 high-risk pregnancies had IUGR. The UA was aberrant in 43% of cases. The study showed that color Doppler can be utilized as most efficient method of fetal monitoring in cases of high-risk pregnancies. Significant benefit is early intervention and improved fetal outcome.[85]

Adverse perinatal outcome is associated with PIH. In these situations, multi-vessel colour Doppler tests are helpful for prompt intervention. The current study's objectives were to determine the importance of UA, MCA and UTA Doppler tests in PIH and to evaluate their contribution in prediction perinatal outcome. **Gaikwad PR et al. (2017)** conducted a prospective study using this paradigm on 106 singleton pregnancies with PIH that were in the third trimester. Within a week of delivery, results of most recent Doppler ultrasound were analyzed. Study of adverse prenatal outcomes included perinatal mortality, meconium-stained amniotic fluid, Apgar at 5 minutes < 7, NICU hospitalization, and emergency caesarean sections for fetal distress (stillbirths and neonatal death). After comparing several Doppler parameters to the standard, sensitivity, specificity, PPV, negative predictive value (NPV), and diagnostic accuracy were obtained.

According to study, all Doppler ultrasonography measures had high specificity and diagnostic accuracy when it came to predicting unfavorable neonatal outcomes. In predicting a adverse neonatal outcome, the CP ratio had the best specificity (98.55%), PPV (94.44%), and diagnostic accuracy (80.19%), outperforming the MCA PI and UA PI separately. UTA Doppler

analysis provides additional data in predicting worse prenatal outcomes. According to the study's findings, the CP ratio (MCA/UA PI) is the strongest predictor of a poor neonatal outcome among the several Doppler measures.[86]

The prospective study by **Gaikwad PR et al. (2018)** sought to determine value of color Doppler scans in cases of IUGR and to correlate with perinatal outcome in order to provide improved techniques for early detection of compromised fetuses & prompt intervention. The study comprised 125 singleton pregnancies with IUGR that were in the third trimester. Within a week of delivery, the results of the most recent Doppler ultrasound were analyzed. After comparing the results with the standard, the sensitivity, specificity, PPV, NPV and diagnostic accuracy of various Doppler parameters were computed. According to the study's findings, 63 patients exhibited at least one undesirable perinatal outcome parameter. In predicting a poor perinatal outcome, UA diagnostic accuracy was higher (71.20%) than that of other indicators. In comparison to other parameters, MCA RI had the best specificity and 100% PPV for predicting a poor outcome. Patients with AEDF and REDF had perinatal mortality rates of 33.3% and 50%, respectively. Based on the study results the authors concluded that MCA Doppler studies showed more specificity and PPV than UA Doppler in prediction of adverse fatal outcome.[87]

In a randomized control trial study, **Aparna G. and Suvarna V. (2018)** examined USG Doppler flow patterns in high-risk pregnancies & contrasted them with those in normal pregnancies. The study, which was conducted from April 2016 to November 2017, had 100 pregnant ladies—50 in study group and 50 in control group. IUGR, preeclampsia, other hypertensive disorders of pregnancy and GDM were among high-risk conditions that led to the selection of 50 women for study group. In contrast, 50 women with normal pregnancies were chosen for control group & their accurate GA was determined by 2nd trimester scan. Color Doppler flow in the MCA and UA of the patients was evaluated for fetal welfare. The results showed that from 28 to 36 weeks of gestation, the A/B ratio, RI and PI values in MCA steadily decreased in comparison to the

study group, with a slight increase at 34 to 36 weeks. Pre-eclampsia accounted for 90% of cases, GDM 6%, and PIH with IUGR in 45 instances. In comparison to the research group, the mean birth weight at various gestational stages was significantly lower. Misoprostol was the preffered technique of induction between 28 and 32 weeks of pregnancy, while elective caesarean sections were the preferred way of delivery beyond 36 weeks. Total 8% were stillbirths and 92% live births were recorded. The authors showed that non-invasive technique, such as colour Doppler velocitometery, is important tool for evaluating fetal wellbeing.[88]

Accuracy of Color Doppler in identifying FGR was studied by **Chakarvarty N et al (2018)** using the waveforms and color flow of the UA, UTA, and MCA. For the objective of this study, total of 100 clinically suspected FGR subjects were taken. USG examination by Color Doppler was performed every 3 weeks beginning at 30 weeks till delivery. To determine proper cut-off points for FGR prediction, receiver-operator curve analysis was performed. The UTA RI was discovered to be the most effective in early identification; it had 84.6% sensitivity, 82.9 % specificity and 84% diagnostic accuracy at 30-week interval. At 30 weeks, UTA PI demonstrated 76.9% sensitivity, 82.9% specificity, and 79% diagnostic accuracy. When compared to UA S/D ratio, which was shown to be 70.8% sensitive and 65.7% specific with a diagnosis accuracy of 69% at a 33-week interval, UA RI was found to be 80% sensitive and 74.3% specific. Compared to MCA PI, which had 66.2% sensitivity and 68.6% specificity over a 36-week interval, MCA RI had just 60% sensitivity and 71.4% specificity. MCA PSV was shown to be an ineffective tool because it failed to distinguish significantly between two groups. According to the study, uterine artery doppler readings were more effective than UA and MCA results.

Even at an early stage (30 weeks), UA RI was found to be the most effective. The results of this study indicated that Color Doppler results are significant for detecting FGR even at the early stages (30 weeks).[89]

MATERIAL & METHODS

MATERIALS AND METHODS:

Study site:

This study was done in Department of Radio-diagnosis at R.L Jalappa Hospital and Research center attached to SDUMC, Kolar.

Study population:

All eligible pregnant women who would have undergone USG & color Doppler at Department of Radio-diagnosis at R.L Jalappa Hospital and Research center were regarded as study population.

Study design: Prospective cohort study.

Sample size:

Richa Choudhary et al 13 had reported that sensitivity of MCA PI in detecting adverse perinatal outcomes to be 75%.

Assuming alpha error = 0.05 (95% Confidence Limit), absolute precision of 10%, and 20% loss of follow up, the sample size of 87 subjects will be included.

The sample size was derived from the following formula:

$$\text{Sample size (n)} = \frac{Z^2(P*Q)}{d^2} \text{where;}$$

Z is the value for Confidence Interval.

D is the absolute precision.

P is the expected sensitivity and

q=1- p.

Master software version 2.0 was used to calculate sample size.

Sampling method:

Using simple sampling, all qualified participants were successively enrolled in the study until the sample size was reached.

Study duration:

Between January 2021 and July 2022 (18 months), data was collected for the study.

Inclusion Criteria:

1. Singleton pregnancy.
2. Pregnant women beyond 28 weeks of gestation deemed by investigators to be at high risk like pre-eclampsia, eclampsia & GDM.

Exclusion criteria:

1. Multiple pregnancies.
2. Pregnancies with congenital anomalies.

High risk factors considered:

1. Pre-eclampsia
2. Eclampsia
3. Gestational diabetes mellitus

Abnormal fetal outcome:

The fetal outcome was considered abnormal when any one or a combination of the following was present.

1. Low/high birth weight
2. Respiratory distress
3. Low APGAR score
4. NICU admission after delivery
5. Intrauterine death

ETHICAL CONSIDERATIONS:

Institution's human ethics committee approved this study. All participants were provided with written informed consent, and only those willing to sign the consent were allowed to take part in the study. Before getting consent, the participants were informed about risks and advantages of study as well as voluntary nature of participation. Privacy of study participants was protected at all times.

DATA COLLECTION TOOLS:

87 high risk pregnancy patients beyond 28 weeks of gestation visiting or getting admitted under the department of Obstetrics and Gynaecology and was referred to the department of Radio-Diagnosis for ultrasonography and color Doppler, R. L. Jalappa Hospital and Research Center, attached to Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar were included in the study. Their willingness to participate in the study was obtained by written informed consent. Along with important clinical history, pertinent lab tests, and baseline data from the patients was gathered.

METHODOLOGY:

Detailed history was taken from the patients meeting the inclusion criteria and referred to department of radio diagnosis. Patients were subjected to ultrasonographic examination and Doppler study. The equipment used is PHILIPS EPIQ 5G system with continuous wave, pulsed wave, HPR Doppler with dual sector transducer. 2D real-time ultrasound scanning was done utilizing a C5-1 MHz convex sector transducer. Gestational age was analyzed by biparietal diameter (BPD), abdominal circumference (AC), head circumference (HC) and femoral length (FL). Placental position and grading were noted. Estimated fetal weight (EFW), amniotic fluid index (AFI) was calculated & BPP scoring was done.

Doppler study was done and the findings of doppler indices in these highrisk pregnancies were documented and compared with standard.[91] This was correlated with fetal outcome.

STATISTICAL METHODS:

SPSS 22 version of software was used to analyse the data, which was entered into a Microsoft Excel data sheet. Data was categorical displayed as frequencies and proportions. Mean and standard deviation were used to depict continuous data. As a test of significance for qualitative data, the **chi-square test or Fischer's exact test** (for 2x2 tables only) were also employed.

Sensitivity: Defined as ability of a test to identify correctly all those who have the disease i.e. true positive.

Sensitivity = a/(a+c) x 100 = True positive / True positive + False Negative

Specificity: It is the ability of test to identify correctly those who do not have the disease i.e. true negative.

Specificity = d/(b+d) x 100 = True Negative / True Negative + False Postive

Positive predictive value (PPV): The proportion of patients who test positive who actually have the disease.

Positive predictive value = a/ (a+b) x 100 = True Postive / True positive + False Postive

Negative predictive value (NPV): The proportion of patients who test negative who are actually free of the disease.

Negative predictive value = d/ (c+d) x 100 = True Negative / True Negative + False Negative.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs

P value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

RESULTS

OBSERVATIONS & RESULTS

Total of 90 subjects were included in final analysis.

Table 4: Descriptive analysis of age among study population (N= 90)

Age	Frequency	Percentage
<20yrs	15	16.7
21-25yrs	39	43.3
26-30yrs	26	28.9
>30yrs	10	11.1
Total	90	100.0

The mean age was 25.4 years, ranged between 18 to 37 years in the study population.

Figure 20 : Bar chart showing distribution of age in the study population (N=90)

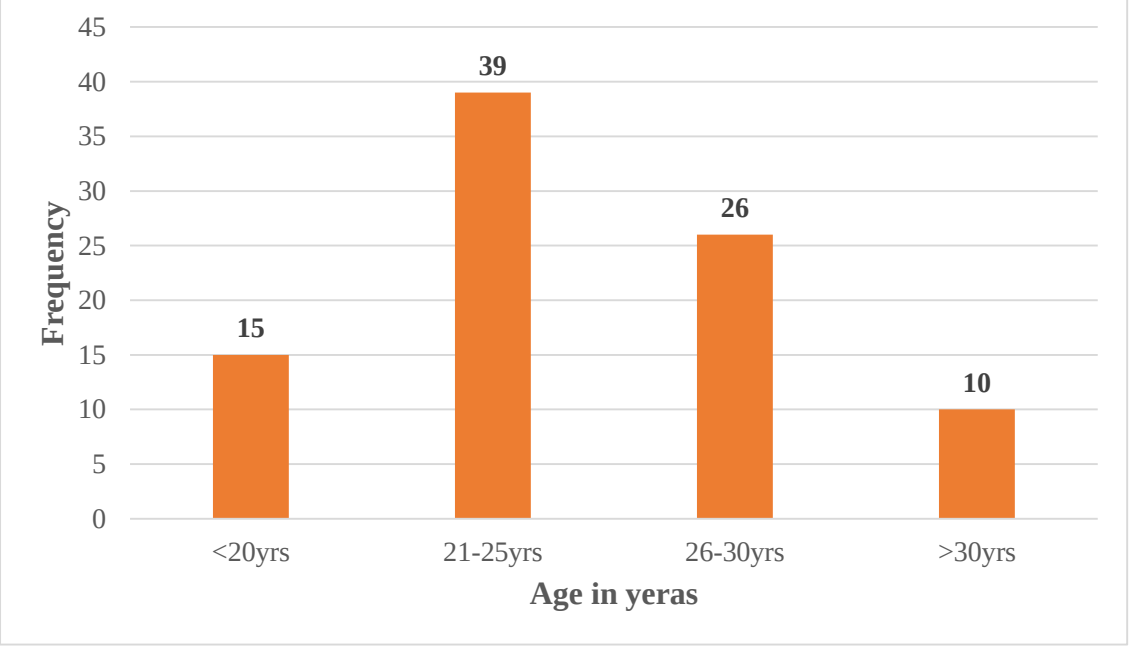
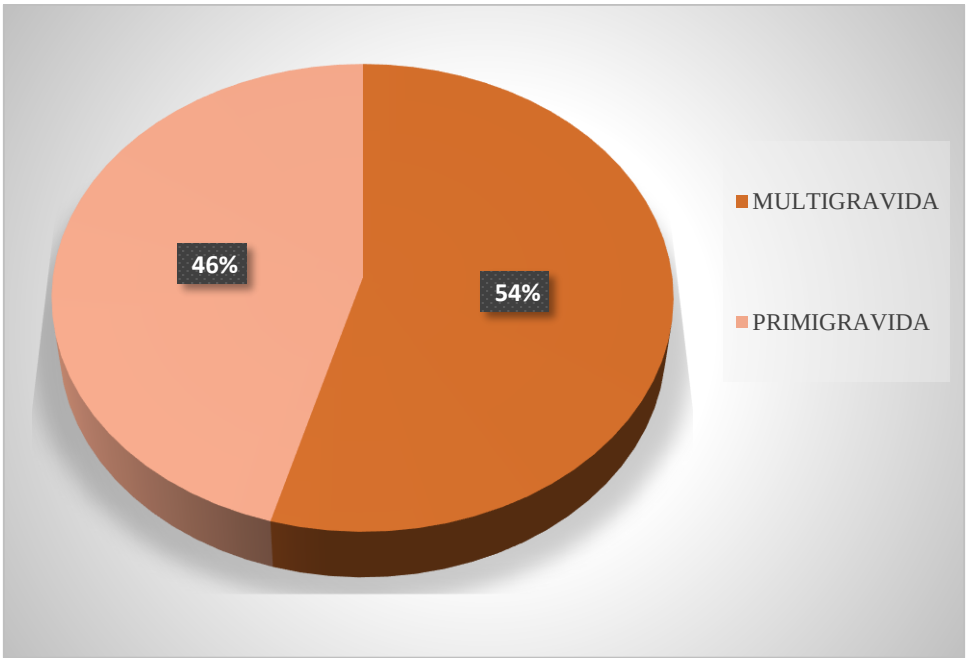


Table 5:- Distribution of subjects according to parity (N=90)

Parameters	Frequency	Percentage
Primigravida	41	45.6
Multigravida	49	54.4
Total	**90**	**100.0**

Figure 21:- Pie chart showing distribution of subjects according to parity (N=90)



Among the subjects (N=90), 41 (45.6%) participants were primigravida, and 49 (54.4%) were multigravida.

Table 6:- Distribution of subjects according to high risk pregnancies (N=90).

High risk pregnancies	Frequency	Percentage
Preeclampsia without severe features	27	30.0
Pre-eclampsia with severe features	18	20.0
Eclampsia	24	26.7
GDM	21	23.3
Total	90	100.0

Among the study population, 27 (30.0 %) participants had Preeclampsia without severe features, 18 (20.0 %) participants had preeclampsia with severe features, 24 (26.7 %) had eclampsia and 21 (23.3%) had GDM.

Figure 22:- Bar chart showing distribution of subjects according to high risk pregnancies (N=90).

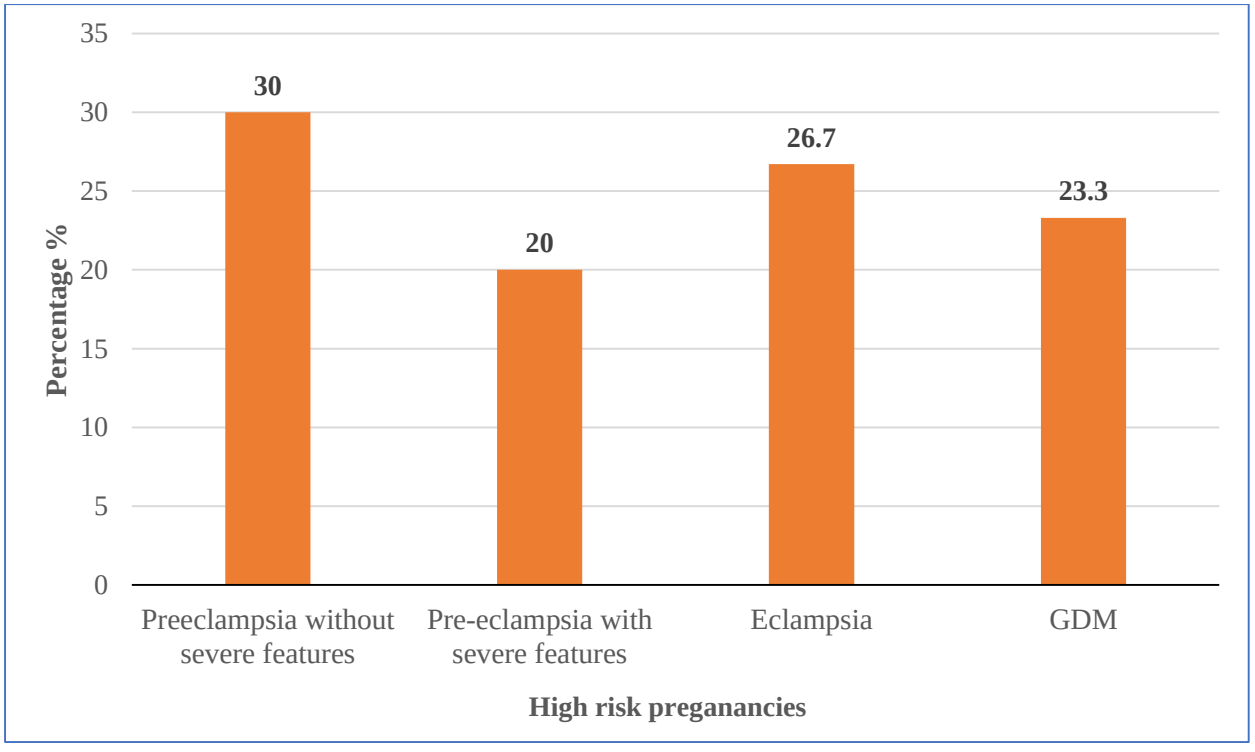


Table 7:- Distribution of subjects according to gestational age (N=90).

Gestational Age	Frequency	Percentage
28-32weeks	10	11.1
32-36weeks	25	27.8
>36weeks	55	61.1
Total	90	100.0

Among the study population, 10 (11.1 %) participants were between 28 – 32 weeks of gestation, 25 (27.8 %) participants were between 32 – 36 weeks of gestation and 55 (61.1 %) were > 36 weeks of gestation.

Figure 23:- Pie chart showing distribution of subjects according to gestational age (N=90)

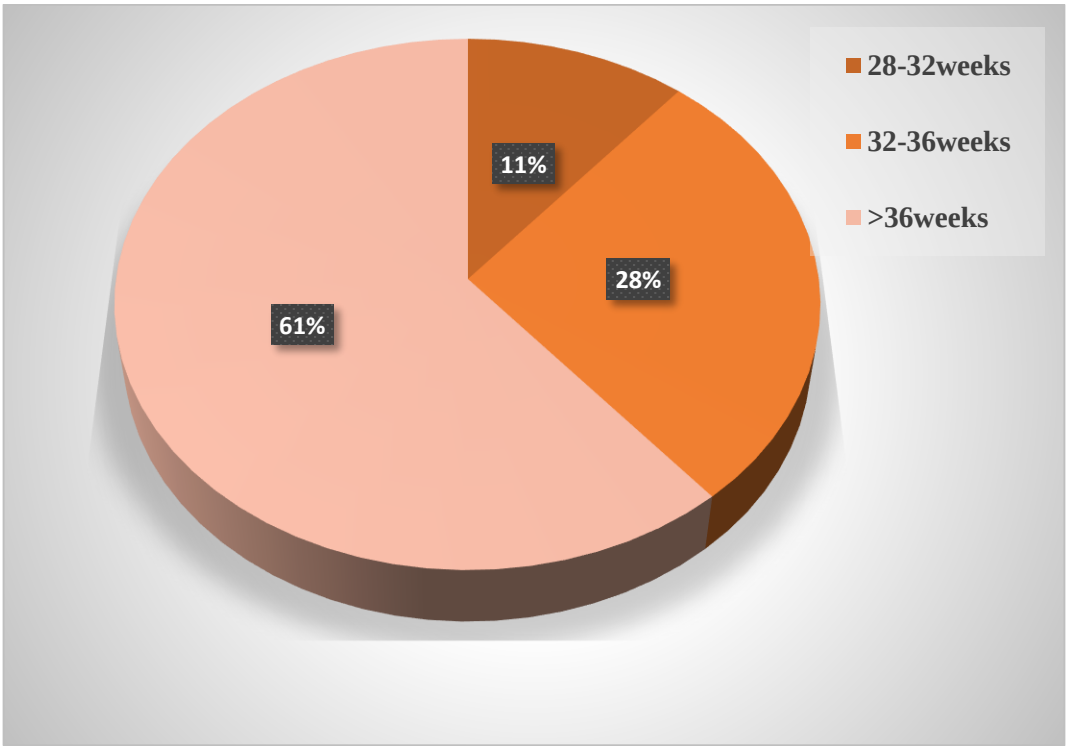


Table 8a:- Distribution of subjects according to amniotic fluid volume (N=90).

Amniotic fluid volume	Frequency	Percentage
Adequate	47	52.2
Oligohydramnios	37	41.1
Polyhydramnios	6	6.7
Total	90	100

Among the study population, 47 (52.2 %) participants had normal amniotic fluid volume, 37 (41.1 %) participants had oligohydramnios and 6 (6.7 %) participants had polyhydramnios.

Figure 24:- Pie chart showing distribution of subjects according to amniotic fluid volume (N=90).

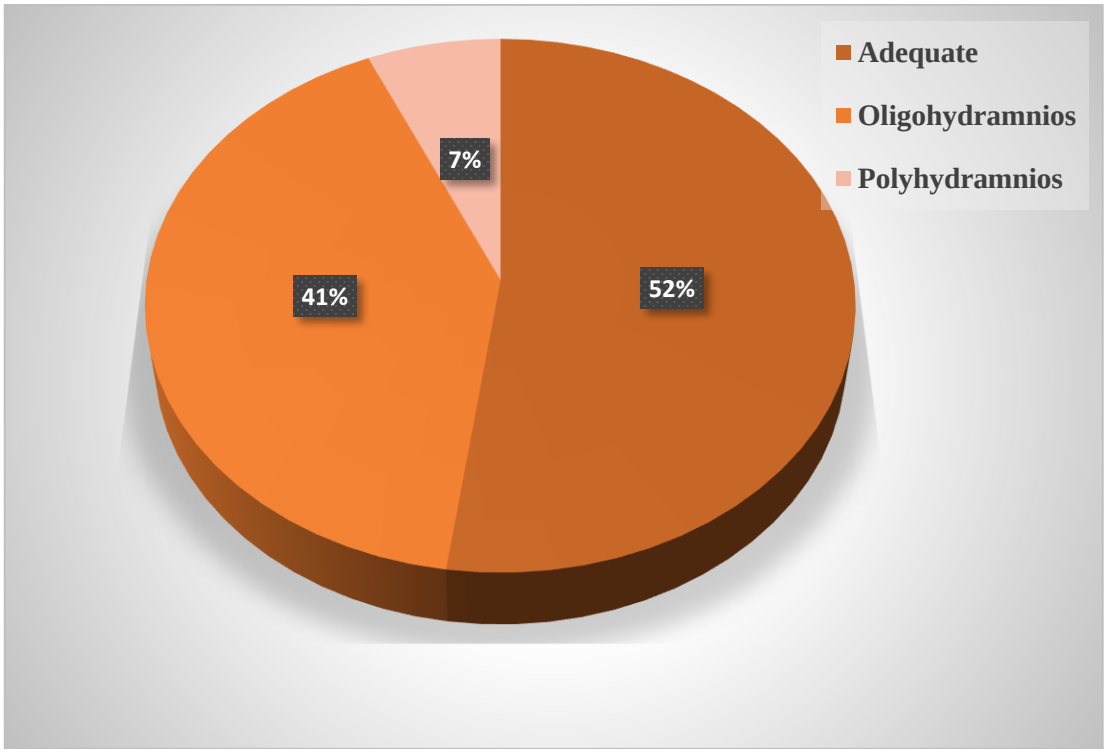


Table 8b:- Distribution of subjects according to amniotic fluid volume in each high risk pregnancies (N=90).

Amniotic fluid volume	Preeclampsia without severe features	Pre-eclampsia with severe features	Eclampsia	GDM
	N	N	N	N
Adequate	10	9	13	15
Oligohydramnios	17	9	11	0
Polyhydramnios	0	0	0	6

Table 9a:- Distribution of subjects according to Growth lag (N=90).

Growth lag	Frequency	Percentage
Absent	47	52.2
Present	43	47.8
Total	90	100.0

Among the study population, growth lag was present in 43 (47.8 %) participants.

Figure 25:- Pie chart showing distribution of subjects according to growth lag (N=90).

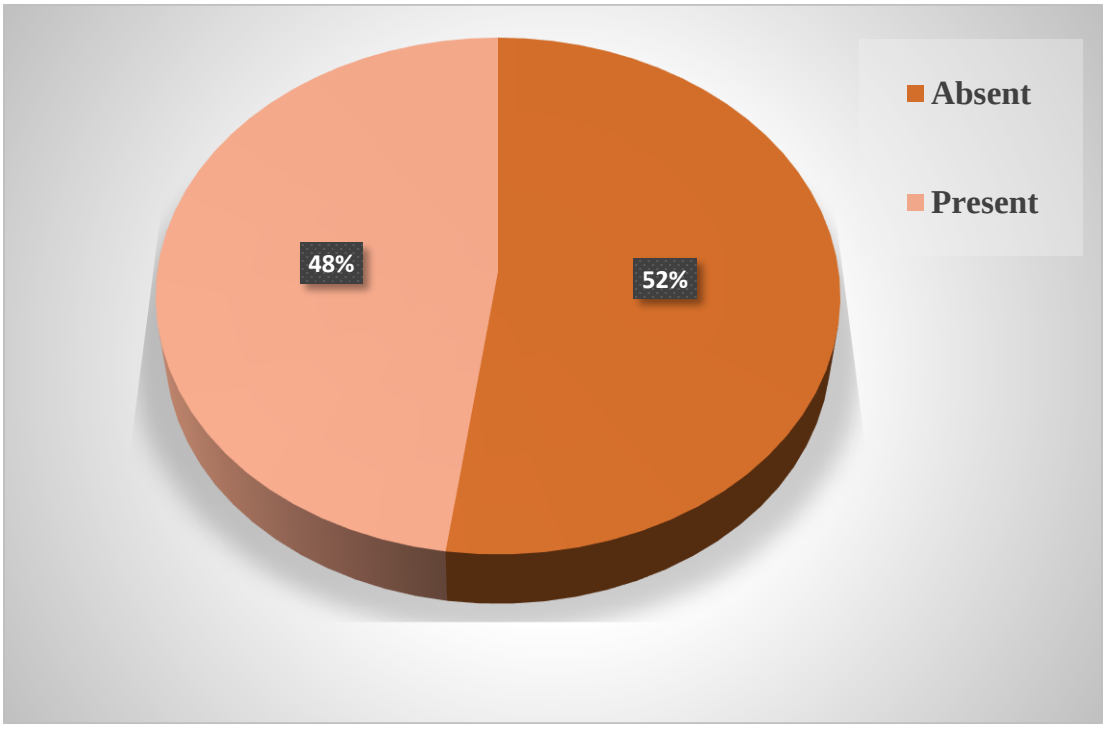


Figure 9b:- Table showing distribution of subjects according to growth Lag (N=90).

Growth lag	Preeclampsia without severe features	Pre-eclampsia with severe features	Eclampsia	GDM
	N	N	N	N
Absent	8	7	11	21
Present	19	11	13	0

Table 10:- Distribution of subjects according to HC/AC Ratio (N=90).

HC/AC Ratio	Frequency	Percentage
Normal	71	78.9
Increased	19	21.1
Decreased	-	-
Total	90	100.0

Among the study population, HC/AC ratio was increased in 19 (21.1 %) participants which indicates that these participants had asymmetrical intrauterine growth restriction.

Figure 26:- Pie chart showing Distribution of subjects according to HC/AC Ratio (N=90).

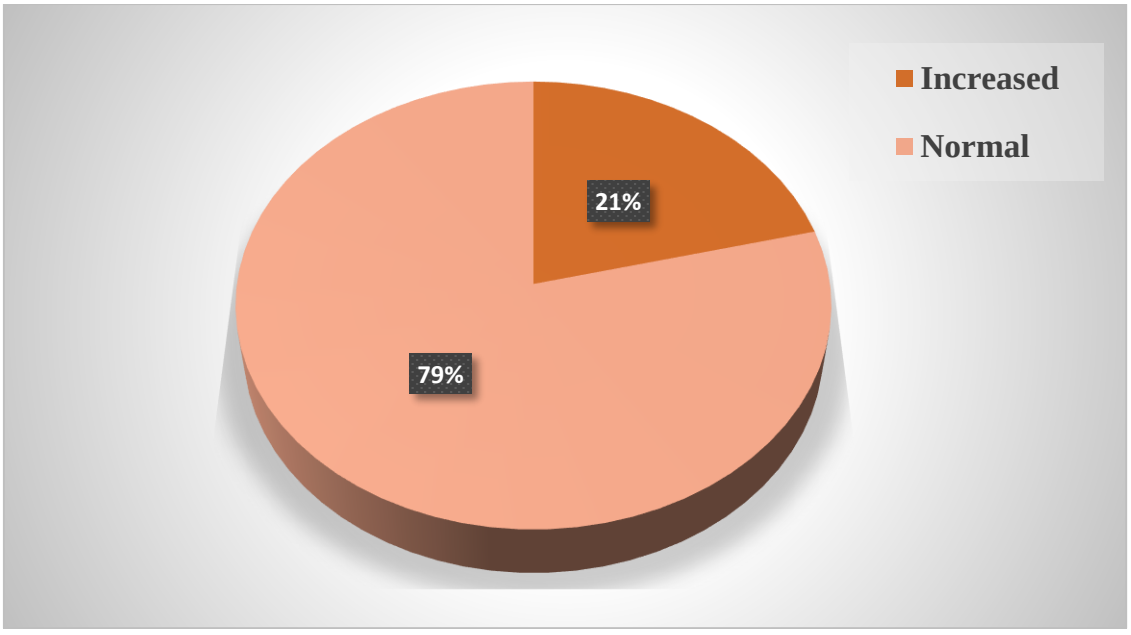


Table 11:- Distribution of subjects according to MCA RI and MCA PI (N=90).

	MCA PI		MCA RI	
	Frequency	**Percent**	**Frequency**	**Percent**
Normal	48	53.3	62	68.9
Abnormal	42	46.7	28	31.1
Total	90	100.0	90	100.0

Among study population, abnormal (below < 5th percentile) MRA PI was seen in 42 (46.7 %) subjects and abnormal (below < 5th percentile) MRA RI was seen in 28 (68.9 %) subjects.

Figure 27:- Bar graph showing distribution of subjects according to MCA RI and MCA PI (N=90).

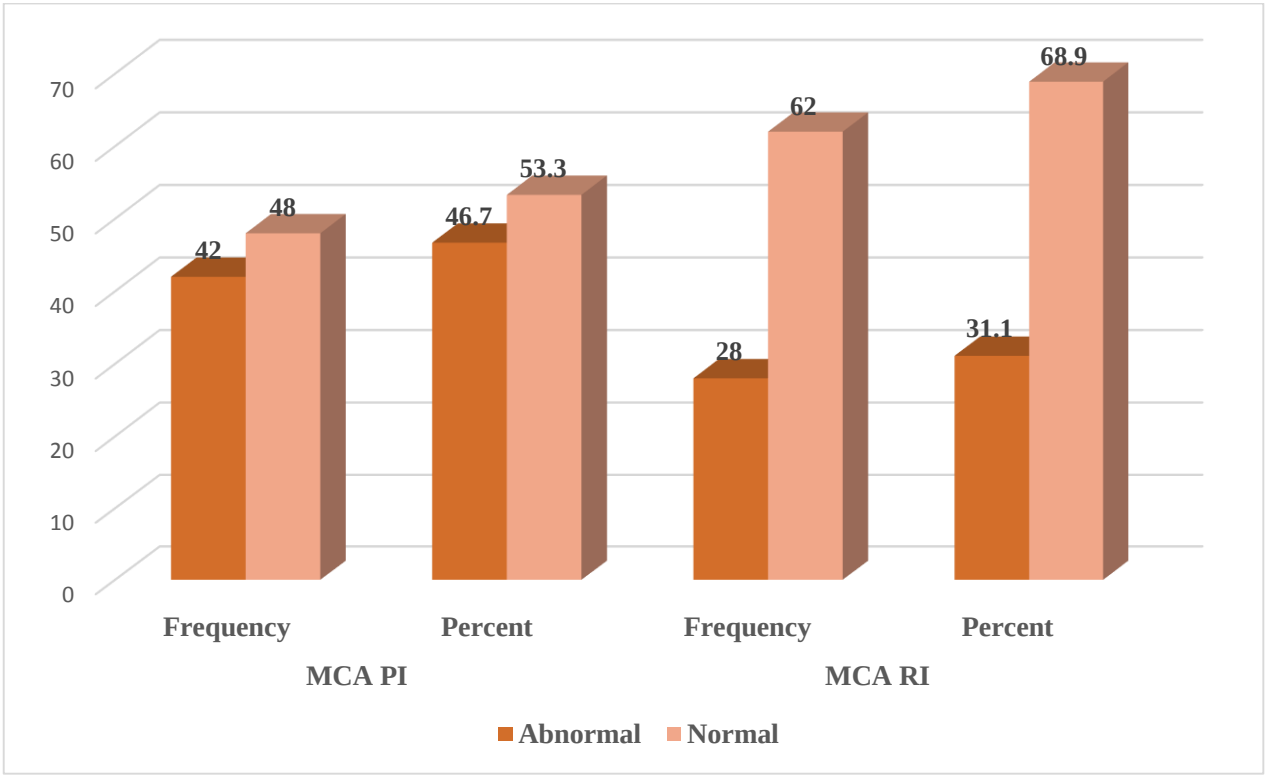


Table 12:- Distribution of subjects according to UA PI and UA RI (N=90)

	UA PI		UA RI	
	Frequency	**Percentage**	**Frequency**	**Percentage**
Normal	34	37.8	47	52.2
Abnormal	56	62.2	43	47.8
Total	90	100.0	90	100.0

Among study population, abnormal (above > 95th percentile) UA PI was seen in 56 (62.2 %) subjects and abnormal (above > 95th percentile) UA RI 43 (68.9 %) subjects.

Figure 28:- Bar graph showing distribution of subjects according to UA PI and UA RI (N=90).

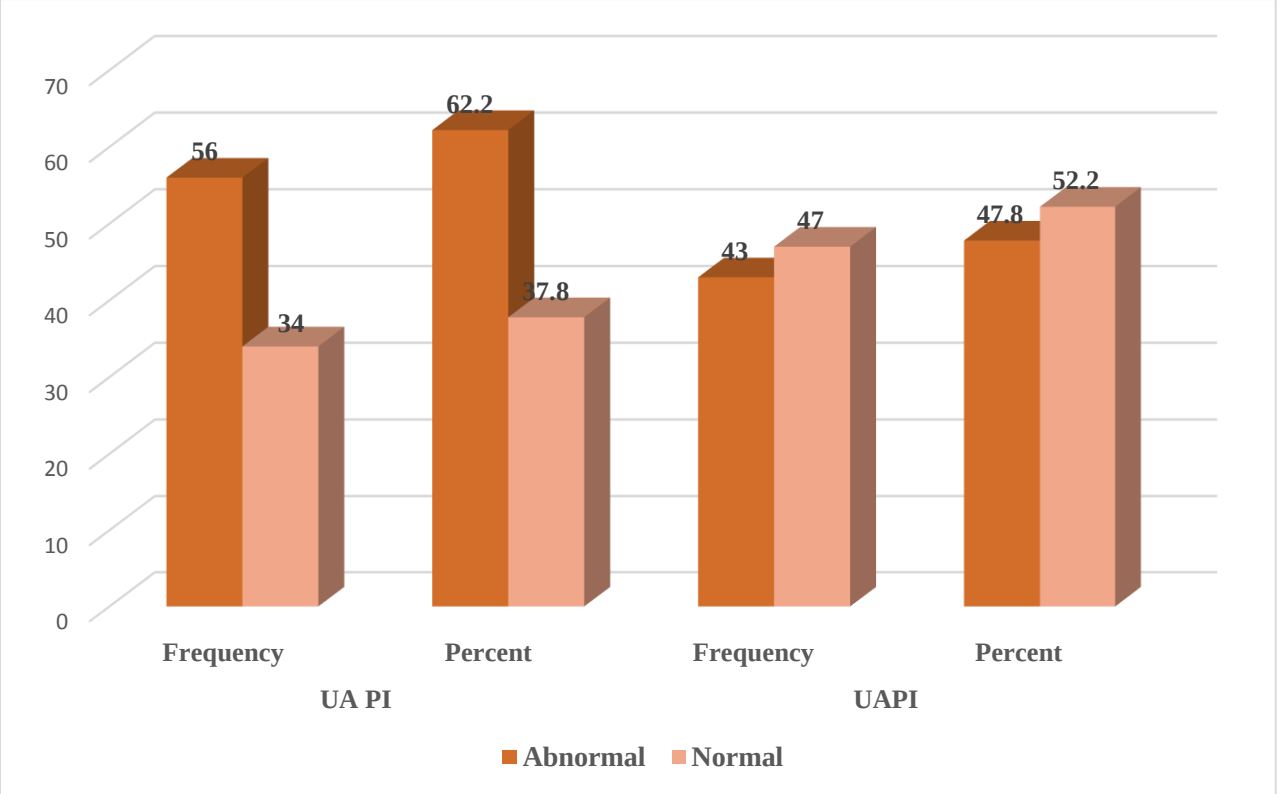


Table 13 a:- Distribution of subjects according to UA Flow patterns (N=90)

	Frequency	Percentage
Normal	40	44.4
Reduced	17	18.9
Absent	15	16.7
Reversal	18	20.0
Total	90	100.0

Among the study population, 40 (44.4 %) subjects had normal umbilical artery flow, 17 (18.9 %) subjects had reduced flow in umbilical artery, 15 (16.7 %) subjects had absent umbilical artery flow and 18 (20 %) subjects had reversal of flow in umbilical artery.

Figure 29:- Pie chart showing distribution of subjects according to UA Flow patterns (N=90)

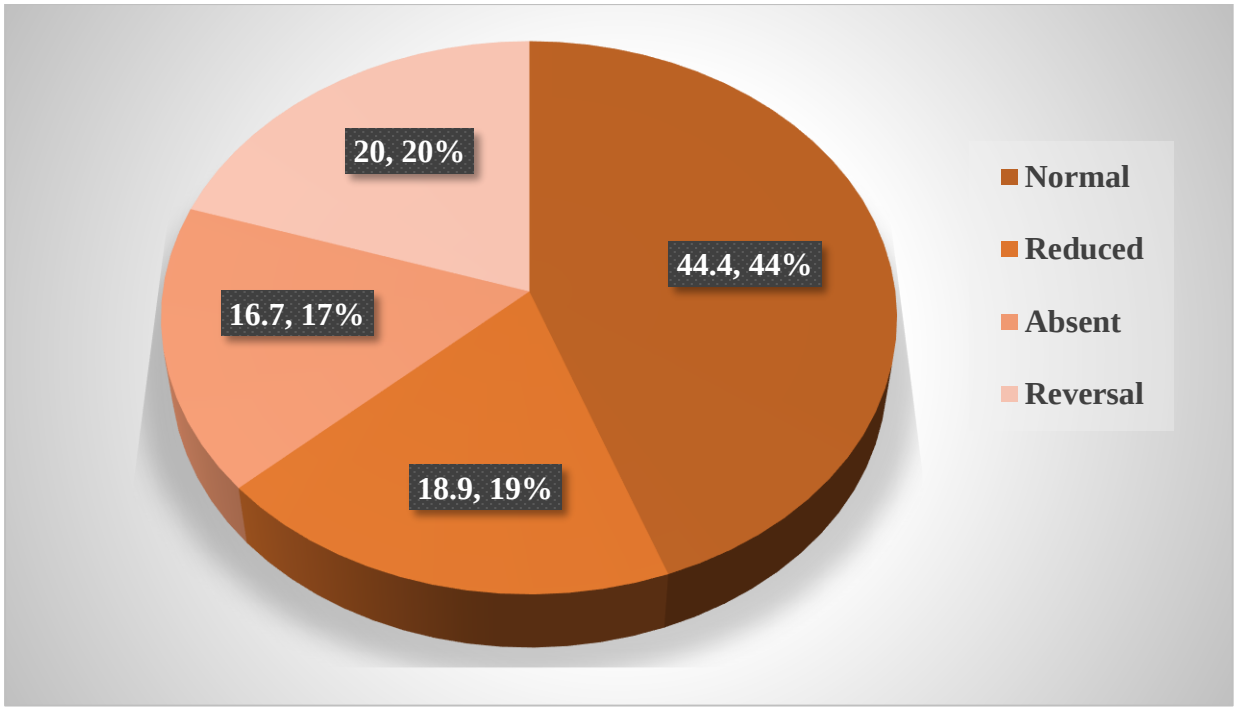


Table 13 b:- Distribution of subjects based on UA Flow patterns in high risk pregnancies (N=90)

UA flow patterns	Preeclampsia without severe features	Pre-eclampsia with severe features	Eclampsia	GDM
	N	N	N	N
Absent	5	3	7	0
Normal	9	5	5	21
Reduced	6	4	7	0
Reversal	7	6	5	0

Table 14:- Distribution of subjects according to mean UTA PI and UTA RI (N=90)

	UTA PI		UTA RI	
	Frequency	Percent	Frequency	Percent
Normal	43	47.8	45	50.0
Abnormal	47	52.2	45	50.0
Total	90	100.0	90	100.0

Among study population, abnormal (above > 95th percentile) UA PI was seen in 56 (62.2 %) subjects and abnormal (above > 95th percentile) UA RI (68.9 %) subjects.

Figure 30:- Bar graph showing distribution of subjects according to UTA RI and UTA PI (N=90).

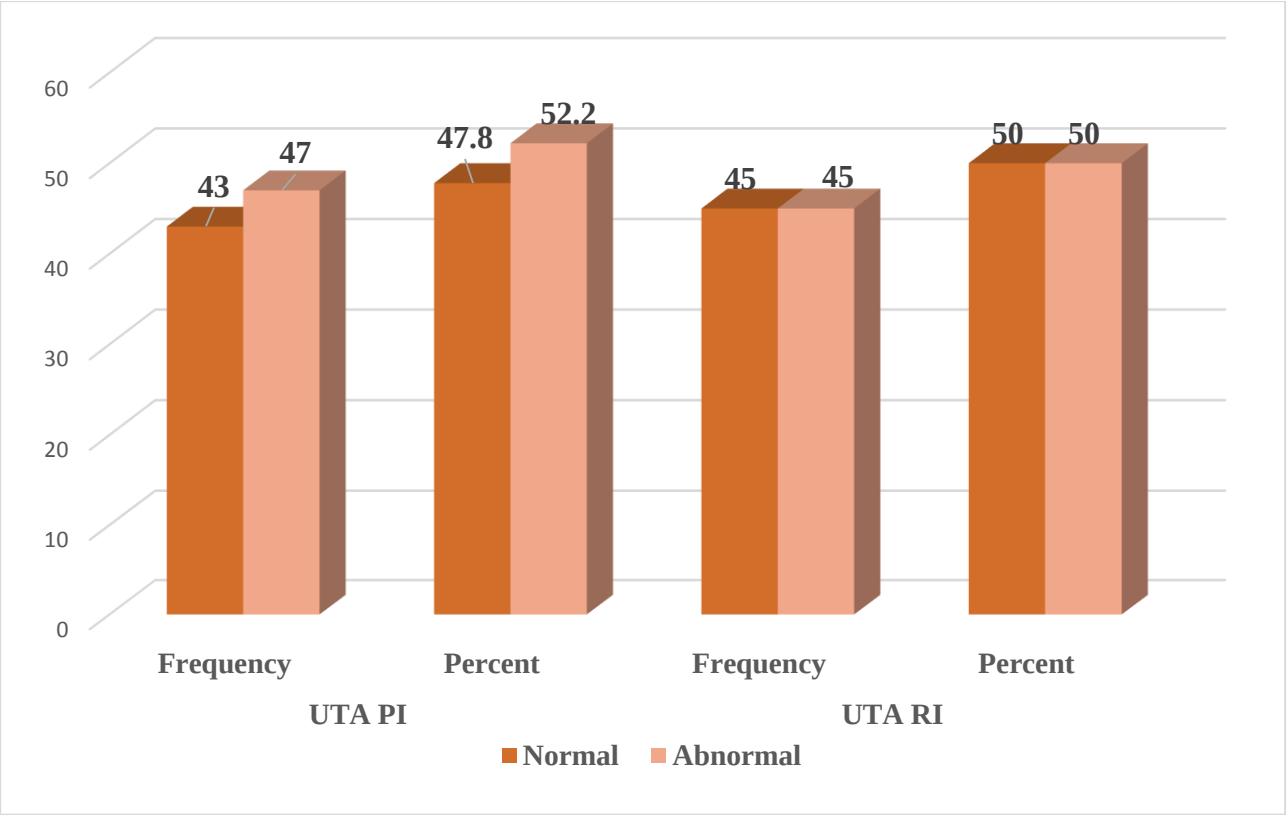


Table 15 a: Distribution of subjects according to flow in uterine artery (N=90).

	Frequency	Percentage
Normal	38	42.2
Early diastolic notching	52	57.8
Total	90	100

Among the study population, 38 (42.2 %) subjects had normal uterine artery flow and 52 (57.9 %) subjects had early diastolic notching in uterine arteries.

Figure 31: Pie chart showing distribution of subjects according to flow in uterine artery (N=90).

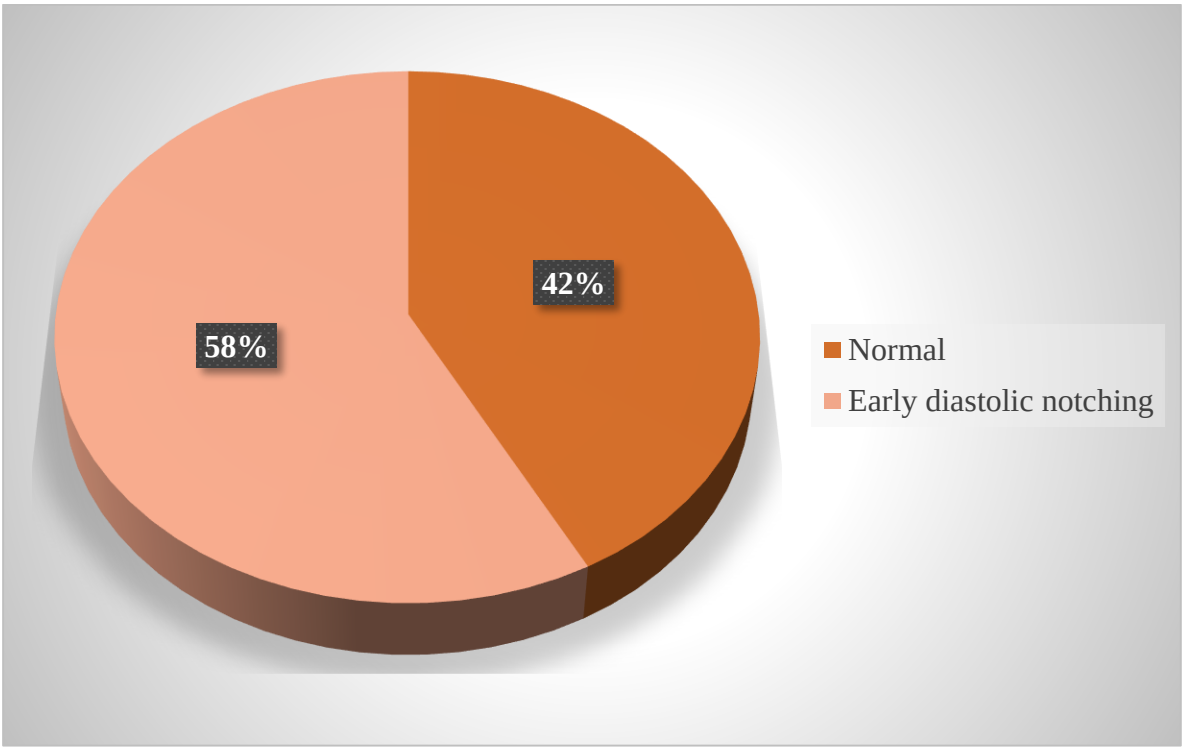


Table 15b: Distribution of subjects based on flow in uterine artery in high risk pregnancies (N=90).

High risk conditions	Early diastolic notching in uterine artery	
	Unilateral	Bilateral
Preeclampsia without severe features	10	12
Pre-eclampsia with severe features	4	8
Eclampsia	7	6
GDM	3	2

Table 16a:- Distribution of subjects according to CP Ratio (N=90)

	Frequency	Percentage
Normal	38	42.2
Reversal	52	57.8
Total	90	100.0

Among the subjects, 52 (57.8 %) subjects had reversal of CP ratio.

Figure 32:- Pie chart showing Distribution of subjects according to CP Ratio

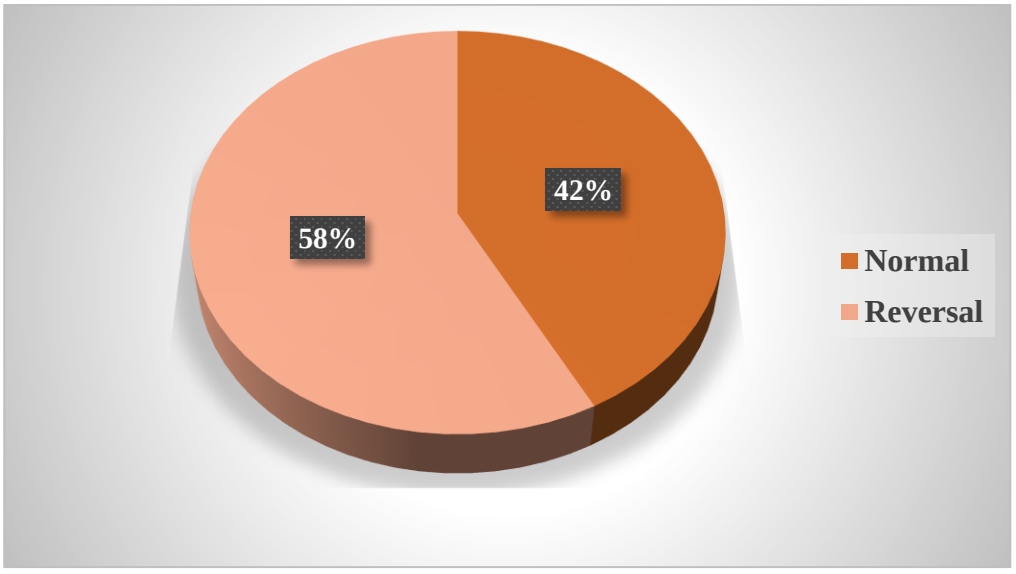


Table 16b:- Distribution of subjects according to CP Ratio (N=90)

	Preeclampsia without severe features	Pre-eclampsia with severe features	Eclampsia	GDM
	N	N	N	N
Normal	6	5	6	21
Reversal	21	13	18	0

Table 17:- Distribution of subjects according to Feto-placental insufficiency (N=90).

	Frequency	Percentage
Absent (Normal)	33	36.7
Present	57	63.3
Total	90	100.0

Among the subjects, feto-placental insufficiency was seen in 57 (63.3 %) of cases

Figure 33:- Pie chart showing Distribution of subjects according to Feto-placental insufficiency (N=90).

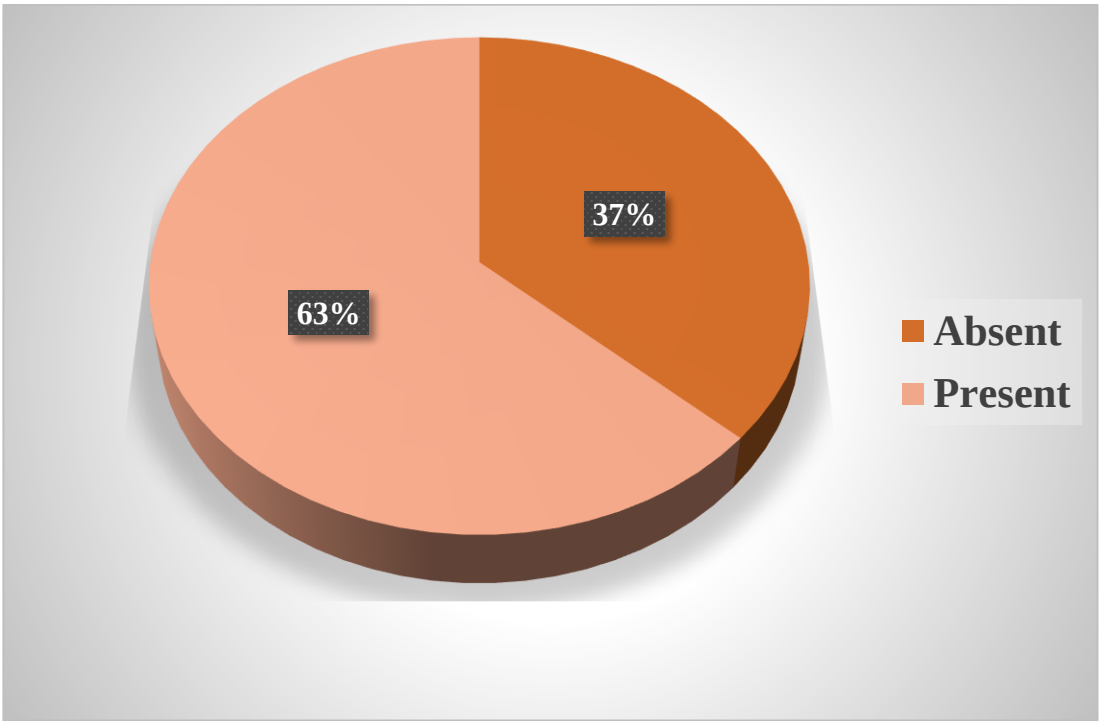


Table 18:- Distribution of subjects according to Utero-placental insufficiency (N=90).

	Frequency	Percentage
Absent	28	31.1
Present	62	68.9
Total	90	100.0

Among the subjects, utero-placental insufficiency was seen in 62 (68.9 %) of cases

Figure 34:- Pie chart showing Distribution of subjects according to Utero-placental insufficiency (N=90).

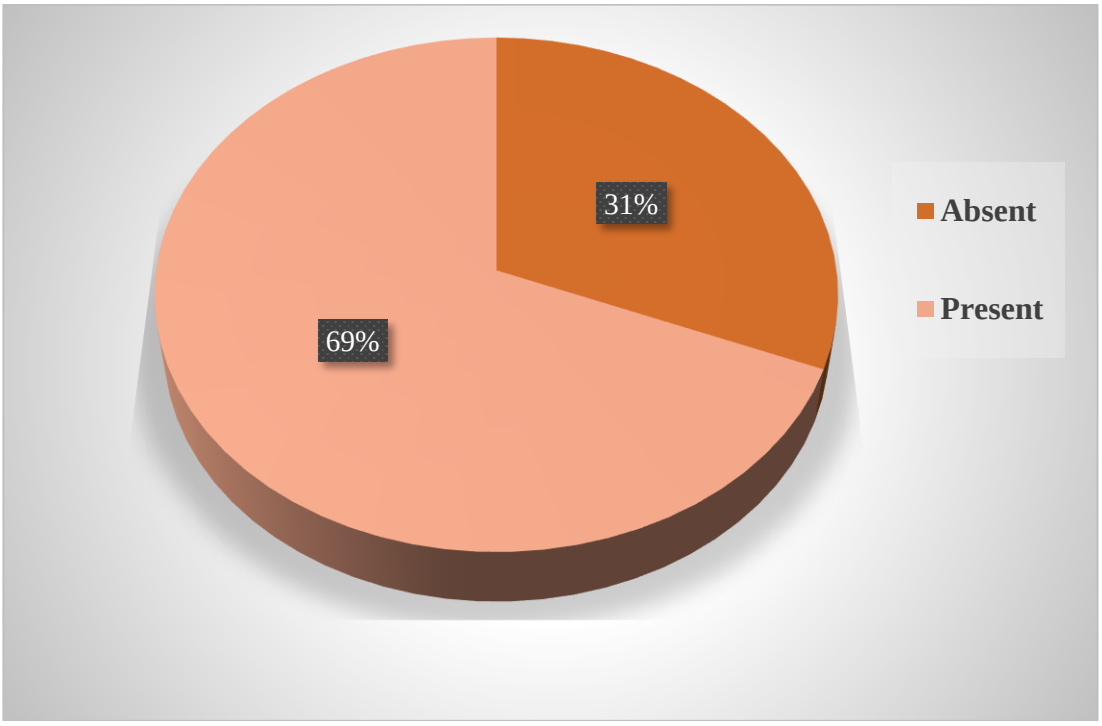


Table 19:- Distribution of subjects according to mode of delivery (N=90).

	Frequency	Percentage
NVD	31	34.4
LSCS	59	65.6
Total	90	100.0

Among the subjects, 31 subjects underwent normal vaginal delivery in 31 (34.4 %) and 59 (65.5 %) underwent lower segment cesarean section.

Figure 35:- Pie chart showing Distribution of subjects according to mode of delivery (N=90).

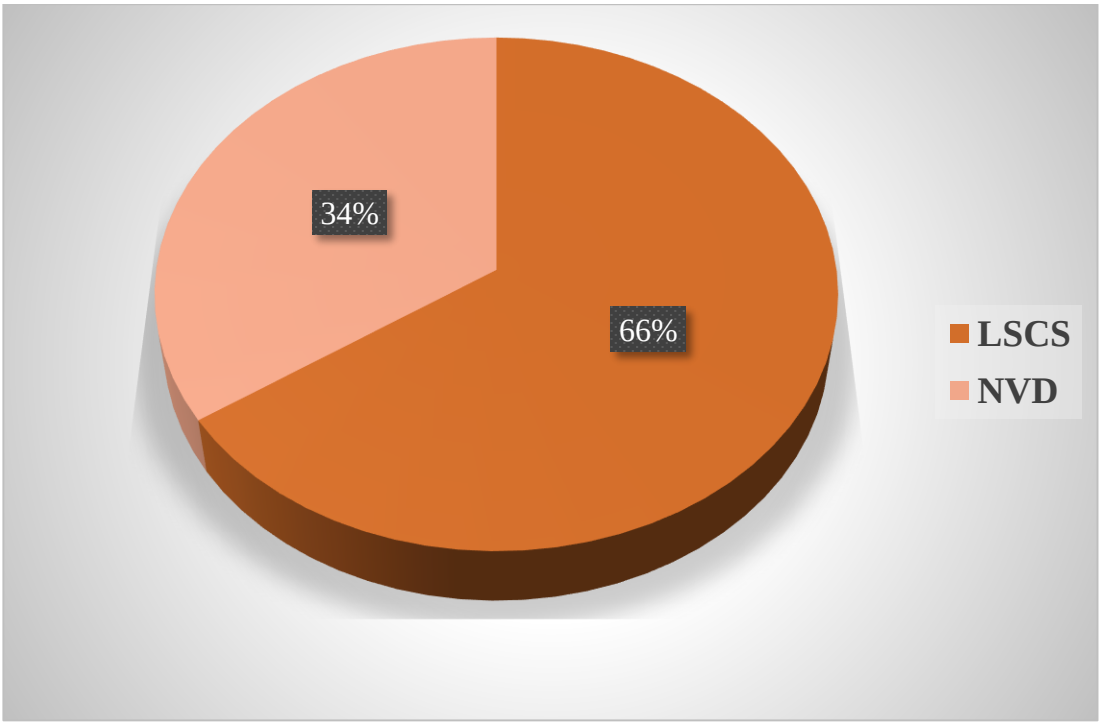


Table 20:- Distribution of subjects according to fetal outcome (N=90).

	Frequency	Percent
Uneventful	31	34.4
Adverse event	59	65.6
Total	90	100.0

Among the subjects, adverse fetal outcomes was seen in 59 (65.6 %) of subjects.

Figure 36:- Pie chart showing Distribution of subjects according to fetal outcome (N=90).

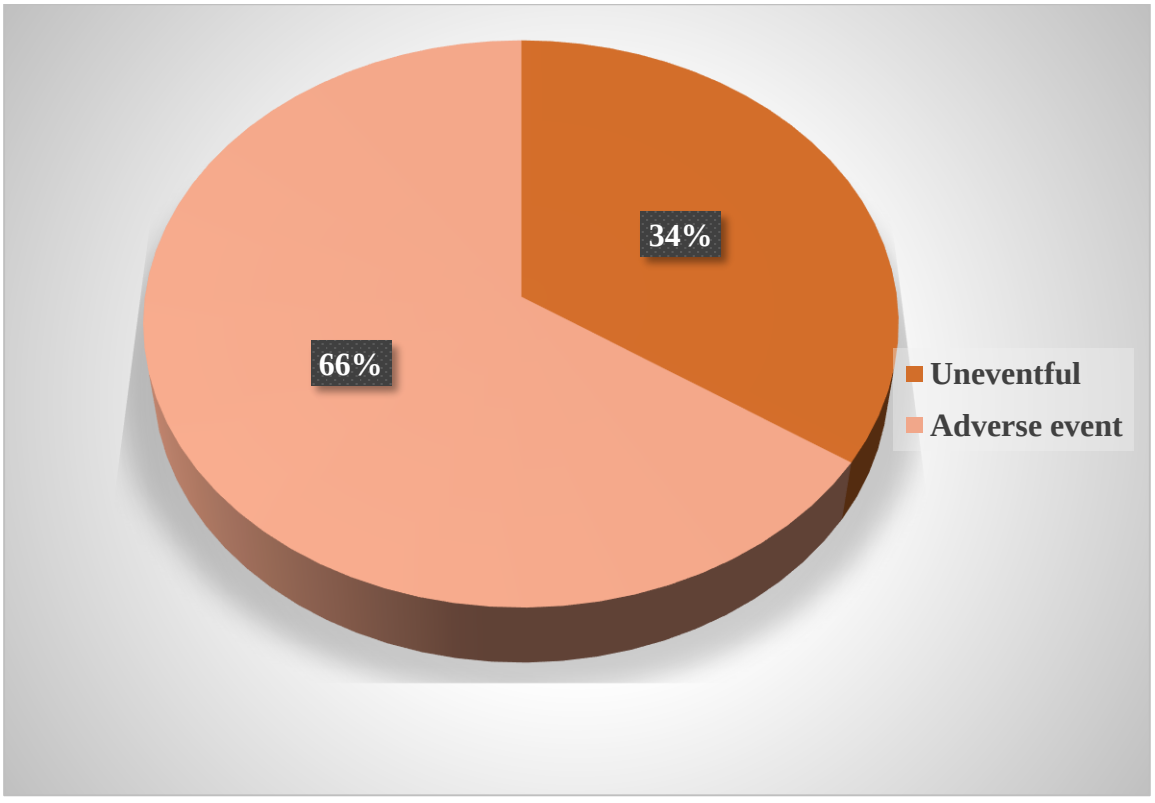


Table 21:- Distribution of subjects according to complication and outcome (N=90).

	Uneventful		Adverse event	
	N	%	N	%
Preeclampsia without severe features	8	29.6	19	70.3
Pre-eclampsia with severe features	4	22.2	14	77.7
Eclampsia	5	20.8	19	79.2
GDM	14	66.7	7	33.3

P Value <0.001, there was a statistically significant difference found between complication and outcome.

Figure 37:- Bar graph showing distribution of subjects according to complication and outcome (N=90).

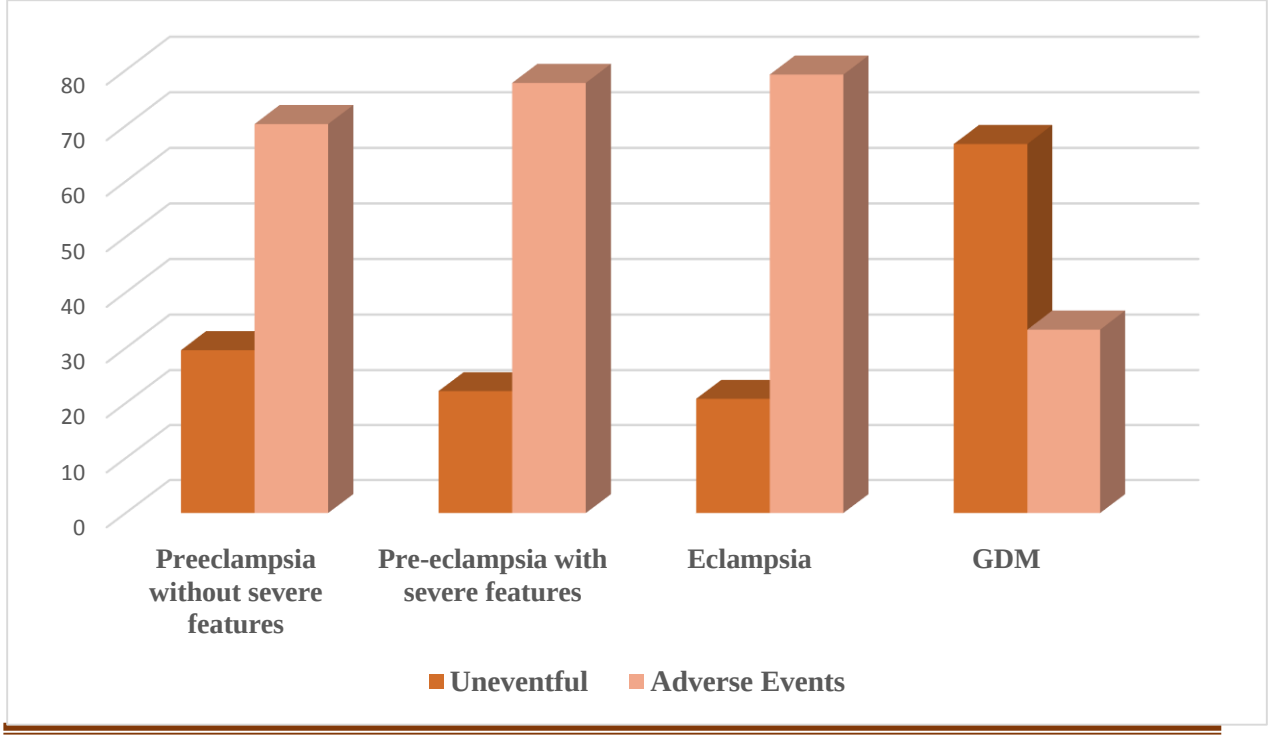


Table 22:- Distribution of subjects according to adverse outcomes (N=90).

	Frequency	Percentage
Low Apgar score	27	36.5
IUD	16	17.8
NICU admission	43	47.8
LBW	32	43.2

Among the study population (N=90), NICU (43/47.8 %) admission was the most common adverse outcome, followed by low birth weight (LBW) (32/43.2 %), low Apgar score (27/36.5 %) and intrauterine fetal demise (IUD) (16/17.8 %).

Table 23: - Distribution of subjects according to Feto-placental insufficiency and outcome (N=90).

	Uneventful	Adverse event
	N	N
Absent	23	10
Present	8	49

P Value <0.001, there was a statistically significant difference found between Feto-placental insufficiency and outcome.

Figure 38:- Bar graph showing distribution of subjects according to Feto-placental insufficiency and outcome (N=90).

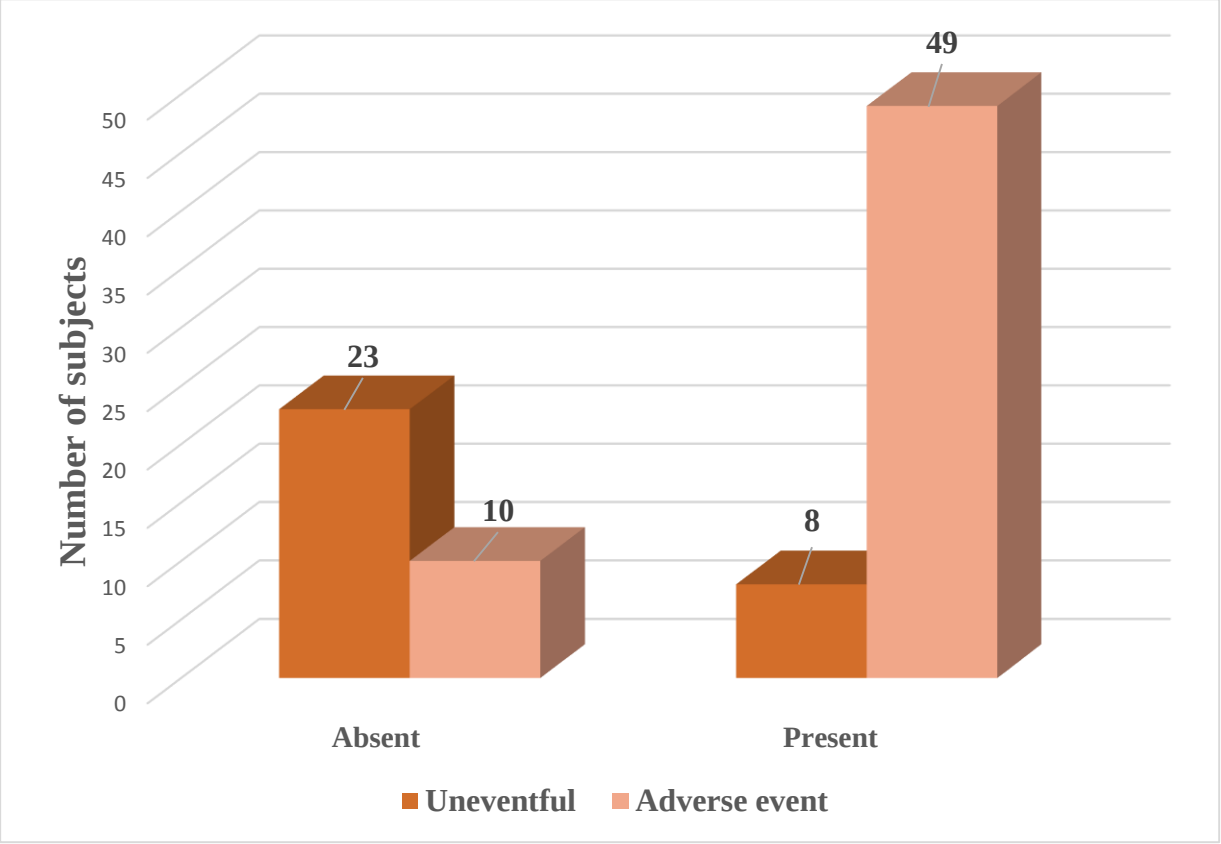


Table 24: - Distribution of subjects according to Utero-placental insufficiency and outcome (N=90).

	Uneventful	**Adverse event**
	N	N
Absent	15	13
Present	16	46

P Value 0.016, there was a statistically significant difference found between Utero-placental insufficiency and outcome.

Figure 39: - Bar graph showing distribution of subjects according to Utero-placental insufficiency and outcome (N=90).

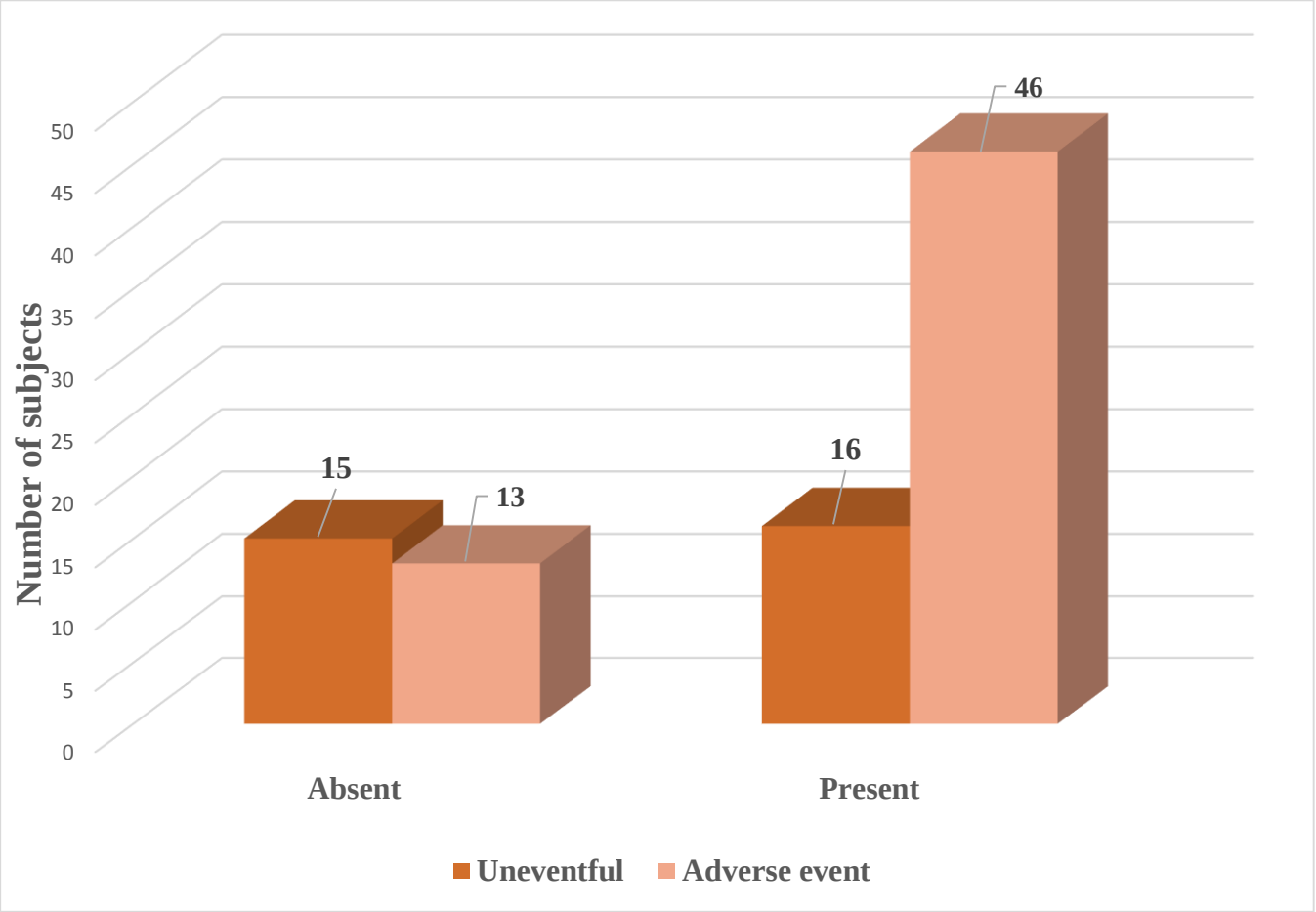


Table 25: - Sensitivity, Specificity, NPV, PPV and Accuracy for MCA PI, MCA RI, UA PI UA RI, UTA PI, UTA RI and CP Ratio in predicting Adverse fetal Outcome

Statistic	Sensitivity	Specificity	PPV	NPV	Diagnostic Accuracy
MCA PI	66.10%	90.32%	92.86%	58.33%	74.44%
MCA RI	40.68%	87.10%	85.71%	43.55%	56.67%
UA PI	83.05%	77.42%	87.50%	70.59%	81.11%
UA RI	59.32%	74.19%	81.40%	48.94%	64.44%
UTA PI	62.71%	67.74%	78.72%	48.84%	64.44%
UTA RI	55.93%	61.29%	73.33%	42.22%	57.78%
CP Ratio	79.66%	83.87%	90.38%	68.42%	81.11%

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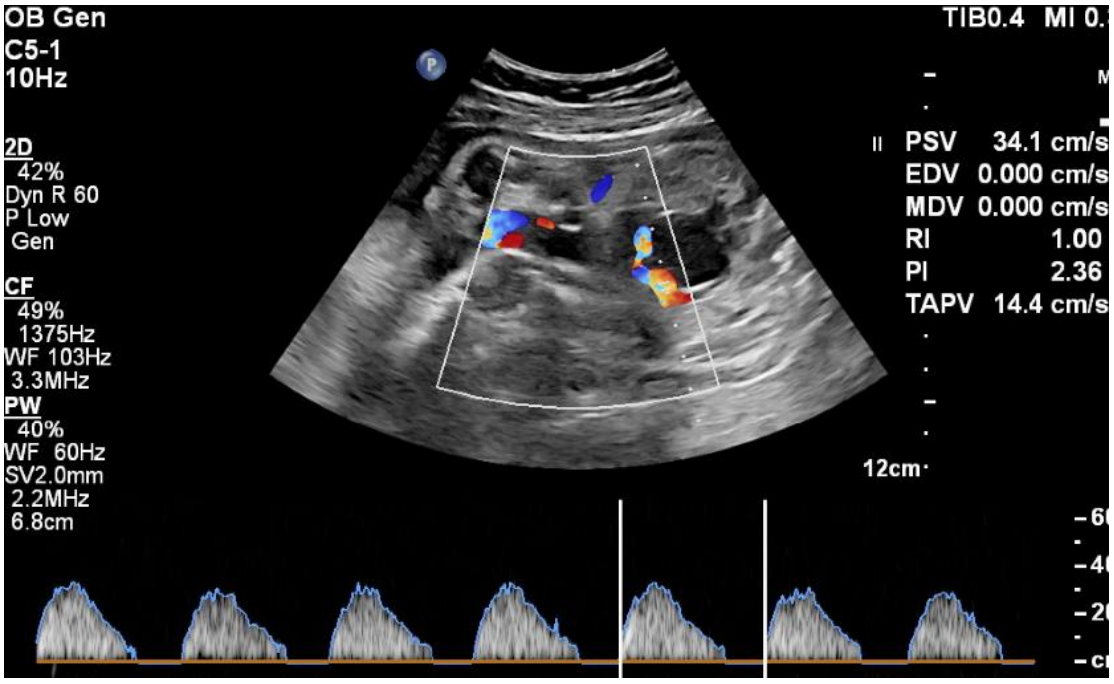

Figure 40: Spectral Doppler of umbilical artery showing absent end diastolic flow (AEDF) with raised UA PI in 25 years old women with high risk pregnancy having pre-eclampsia with severe features.

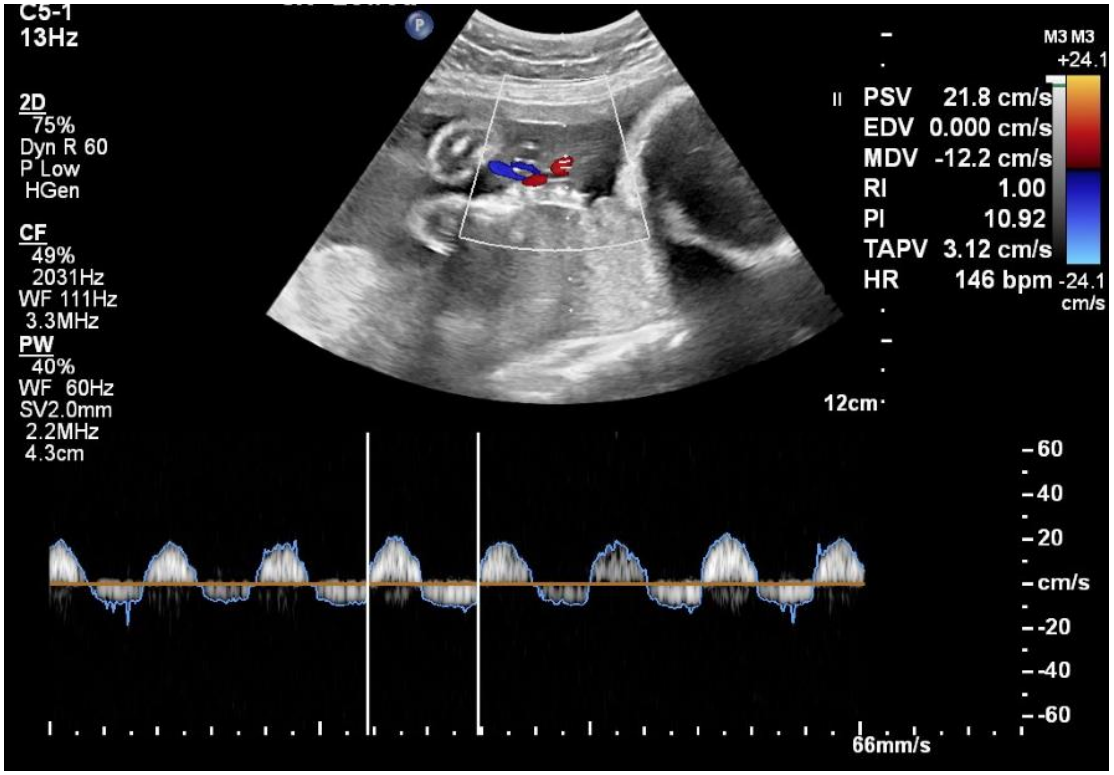

Figure 41: Spectral Doppler of umbilical artery showing reversal of end diastolic flow (REDF) with raised UA PI in 22 years old women having pre-eclampsia without sever features.

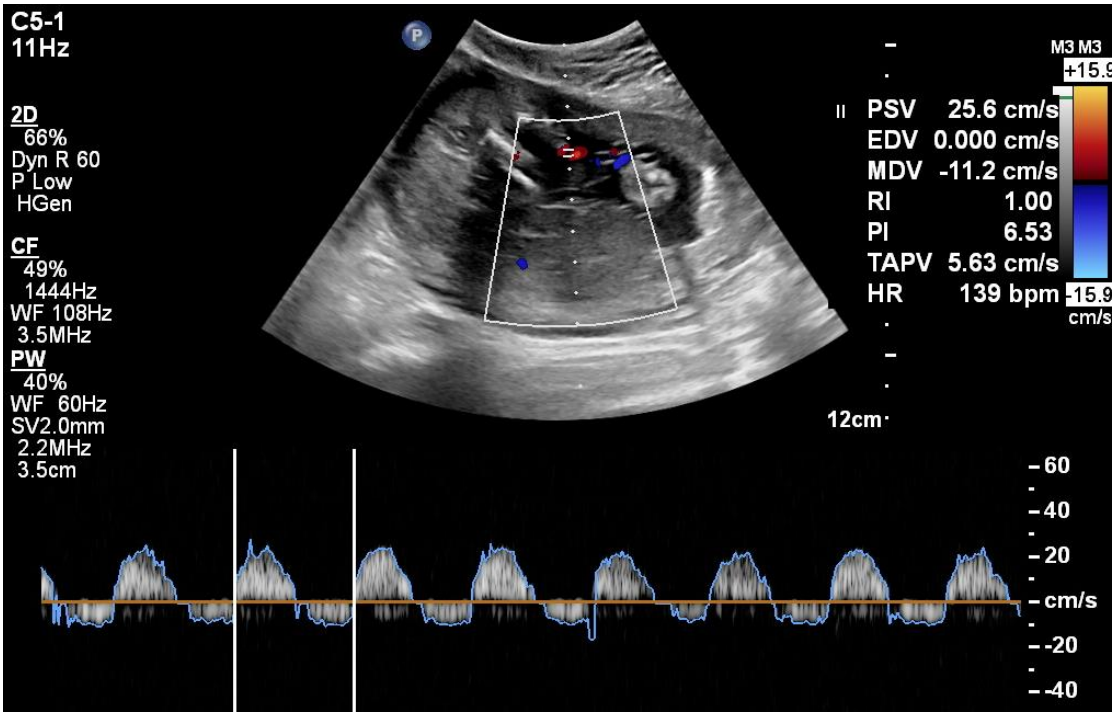

Figure 42: Spectral Doppler of umbilical artery showing reversal of end diastolic flow (REDF) with raised UA PI in 22 years old women with eclampsia.

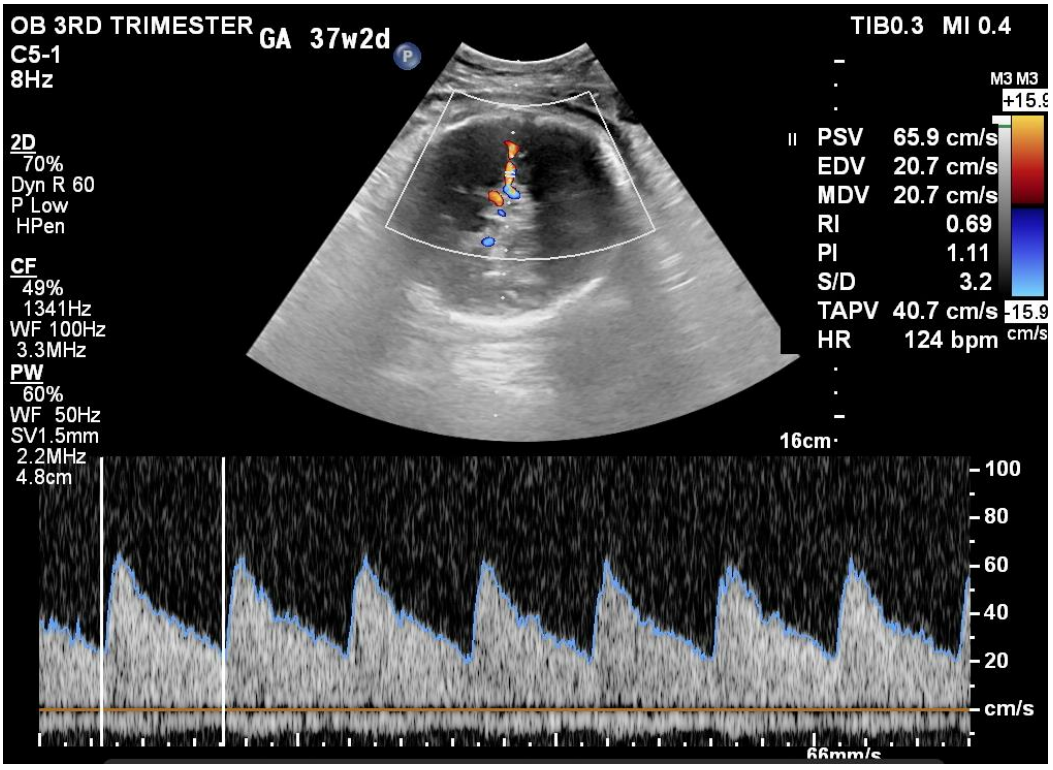

Figure 43: Spectral Doppler of middle cerebral artery showing raised diastolic flow in 30 years old women with pre-eclampsia with severe features.

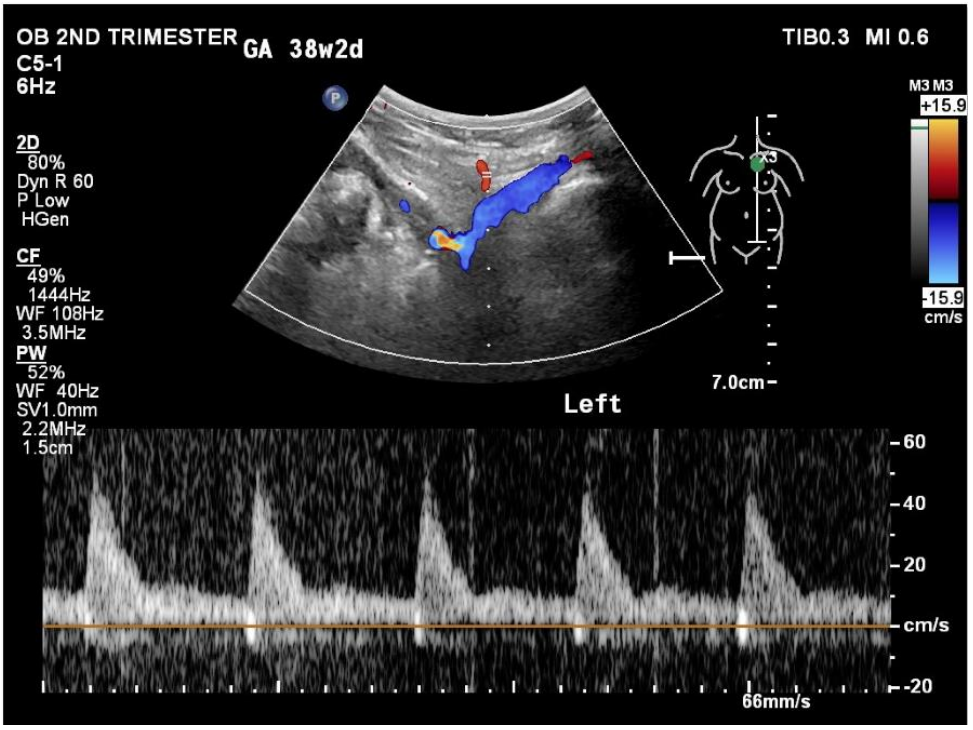

Figure 44: Spectral Doppler study of left uterine artery showing early diastolic notching in a patient with eclampsia

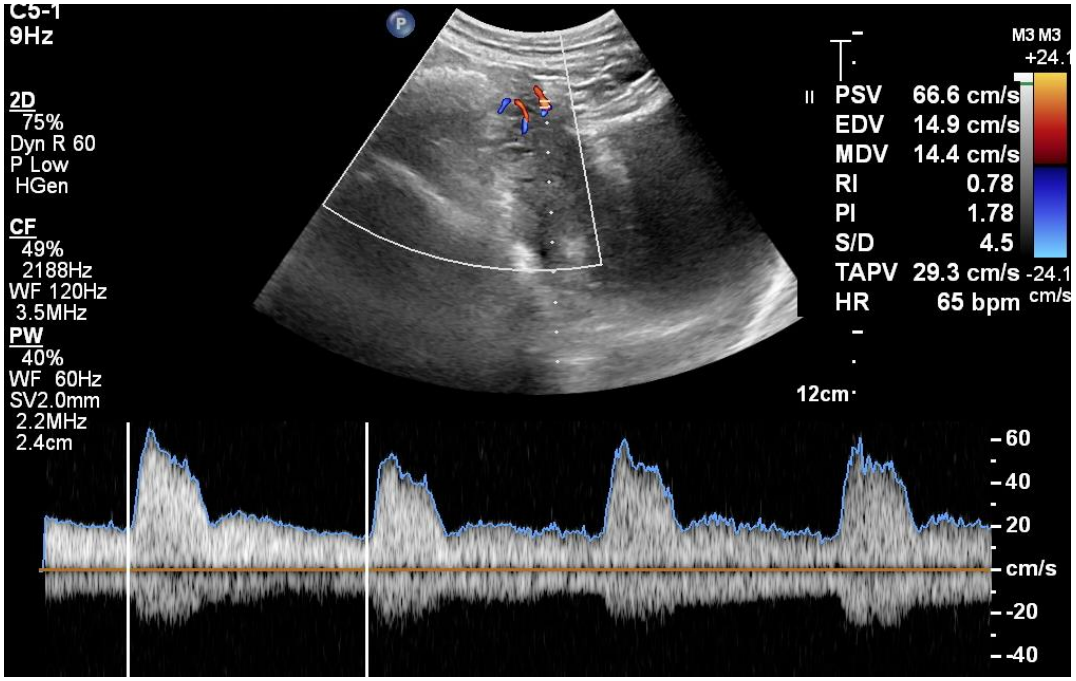

Figure 45: Spectral Doppler of uterine artery in 26 years old women with pre-eclampsia without severe features showing early diastolic notching with raised uterine artery pulsatility index (UTA PI).

DISCUSSION

DISCUSSION:

Studies using perinatal Doppler velocimetry are able to identify fetuses who are at risk of having adverse outcome, allowing for prompt intervention. It is challenging to analyse each parameter independently with different research because definition of adverse fetal outcomes is not fixed. Since various clinical factors and the Doppler results were taken into consideration for patient management in current study, it is possible that fetal outcome will differ from that of other studies. We studied role of color doppler & USG in high-risk pregnancies. Doppler finding's predictive value in a various high-risk pregnancy was studied and its significance in management of fetal outcome was established.

In current study, out of 90 (100%) patients, most of patients were in age group 21-25 years (39/43.3%). Similar finding was found in a study by Kavitha et al., where most of patients were in age group 26-30 years (n=80) followed by 20-25 years (n=60).[85] In current study, mean age was 25.4 years, ranged between 18 to 37 years in study population. Age ranged from 19 to 35 years & mean was calculated as 26.73 years in study of Gaikwad et al which is similar to our study. [87]

According to complications, out of 90 (100%) majority of the patients had PE without severe features (27/30%), followed by eclampsia (24/26.7%), GDM (21/23.3%), PE with severe features (18/20%). Statistically significant difference found between complication & outcome with p value of <0.001. This is similar to study of Aparna et al. about 90% of patients at risk had preeclampsia, and 6% had gestational diabetes.[88] In our study, out of the 90 patients 54.4 % were multigravidas and rest were primigravida's.

In our study, most of the patients (55/61.1%) were in >36 weeks of gestation period, followed by 25 (27.8%) of the patients were in 32-36 weeks and 10 (11.1%) were in 28-32 weeks. Aparna et al had the patients with gestational age of 28 to 37 weeks.[88]

Among the study population, 47 (52.2 %) participants had normal amniotic fluid volume, 37 (41.1 %) participants had oligohydramnios and 6 (6.7 %) participants had polyhydramnios. Oligohydramnios was seen in 20% of the patients in Kavitha et al.[85]

Among the total study population, growth lag was present in 43 (47.8 %) participants. According to fetal head circumference and abdominal circumference (HA/AC) ratio, out of 90 (100%) of the patients, 71 (78.9%) of the them were found to be in normal range and about 19 (21,2%) of them found to have increased HC/AC ratio which indicated asymmetrical IUGR in fetuses.

Out of 90 (100 %) patients, 47 (52.2 %) participants had normal amniotic fluid volume, 37 (41.1 %) participants had oligohydramnios and 6 (6.7 %) participants had polyhydramnios. Out of 90 (100%) patients, most of them (62/53.3%) had normal MCA-RI and (41/46.7%) normal MCA-PI. 28 (31.1%) of the patients had abnormal MCA-RI and 42 (46.7%) had abnormal MCA-PI.

In our study, out of 90 (100%) patients, most of them (47/53.2%) had normal UA-RI whereas most of the patients – 56 (62.2%) of the patients had abnormal UA-PI. Around 43% cases had abnormal UA, in all these cases early intervention was carried out and fetal survivability was 65% as observed by Kavitha et al.[85]

In the present study among the study population, 40 (44.4 %) subjects had normal umbilical artery flow, 17 (18.9 %) subjects had reduced flow in umbilical artery, 15 (16.7 %) subjects had absent umbilical artery flow and 18 (20 %) subjects had reversal of flow in umbilical artery. Kumbar et al evaluated role of Doppler parameters in IUGR babies and found that 50% perinatal deaths were seen in cases showing absent diastolic flow and 100% in cases having reverse end diastolic flow.[90] In Gaikwad et al, 50% mortality in cases of REDF and 33.33% mortality was seen in cases with AEDF.[87]

In our study, out of 90 (100%) patients, most of them (45/50.0%) had normal UTA-RI whereas most of the patients – 47 (52.2%) of the patients had abnormal UTA-PI. Among the study population, 38 (42.2 %) subjects had normal uterine artery flow and 52 (57.9 %) subjects had early diastolic notching in uterine arteries. In Kavitha et al.[85] IUD was seen in 67% fetuses with absent or reversal of diastolic flow. Ochi et al, stated that "elevated PI and the presence of diastolic notch in the uterine artery flow velocity are indicators of increased uterine arterial resistance and impaired uterine circulation".[91]

In the present study, out of 90 (100%) patients, 57 (63.3%) were found to have feto-placental insufficiency and 62/68.9% were found to have utero-placental insufficiency. P Value is <0.001, which showed statistically significant difference between Fetoplacental insufficiency and outcome. P Value 0.016, there was a statistically significant difference found between Utero-placental insufficiency and outcome.

Majority of patients out of 90(100%), about 59 (65.6%) had LSCS mode of delivery and remaining (31/34.4%) of the patients had normal vaginal delivery (NVD). This is in accordance with the study of Aparna et al, more than 90% of the women delivered by caesarean section.[88]

Out of 90 (100%) patients, about 59 (65.6%) had adverse event outcome and 31 (34.4%) had uneventful outcome. Which is similar to the study of Kavitha et al. out of these 15, 14 cases had adverse perinatal outcome.[85] Gaikwad et al. found that adverse perinatal outcome in 63 (50.4%) patients.[87]

Out of 59 (65.6%) adverse event outcome patients, most of the patients (20/74.1%) had Preeclampsia without severe features followed by eclampsia (19/79.25%), preeclampsia with severe features (13/72.2%) and GDM (7/33/3%). Statistically significant difference was found between complication and outcome with p-value of <0.001.

Among 90 (100%) of the patients, majority of the babies (43/47.8%) had NICU admission, about 32 (43.2%) babies had low birth weight (LBW) and 27 (36.5%) had low APGAR score. In Gaikwad study out of total 125 babies, 80.8% babies had LBW (< 2.5 kg). NICU admission was in 88.8% of babies for various reasons like LBW, asphyxia, prematurity etc.

In the present study the Sensitivity, Specificity, NPV, PPV and diagnostic accuracy for MCA RI in predicting adverse outcome was 40.68%, 87.10 %, 43.55, 85.71% and 56.67%. Gaikwad et al also studied the fetal middle cerebral artery by Doppler studies, MCA RI was found to be abnormal in 25.6% patients respectively. It was discovered that MCA RI had the highest specificity of 100% for predicting unfavorable outcomes. Additionally, MCA RI was found to have the PPV of 100% for predicting unfavorable outcomes.[87]

Our study found that the Sensitivity, Specificity, NPV, PPV and Accuracy for UA RI in predicting adverse outcome was 59.32%, 74.19%, 48.94%, 81.40% and 64.44%. In Gaikwad et al. study, the UA RI had a higher specificity and PPV of 91.94% and 81.48% in predicting adverse perinatal outcome. In Gaikwad et al, correlation between UA RI and pregnancy outcome was found statistically significant ($p<0.05$).[87] Another study conducted by Lakshmi et al. showed that the UA RI had sensitivity of 84.9%, specificity of 72.3%, PPV OF 77.5%, NPV OF 89 % and diagnostic accuracy of 79 %.[92] As described in table

Table 26: Comparison of UA RI Ratio in predicting adverse outcome

Study	Sensitivity	Specificity	PPV	NPV	Diagnostic accuracy
Lakshmi et al[92]	84.9%	72.3%	77.5%	89%	79%
Lakhkar et al[93]	44.4%	81.8%	80%	47.3%	-
Gaikwad et al[87]	34.92%	91.94%	81.48%	58.16%	63.20%
Present study	59.32%	74.19%	48.94%	81.40%	64.44%

The Sensitivity, Specificity, NPV, PPV and Accuracy for UTA RI in predicting adverse outcome was 55.93%, 61.29%, 73.33%, 42.22% and 57.78%. In Biswas S et al study, sensitivity of 30.30 %, specificity of 86.20%, PPV of 45.45%, NPV of 76.50 % and diagnostic accuracy of 70.8 % of UTA RI in predicting adverse fetal outcome at 31 to 36 weeks.

Our study showed that the Sensitivity, Specificity, NPV, PPV and Accuracy for MCA PI in predicting adverse outcome was 66.10%, 90.32%, 92.86%, 58.33% and 74.44%. Gaikwad et al[87] revealed MCA PI had highest specificity of 98.39% in predicting adverse perinatal outcome. Correlation of MCA PI and pregnancy outcome was statistically significant ($p<0.05$).

The PPV of MCA PI in present study is 92.31% in predicting unfavorable outcome that is compared with studies done is shown in Table below.

Table 27: MCA P.I. in predicting adverse outcomes

Study	Sensitivity	Specificity	PPV	NPV	Diagnostic accuracy
Yash et al[94]	76%	78%	-	-	52%
Lakshmi et al[92]	62,2%	78.7%	76.7%	64,9%	63%
Kumbar et al[90]	78.9%	68.4%	65.2%	76.4%	70%
Netam et al[95]	47.06%	81.81%	57.14%	75%	70%
Bano et al[96]	8.9%	100%	100%	-	-
Khanduri et al[97]	35.7%	92.6%	91.8%	38.2%	-
Fong et al[98]	72.4%	58.1%	37.7%	85.7%	-
Gaikwad et al[87]	19.5%	98.39%	92.31%	54.31%	58.40%
Present study	66.10%	90.32%	58.33%	92.86%	74.44%

We had the Sensitivity, Specificity, NPV, PPV and Accuracy for UA PI in predicting adverse outcome was 83.05%, 77.42%, 87.50%, 70.59% and 81.11%. Which is similar to the study of Gaikwad, UA RI and PI had high specificity of 91.94% and 93.55% respectively in identifying unfavorable perinatal outcome.[87] The sensitivity and specificity are comparable with studies of Yoon et al, Lakshmi et al and Khanduri et al.[99,92,97] (Table).

Table 28: UA PI in predicting adverse outcomes in different studies

Study	Sensitivity	Specificity	PPV	NPV	Diagnostic accuracy
Lakshmi et al[92]	86.7%	63%	73%	85.71%	76%
Lakhkar et al[93]	50%	59%	66.6%	41%	-
Kumbar et al[90]	89%	85.7%	85%	90%	87.5%
Yoon et al[99]	89%	86%	86%	89%	-
Bano et al[96]	46.7%	93%	87%	63%	70%
Khanduri et al[97]	73.8%	75.9%	87.7%	55.4%	75%
Gaikwad et al[87]	38.10%	75.9%	85.71%	59.79%	65.60%
Present et al	83.05%	77.42%	81.11%	70.59%	81.11%

The Sensitivity, Specificity, NPV, PPV and Accuracy for UTA PI in predicting adverse outcome was 62.71%, 67.74%, 78.72%, 48.84% and 64.44%. In Biswas S et al study, sensitivity (32.2 %), specificity (84.8%), PPV (38.4%), NPV (77.6 %) and diagnostic accuracy (70 %) of UTA PI in predicting adverse fetal outcome at 31 to 36 weeks.

In present study out of 90 (100%) patients, majority of patients (52/57.8%) had reversed fetal cerebroplacental (CP) ratio. The sensitivity of 79.66%, Specificity of 83.87%, NPV of 68.42%, PPV of 90.38% & accuracy of 81.11% for CP Ratio in predicting adverse outcome was observed in present study. In a similar study by Lakshmi et al it was found that CP ratio has 90 % sensitivity and 94 % PPV.[92] CP ratio has highest specificity and PPV of 90.32% and

83.33% respectively in identifying adverse perinatal outcome as seen in Gaikwad et al. study.[87]

In 2010, Bansal et al. did a study on role of pan vessel Doppler studies in high-risk pregnancies. Their conclusion was similar to our study, women with aberrant Doppler indices had higher rates of LSCS (78%), low Apgar scores, LBW & NICU admissions (36%).[100]

In 2010, Urmila and Beena did a study on triple vessel wave pattern in high-risk & normal pregnancies. They came to same conclusion as ours; in study group there was an increased incidence of LSCS and NICU admissions as compared to control group.[101]

In a 2016 study on colour Doppler ultrasonography in high-risk pregnancies, Amin et al. found that perinatal mortality and morbidity were 41.3% and 23.9% respectively, among 46 pregnancies with abnormal Doppler waveforms, in comparison with patients having normal Doppler waveforms, had 3.7% (mortality) and 11.1% (morbidity).[102]

In a study conducted by Kirkinen et al., it was discovered that Doppler investigations had advanced significantly and were now acknowledged as a crucial examination to forecast patient heart failure in hypoxic foetuses. In 83 low-risk and 84 high-risk pregnancies, pulsed Doppler method was utilized to record the blood flow velocity waveforms from fetal intracranial arteries. In the typical cases, the waveform's RI decreased as the pregnancy progressed, and these arteries continuous forward flow. A low RI had a 57% sensitivity and 94% specificity for predicting the birth of an infant that was small-for-dates and/or the subsequent development of a cardiotocographic abnormality.[103]

CONCLUSION

CONCLUSION:

This prospective study was carried out to enhance the fetal outcome by accurately assessing well-being of fetus in all high-risk pregnancies, and to make this method a part of protocol for assessing well-being of fetus in these patients. Total of 90 pregnant ladies were assessed.

- In current study, out of 90 (100%) subjects, majority of cases were in age group, 21-25 years (39/43.3%).
- The patients were distributed based on the parity. Of 90 (100%) patients most of them were multipara (49/54.4%) and rest 41 (45.6%) were primipara.
- According to complications, out of 90 (100%) majority of the patients had preeclampsia without severe features (27/30%), followed by eclampsia (24/26.7%), gestational diabetes mellitus (21/23.3%), preeclampsia with severe features (18/20%).
- In our study, most of the patients (55/61.1%) were in >36 weeks of gestation period, followed by 25 (27.8%) of the patients were in 32-36 weeks and 10 (11.1%) were in 28-32 weeks.
- Based on amniotic fluid index, 37 (41.1%) patients had oligohydramnios and about 6 (6.7%) had polyhydramnios.
- In our study, among 90 subjects, growth lag was present in 43 (47.8 %) participants.
- According to fetal head circumference and abdominal circumference (HA/AC) ratio, out of 90 (100%) patients, 71 (78.9%) of the them were found to be in normal range and about 19 (21.2%) of them found to have increased HC/AC ratio which indicated asymmetrical IUGR.
- Out of 90 (100%) patients, 28 (31.1%) of the patients had abnormal (< 5^{th} percentile) MCA-RI and 42 (46.7%) had abnormal (< 5^{th} percentile) MCA-PI.
- Out of 90 (100%) patients, most of the patients – 56 (62.2%) of the patients had abnormal UA-PI and 43 (47.8 %) had abnormal UA RI.

- In current study, out of 90 (100%) patients, umbilical artery flow (UA) was reduced in 17 (18.9%), absent in 15 (16.7 %) and reversed in 18 (20%) of the patients.
- Out of 90 (100%) patients most of the patients (52/57.8%) had reversal of cerebroplacental (CP) ratio.
- Out of 52 (57.8%) reversed fetal CP ratio patients, 18 (75%) of them had eclampsia, 13 (72.2%) of them had Pre-eclampsia with severe features and 6 (22.2%) had Preeclampsia without severe features.
- Out of 90 (100%) patients, about (45/50%) had abnormal (above > 95^{th} percentile) UTA RI and 47 (52.2%) had abnormal (above > 95^{th} percentile) UTA PI.
- Out of 90 (100%) patients, according to uterine artery flow (UTA flow), most of the patients (52/57%) had early diastolic notching reversed.
- Out of 90 (100%) patients, 57 (63.3%) found to have fetoplacental insufficiency and 62 (68.9%) found to have utero-placental insufficiency.
- Majority of the patients out of 90 (100%), about 59 (65.6%) underwent LSCS and remaining (31/34.4%) of patients had normal vaginal delivery (NVD).
- Out of 90 (100%) patients, about 59 (65.6%) had adverse event outcome and 31 (34.4%) had uneventful outcome. Out of the 59 (65.6 %), majority of adverse outcomes were seen in preeclampsia with severe features.
- Among the study population (N=90), NICU (43/47.8 %) admission was the most common adverse outcome, followed by low birth weight (LBW) (32/43.2 %), low Apgar score (27/36.5 %) and intrauterine fetal demise (IUD) (16/17.8 %).
- Sensitivity, Specificity, NPV, PPV and accuracy of MCA RI in predicting adverse outcome was 40.68%, 87.10 %, 85.71%, 43.55% and 56.67%. Whereas, the Sensitivity, Specificity, NPV, PPV and Accuracy for UA RI in predicting adverse outcome was 59.32%, 74.19%, 81.40%, 48.94% and 64.44%.

- The Sensitivity, Specificity, NPV, PPV and Accuracy for UTA RI in predicting adverse outcome was 55.93%, 61.29%, 73.33%, 42.22% and 57.78%.
- Sensitivity, Specificity, NPV, PPV and accuracy of MCA PI in predicting adverse outcome was 66.10%, 90.32%, 92.86%, 58.33% and 74.44%. Whereas the Sensitivity, Specificity, NPV, PPV and Accuracy for UA PI in predicting adverse outcome was 83.05%, 77.42%, 87.50%, 70.59% and 81.11%. The Sensitivity, Specificity, NPV, PPV and Accuracy for UTA PI in predicting adverse outcome was 62.71%, 67.74%, 78.72%, 48.84% and 64.44%.
- CP ratio has sensitivity of 79.66%, Specificity of 83.87%, NPV of 68.42%, PPV of 90.38% and Accuracy of 81.11% in predicting adverse outcome.

SUMMARY

SUMMARY:

Based on the study results it was concluded that: -

- Doppler analysis along with ultrasonography is a standard for evaluating fetus in high-risk pregnancies.
- CP Ratio & UA PI had better sensitivity & PPV in identifying adverse outcomes.
- Diagnostic accuracy of CP ratio was highest in predicting adverse outcomes.
- It was seen that MCA RI and PI together also had better highest specificity and PPV in identifying adverse outcomes.
- Thus, study's findings support use of colour Doppler imaging in high-risk pregnancies will help in early identification of adverse fetal outcomes and aids in early intervention.

LIMITATIONS AND RECOMMENDATIONS:

- This study is not widely accepted for screening low-risk pregnancies. However, this study accurately assesses fetal well-being in all high-risk pregnancies, in order to improve fetal outcome. Hence fetal Doppler can be incorporated as a protocol for assessment of fetal well-being in all high-risk pregnancy.

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ANNEXURE

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

ANNEXURE I - PATIENT PROFORMA

STUDY TITLE: ROLE OF ULTRASONOGRAPHY AND COLOR DOPPLER IN ASSESSMENT OF HIGH-RISK PREGNANCIES AND ITS ACCURACY IN PREDICTING FETAL OUTCOME

DEMOGRAPHIC DETAILS

1. Name:
2. Age:
3. UHID No / IP No:

OBSTETRIC HISTORY

1. Obstetric score:
2. LMP:
3. EDD:
4. Presenting complaints:

5. Previous obstetric history:

6. Clinical examination:

ULTRASONOGRAPHIC FINDINGS

1. FHR
2. BPD
3. HC
4. AC
5. FL
6. EFW
7. Placenta
8. Liquor
9. AUA

DOPPLER FINDINGS:

1. Uterine artery
2. Umbilical artery
3. Middle cerebral artery

FETAL OUTCOME:

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

INFORMED CONSENT FORM

PG guide's name: Dr. RAJESWARI

PG co-guide's name: Dr. S. R. SHEELA.

Principal investigator: Dr. R. MAHIMA KALE

NAME OF THE SUBJECT:

AGE :

GENDER :

a. I have been informed in my own language that this study involves MRI as part of procedure. I have been explained thoroughly and understand the procedure.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).
e. I confirm that Dr. RAJESWARI / Dr. Dr. R. MAHIMA KALE. (guide /name of PG guide) has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that I may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Participant's signature/thumb impression
Signature of the witness: Date:

1)

2)
I have explained to_____(subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Chief Researcher/Guide signature Date:

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

PATIENT INFORMATION SHEET

PRINCIPAL INVESTIGATOR: Dr. R Mahima Kale /Dr. Rajeswari /Dr. S. R. Sheela

This is to inform you that,

I, Dr. R Mahima Kale, post-graduate student in Department of Radiodiagnosis at Sri Devaraj Urs Medical College. I will be conducting a study titled "Role of ultrasonography and color doppler in assessment of high-risk pregnancies and its accuracy in predicting fetal outcome." for my dissertation under the guidance of Dr. Rajeswari, Associate Professor, Department of Radiodiagnosis and under the co-guidance of Dr. S. R. Sheela, Professor, Department of Obstetrics and Gynaecology. In this study, we will assess the role of ultrasonography color Doppler in various high-risk pregnancies and its role in predicting the fetal outcome.

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.

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. R Mahima Kale or any other member of the above research team for any doubt or clarification you have.

Name and Signature of the Principal Investigator

Date

MASTER CHART

MASTER CHART

SL.NO	UHID NO	AGE	OBS SCORE	COMPLICATION	GA BY LMP	FHR	AUA	BPD	HC	AC	FL	PLACENTA	EFW	AFI	LIQUOR	LAG	HC/AC Ratio	MCA RI	MCA PI	MCA PI	UA RI	UA RI	UA PI	UA PI
1	891166	22	Primi	Eclampsia	39 weeks	133	35 weeks 3 days	8.7	31.6	30.1	6.9	Left lateral, grade III maturity	2.6 ± 370 g	4 to 5	Oligohydramnios	4 weeks	Normal	0.8	Normal	1.7	Normal	0.6	Normal	1	Normal
2	890269	27	Multipara	Pre-eclampsia with severe features	30 weeks 1 day	135	26 weeks 1 day	6.5	25	20	4.5	Posterior, grade II maturity	900 g ± 124 g	5 to 6	Oligohydramnios	4 weeks	Increased	0.8	Normal	1.5	Abnormal	1	Abnormal	10.9	Raised
3	896783	22	Multipara	Preeclampsia without severe features	38 weeks 2 days	151	35 weeks	8.3	30.9	26.2	6.8	Fundal, grade II maturity	2.3 ± 291 g	3 to 4	Oligohydramnios	3 weeks	Increased	0.7	Normal	1.3	Abnormal	0.6	Normal	1.4	Raised
4	893893	24	Multipara	Pre-eclampsia with severe features	35 weeks 3 days	144	29 weeks 6 days	7.7	28.3	23.6	5.5	Anterior, grade II maturity	1.1 ± 250 g	4	Oligohydramnios	6 weeks	Increased	0.6	Abnormal	0.9	Abnormal	1	Abnormal	4.8	Raised
5	895935	25	Multipara	Eclampsia	34 weeks 6 days	144	32 weeks 1 day	7.9	30.2	25.4	6.5	Fundal left lateral, grade II maturity	1.7 ± 295 g	5 TO 6	Oligohydramnios	Nil	Normal	0.8	Normal	2.3	Normal	0.8	Abnormal	1.6	Raised
6	891976	25	Multipara	Eclampsia	34 weeks 5 days	126	33 weeks 2 days	8.5	30.7	27.5	6.4	Left lateral, grade II maturity	1.8 ± 277 g	9 to 10	Adequate	Nil	Normal	0.7	Normal	1.5	Abnormal	1	Abnormal	2.3	Raised
7	889637	18	Primi	GDM	37 weeks 3 days	140	37 weeks 1 day	9.2	30.9	32.6	7.2	Fundal posterior, grade III maturity	3 ± 447 g	19 to 20	Polyhydramnios	Nil	Normal	0.7	Normal	1.3	Normal	0.6	Normal	1	Normal
8	891701	26	Primi	Preeclampsia without severe features	38 weeks 3 days	151	36 weeks 3 days	8.7	32.2	32.1	7.3	Anterior, grade II maturity	2.9 ± 350 g	10 to 11	Adequate	Nil	Normal	0.6	Abnormal	1.4	Normal	0.5	Normal	0.6	Normal
9	885771	37	Multipara	GDM	33 weeks 3 days	157	35 weeks 5 days	9.1	32.7	31	6.4	Fundal posterior, grade III maturity	2.5 ± 328 G	17 TO 18	Polyhydramnios	Nil	Normal	1	Normal	2.5	Normal	0.6	Normal	0.9	Normal
10	903192	20	Primi	Preeclampsia without severe features	30 weeks	155	24 weeks 1 day	5.9	22	18.6	4.4	Posterior, grade I maturity	1.0 ± 150 g	11 to 12	Adequate	6 weeks	Increased	0.6	Abnormal	1.3	Abnormal	0.8	Abnormal	1.6	Raised
11	907456	30	Multipara	Preeclampsia without severe features	37 weeks 6 days	156	33 weeks 6 days	8.4	30.6	28.6	6.6	Anterior, grade I maturity	2.0 ± 302 g	3 to 4	Oligohydramnios	4 weeks	Normal	0.6	Abnormal	1.1	Abnormal	0.9	Abnormal	1.8	Raised
12	907459	24	multipara	Eclampsia	36 weeks 5 days	140	33 weeks 2 days	8.4	30.5	27.9	6.3	Fundal posterior, grade II maturity	2.0 ± 300 g	5 to 6	Oligohydramnios	3 weeks	Normal	0.7	Normal	1.7	Normal	0.6	Normal	1.9	Raised
13	908798	23	Primi	Eclampsia	32 weeks 4 days	121	30 weeks 3 days	7.7	28	24.3	5.9	Anterior, grade II maturity	1.4 ± 210 g	14 to 15	Adequate	Nil	Normal	0.6	Abnormal	1.5	Abnormal	0.8	Abnormal	1.7	Raised
14	910181	22	Multipara	Pre-eclampsia with severe features	34 weeks 3 days	138	32 weeks	8.4	30.1	26.2	5.6	Right lateral, grade II maturity	1.8 ± 273g	9 to 10	Adequate	Nil	Normal	0.7	Normal	1.3	Abnormal	0.7	Normal	2.7	Raised
15	919907	25	Primi	Preeclampsia without severe features	35 weeks 3 days	134	31 weeks 5 days	7.8	28.8	27.6	6	Fundal posterior, grade II maturity	1.9 ± 264 g	9 to 10	Adequate	3 weeks	Normal	0.7	Normal	1.3	Abnormal	0.7	Normal	1.6	Raised
16	902708	32	Multipara	Pre-eclampsia with sever features	33 weeks 5 days	160	31 weeks 5 days	7.8	28.5	28.2	6	Anterior right lateral, grade II maturity	1.9 ± 270 g	8 TO 10	Adequate	Nil	Normal	0.7	Normal	1.7	Abnormal	0.7	Normal	1.2	Normal
17	898113	20	Primi	Preeclampsia without severe features	38 weeks 6 days	147	33 weeks 6 days	8.4	30.6	28.6	6.6	Anterior, grade III maturity	2 ± 300 g	5 to 6	Oligohydramnios	5 weeks	Normal	0.8	Normal	1.7	Normal	0.6	Normal	2	Raised
18	917553	25	Multipara	Eclampsia	38 weeks 3 days	150	34 weeks 3 days	8.7	32.3	27	6.7	Fundal grade III maturity	2.1 ± 383 g	4 to 5	Oligohydramnios	4 weeks	Increased	0.7	Normal	1.5	Normal	0.5	Normal	1.8	Raised
19	908393	21	Primi	GDM	39 weeks 5 days	143	39 WEEKS 2 DAYS	9.9	35.9	39.4	7.6	Posterior, grade III maturity	3.7 ± 450 g	15 to 16	Adequate	Nil	Normal	0.7	Normal	1.3	Normal	0.5	Normal	1	Normal
20	923556	36	Multipara	Preeclampsia without severe features	35 weeks 6 days	133	32 weeks 5 days	7.7	28.7	29.1	6.6	Posterior left lateral, grade II maturity	2 ± 350 g	5 to 6	Oligohydramnios	3 weeks	Normal	0.6	Normal	1.1	Abnormal	1	Abnormal	2.7	Raised
21	925046	21	Primi	Pre-eclampsia with severe features	33 WEEKS 1 DAY	150	29 WEEKS 2 DAYS	7.7	28.9	23.2	5.7	Anterior, grade III maturity	1.1 ± 195 g	5	Oligohydramnios	3 weeks	Increased	0.7	Normal	1.6	Normal	1	Abnormal	3.8	Raised
22	928928	20	Multipara	Preeclampsia without severe features	35 WEEKS 2 DAYS	138	31 WEEKS 3 DAYS	7.3	28.3	27.7	6.4	Posterior, grade III maturity	1.8 ± 319 g	13 to 14	Adequate	4 weeks	Normal	0.8	Normal	1.7	Normal	0.7	Normal	2	Raised
23	930657	28	Primi	GDM	41 WEEKS 1 DAY	152	40 WEEKS 2 DAYS	8.9	32.1	32.2	7.8	Posterior, grade III maturity	3.1 ± 464g	14 to 15	Adequate	Nil	Normal	0.7	Normal	1.2	Normal	0.5	Normal	1	Normal
24	932536	20	Primi	Pre-eclampsia with severe features	38 WEEKS 4 DAYS	138	34 WEEKS 2 DAYS	8.3	28.7	26.8	6.3	Posterior, grade III maturity	2.3 ± 340 g	3 to 4	Oligohydramnios	4 weeks	Normal	0.6	Abnormal	1.1	Abnormal	0.8	Abnormal	1.7	Raised
25	935180	25	Multipara	Eclampsia	38 WEEKS 3 DAYS	120	32 WEEKS 6 DAYS	7.8	29.1	27.5	6.3	Anterior, grade II maturity	1.8 ± 275 g	3 to 4	Oligohydramnios	5-6 weeks	Normal	0.7	Normal	1.3	Abnormal	0.8	Abnormal	1.7	Raised
26	925733	20	Primi	GDM	36 WEEKS 1 DAY	135	37 WEEKS 4 DAYS	9.1	32.7	33.9	7.3	Anterior, grade III maturity.	3.2 kg ± 475 g	13 to 14	Adequate	Nil	Normal	0.5	Normal	1.3	Normal	0.6	Normal	1	Normal
27	928992	24	Multipara	GDM	35 WEEKS 1 DAY	143	34 WEEKS 6 DAYS	8.4	30.4	31.6	6.8	Posterior, grade III maturity	2.6 ± 381g	11 to 12	Adequate	Nil	Normal	0.9	Normal	2.5	Normal	0.7	Normal	1.2	Normal
28	936450	27	Primi	Preeclampsia without severe features	38 weeks 6 days	161	34 WEEKS 4 DAYS	8.4	31.1	26.2	6.7	Fundal posterior, grade II maturity	2.3 ± 307g	3 to 4	Oligohydramnios	4-5 weeks	Increased	0.6	Abnormal	1	Abnormal	1	Abnormal	3.6	Raised
29	936987	30	Primi	GDM	41 WEEKS 1 DAY	152	40 WEEKS 2 DAYS	8.9	32.1	32.2	7.8	Posterior, grade III maturity	3.5 ± 464g	14 to 15	Adequate	nil	Normal	0.6	Normal	1.1	Normal	0.5	Normal	0.9	Normal
30	937813	29	Multipara	Eclampsia	31 weeks 5 days	146	27 WEEKS 1 DAY	7	26.2	22.9	4.7	Fundal posterior, grade II maturity	1.2 ± 150 g	5 TO 6	Oligohydramnios	4 weeks	Increased	0.7	Abnormal	1.7	Abnormal	1	Abnormal	3.2	Raised
31	938201	32	Multipara	Pre-eclampsia with severe features	32 WEEKS 4 DAYS	143	31 WEEKS 4 DAYS	7.9	28.9	26	6.1	Posterior, grade II maturity	1.6 ± 245g	5	Oligohydramnios	nil	Normal	0.6	Abnormal	1.2	Abnormal	1	Abnormal	2.5	Raised
32	939954	23	Multipara	Preeclampsia with severe features	37 WEEKS 4 DAYS	140	33 WEEKS 4 DAYS	8.1	30.2	29.8	6.5	Posterior, grade III maturity	2.0 ± 328g	4	Oligohydramnios	4 weeks	Normal	0.8	Normal	1.3	Abnormal	0.7	Normal	1.7	Raised
33	941367	32	Primi	Preeclampsia without severe features	38 WEEKS	134	36 WEEKS 1 DAY	8.8	31.7	29.1	7	Posterior, grade III maturity	2.6 ± 357 g	7	Adequate	Nil	Normal	0.6	Abnormal	1.9	Normal	0.8	Abnormal	1.5	Raised
34	56647	21	Multipara	Preeclampsia without severe features	37 WEEKS 3 DAYS	142	33 WEEKS 3 DAYS	8.2	30.7	28.8	6.2	Posterior, grade III maturity	2.0 ± 303 g	4	Oligohydramnios	4 weeks	Normal	0.8	Normal	1.2	Abnormal	0.6	Normal	1.4	Raised
35	61780	20	Primi	Pre-eclampsia without severe features	38 weeks 6 days	161	35 WEEKS 1 DAYS	8.4	31.1	26.2	6.7	Fundal posterior , grade II maturity	2.4 ± 307g	3 to 4	Oligohydramnios	3 weeks	Increased	0.7	Normal	1.3	Abnormal	1	Abnormal	1.7	Raised
36	62720	26	Primi	Preeclampsia without severe features	37 WEEKS 2 DAYS	140	35 weeks	8.9	32.5	28.8	7.3	Posterior, grade III maturity	2.5 ± 380 g	5	Oligohydramnios	nil	Normal	0.7	Normal	1.3	Abnormal	0.7	Normal	1.2	Normal
37	63297	27	Multipara	Preeclampsia without severe features	30 WEEKS 4 DAYS	148	26 WEEKS 3 DAYS	7.1	26.3	22.1	5.1	Anterior, grade –II maturity	1.0 kg ± 153 g	12 to 13	Adequate	4 weeks	Normal	0.5	Abnormal	1.3	Abnormal	0.9	Abnormal	1.7	Raised
38	64699	23	Primi	Pre-eclampsia with severe features	38 WEEKS 4 DAYS	138	36 WEEKS 1 DAY	8.8	31.7	29.1	7	Posterior, grade III maturity	2.8 ± 357 g	7	Adequate	Nil	Normal	0.8	Normal	1.9	Normal	0.8	Abnormal	1.5	Raised
39	64817	23	Multipara	Eclampsia	33 WEEKS 1 DAY	160	32 WEEKS 6 DAYS	8.6	30.7	29	6.5	Anterior, grade II maturity	1.7 ± 210 g	8	Adequate	nil	Normal	0.6	Abnormal	1.3	Abnormal	1	Abnormal	3.2	Raised
40	68557	29	Multipara	Pre-eclampsia with severe features	31 WEEKS	135	26 WEEKS 2 DAYS	6.2	24.6	20	5.1	Anterior, grade II maturity	880 g ± 110	6 to 7	Adequate	5 weeks	Increased	0.6	Abnormal	1.2	Abnormal	1	Abnormal	2.3	Raised
41	69626	26	Primi	Pre-eclampsia with severe features	38 weeks 6 days	161	33 WEEKS 4 DAYS	8.4	30.2	27.8	6.7	Fundal posterior, grade II maturity	2.2 ± 307g	3 to 4	Oligohydramnios	4-5 weeks	Normal	0.6	Abnormal	1	Abnormal	1	Abnormal	3.6	Raised
42	60048	30	Multipara	Pre-eclampsia with severe features	34 WEEKS 1 DAY	152	32 WEEKS 1 DAY	7.6	28.5	26.5	6	Anterior, grade II maturity.	2.0 kg ± 300 g	9 to 10	Adequate	Nil	Normal	0.5	Abnormal	1.3	Abnormal	1.1	Abnormal	2.9	Raised
43	41116	20	Primi	Pre-eclampsia with severe features	30 WEEKS	152	26 WEEKS 6 DAYS	6.6	25.2	21.2	4.8	Anterior , grade II maturity	1.6 kg ± 260 g	5	Oligohydramnios	3 weeks	Increased	0.8	Normal	0.8	Abnormal	0.7	Normal	1.5	Raised
44	50501	21	Multipara	Eclampsia	32 WEEKS	146	28 WEEKS 4 DAYS	7	27.2	24.9	4.7	Fundal posterior, grade II maturity	1.0 ± 150 g	5	Oligohydramnios	4 weeks	Normal	0.6	Abnormal	1.2	Abnormal	1	Abnormal	2.5	Raised
45	916423	28	Multipara	Eclampsia	37 WEEKS 5 DAYS	140	32 WEEKS 3 DAYS	7.9	29	28.6	6.3	Anterior, grade II maturity	1.9 kg ± 310 g	4 to 5	Oligohydramnios	4 weeks	Normal	0.5	Abnormal	0.8	Abnormal	1	Abnormal	2.8	Raised
46	936211	33	Multipara	GDM	37 WEEKS 1 DAY	167	37 WEEKS 4 DAYS	9.1	32.7	33.9	7.3	Anterior, grade III maturity.	3.4 kg ± 475 g	22 TO 23	Polyhydramnios	nil	Normal	0.7	Normal	1.4	Normal	0.6	Normal	1	Normal
47	946997	23	Primi	Eclampsia	30 WEEKS 4 DAYS	148	28 WEEKS 3 DAYS	7.1	26.3	22.1	5.1	Anterior, grade –II maturity	1.0 kg ± 153 g	12 to 13	Adequate	nil	Normal	0.7	Abnormal	1.3	Abnormal	0.9	Abnormal	1.7	Raised
48	40499	25	Multipara	Eclampsia	36 weeks 3 days	150	35 weeks 3 days	8.7	31.3	27.8	6.7	Fundal grade III maturity	2.6 ± 383 g	7 to 8	Adequate	Nil	Normal	0.7	Normal	1.5	Normal	0.5	Normal	1.8	Raised
49	951507	26	Primi	Preeclampsia without severe features	37 WEEKS 2 DAYS	140	35 weeks 5 days	8.9	32.5	28.8	7.3	Anterior, grade II maturity	2.5 ± 210 g	14 to 15	Adequate	Nil	Normal	0.8	Normal	1.8	Normal	0.6	Normal	0.8	Normal
50	37419	21	Multipara	Preeclampsia without severe features	37 WEEKS 2 DAYS	140	35 weeks	8.9	32.5	28.8	7.3	Posterior, grade III maturity	2.5 ± 380 g	10 TO 11	Adequate	nil	Normal	0.7	Normal	1.5	Normal	0.7	Abnormal	1.1	Normal
51	43138	28	Primi	Eclampsia	36 weeks 5 days	140	33 weeks 2 days	8.4	30.5	27.9	6.3	Fundal posterior, grade II maturity	2.0 ± 300 g	7 to 8	Adequate	3 weeks	Normal	0.7	Normal	1.7	Normal	0.6	Normal	1.9	Raised
52	43186	30	Primi	Eclampsia	33 WEEKS 1 DAY	146	32 WEEKS 4 DAYS	8.2	29.7	28.1	6.1	Posterior, grade –II maturity	2.0 kg ± 280 g	13 TO 14	Adequate	nil	Normal	0.8	Normal	1.8	Normal	0.6	Normal	1	Normal
53	44591	26	Multipara	Preeclampsia without severe features	38 WEEKS	139	34 WEEKS 1 DAY	8.2	30.1	28.9	6.2	Fundal posterior, grade III maturity	2.3 kg ± 330 g	7 to 8	Adequate	4 weeks	Normal	0.7	Normal	1.4	Abnormal	0.8	Abnormal	1.7	Raised
54	55650	26	Primi	GDM	37 WEEKS 5 DAYS	160	38 WEEKS	7.8	28.5	28.2	6	Anterior right lateral, grade II maturity	3.4 KG ± 500 G	16 - 17	Adequate	nil	Normal	0.7	Normal	1.4	Normal	0.58	Normal	0.9	Normal
55	57934	28	Multipara	GDM	36 weeks 5 days	140	37 WEEKS 4 DAYS	9.1	32.7	33.9	7.3	Anterior, grade III maturity.	3.2 kg ± 475 g	13 to 14	Adequate	Nil	Normal	0.7	Normal	1.3	Normal	0.6	Normal	1.1	Normal
56	58606	25	Primi	Preeclampsia without severe features	30 weeks	155	25 weeks 1 day	5.9	22	18.6	4.4	Posterior, grade I maturity	980 ± 110 g	5 to 6	Oligohydramnios	5 weeks	Increased	0.6	Abnormal	1.8	Normal	1	Abnormal	3.2	Raised
57	59247	20	Primi	GDM	40 WEEKS 2 DAYS	149	39 WEEKS 2 DAYS	9.9	35.9	39.4	7.6	Posterior, grade III maturity	3.7 ± 450 g	10to 11	Adequate	nil	Normal	0.8	Normal	1.8	Normal	0.6	Normal	0.8	Normal
58	58650	29	Multipara	GDM	37 WEEKS 1 DAY	167	37 WEEKS 4 DAYS	9.1	32.7	33.9	7.3	Anterior, grade III maturity.	3.2 kg ± 475 g	22 TO 23	Polyhydramnios	nil	Normal	0.7	Normal	1.4	Normal	0.5	Normal	0.8	Normal
59	59816	34	Multipara	Pre-eclampsia with severe features	37 weeks 3 days	140	37 weeks 1 day	9.2	30.9	32.6	7.2	Fundal posterior, grade III maturity	3 ± 447 g	8 to 9	Adequate	nil	Normal	0.7	Normal	1.4	Normal	0.6	Normal	1	Normal
60	62663	23	Multipara	GDM	38 WEEKS	138	38 WEEKS 3 DAYS	8.9	32.1	32.2	7.8	Posterior, grade III maturity	3.1 ± 464g	14 to 15	Adequate	nil	Normal	0.6	Normal	1.1	Normal	0.6	Normal	0.9	Normal
61	72247	22	Primi	Pre-eclampsia with severe features	38 weeks 6 days	161	34 WEEKS 4 DAYS	8.4	31.1	26.2	6.7	Fundal posterior, grade II maturity	2.4 ± 307g	3 to 4	Oligohydramnios	4 weeks	Increased	0.8	Normal	1.7	Normal	0.6	Normal	1	Normal
62	74990	27	Multipara	Eclampsia	39 weeks	133	35 weeks 3 days	8.7	31.6	30.1	6.9	Left lateral, grade III maturity	2.6 ± 370 g	4 to 5	Oligohydramnios	4 weeks	Normal	0.8	Normal	1.8	Normal	1	Abnormal	10.9	Raised
63	79930	22	Multipara	Eclampsia	37 weeks 3 days	150	36 weeks 3 days	8.7	32.2	32.1	7.3	Fundal grade III maturity	2.9 ± 383 g	7 to 8	Adequate	Nil	Normal	0.7	Normal	1.4	Normal	0.6	Normal	1	Normal
64	74123	24	Multipara	GDM	40 WEEKS	149	39 WEEKS 2 DAYS	9.9	35.9	39.4	7.6	Posterior, grade III maturity	3.0 ± 450 g	7.7	Adequate	nil	Normal	0.6	Normal	1.2	Normal	0.5	Normal	1	Normal
65	75917	25	Multipara	Preeclampsia without severe features	32 weeks 3 days	144	26 weeks 6 days	7.7	28.3	23.6	5.5	Anterior, grade II maturity	1.1 ± 150 g	4	Oligohydramnios	6 weeks	Increased	0.5	Abnormal	1.3	Abnormal	0.8	Abnormal	1.6	Raised
66	89116	25	Multipara	GDM	37 weeks 3 days	140	37 weeks 1 day	9.2	30.9	32.6	7.2	Fundal posterior, grade III maturity	3 ± 447 g	19 to 20	Polyhydramnios	Nil	Normal	0.7	Normal	1.5	Normal	0.48	Normal	1	Normal
67	89029	18	Primi	Eclampsia	37 WEEKS 5 DAYS	140	32 WEEKS 3 DAYS	7.9	29	28.6	6.3	Anterior, grade II maturity	1.9 kg ± 310 g	4 to 5	Oligohydramnios	5 weeks	Normal	0.5	Abnormal	1.3	Abnormal	0.8	Abnormal	1.4	Raised
68	89783	26	Primi	Pre-eclampsia with severe features	35 weeks 3 days	144	29 weeks 6 days	7.7	28.3	23.6	5.5	Anterior, grade II maturity	1.1 ± 250 g	8 to9	Adequate	6 weeks	Increased	0.6	Abnormal	1.1	Abnormal	0.9	Abnormal	1.8	Raised
69	89393	37	Multipara	Preeclampsia without severe features	32 weeks 4 days	121	30 weeks 3 days	7.7	28	24.3	5.9	Anterior, grade II maturity	1.8 ± 210 g	6 to 7	Oligohydramnios	Nil	Normal	0.7	Normal	1.7	Abnormal	0.6	Normal	2	Raised
70	85935	20	Primi	Pre-eclampsia with severe features	37 WEEKS 4 DAYS	140	33 WEEKS 4 DAYS	8.1	30.2	29.8	6.5	Posterior, grade III maturity	2.2 ± 328g	5	Oligohydramnios	4 weeks	Normal	0.7	Normal	1.5	Abnormal	0.8	Abnormal	1.7	Raised
71	89156	25	Multipara	Preeclampsia without severe features	39 weeks 6 days	161	34 WEEKS	8.4	31.1	26.2	6.7	Fundal posterior , grade II maturity	2.7 ± 307g	3 to 4	Oligohydramnios	4-5 weeks	Increased	0.7	Normal	1.3	Normal	0.7	Normal	2.7	Raised
72	91643	24	multipara	Pre-eclampsia with severe features	39 WEEKS 1 DAY	140	35 weeks 5 days	9.1	32.7	31	6.4	Fundal posterior, grade III maturity	2.5 ± 328 G	10 TO 11	Adequate	4 weeks	Normal	0.7	Normal	1.3	Normal	0.7	Abnormal	1.6	Raised
73	93211	23	Primi	GDM	33 weeks 3 days	157	35 weeks 5 days	9.1	32.7	31	6.4	Fundal posterior, grade III maturity	2.8 ± 328 G	17 TO 18	Polyhydramnios	Nil	Normal	0.7	Normal	1.4	Normal	0.7	Normal	1.16	Normal
74	94699	22	Multipara	Eclampsia	37 weeks 6 days	144	36 weeks 1 day	7.9	30.2	25.4	6.5	Fundal left lateral, grade II maturity	2.8 ± 295 g	7 to 8	Adequate	Nil	Normal	0.8	Normal	1.7	Normal	0.6	Normal	1	Normal
75	100231	25	Primi	Eclampsia	34 weeks 5 days	126	33 weeks 2 days	8.5	30.7	27.5	6.4	Left lateral, grade II maturity	2.1 ± 277 g	9 to 10	Adequate	Nil	Normal	0.7	Normal	1.5	Abnormal	0.5	Normal	1.8	Raised
76	100533	25	Multipara	Preeclampsia without severe features	32 weeks 4 days	121	30 weeks 3 days	7.7	28	24.3	5.9	Anterior, grade II maturity	1.8 ± 210 g	5	Oligohydramnios	Nil	Normal	0.6	Abnormal	1.4	Abnormal	0.8	Abnormal	1.5	Raised
77	101233	20	Primi	Preeclampsia without severe features	37 WEEKS 2 DAYS	140	35 weeks 5 days	8.9	32.5	28.8	7.3	Posterior, grade III maturity	2.7 ± 390 g	9 to 10	Adequate	nil	Normal	0.7	Normal	1.4	Normal	0.8	Abnormal	1.2	Normal
78	102244	25	Multipara	Eclampsia	40 weeks	151	37 WEEKS 4 DAYS	9.1	32.7	33.9	7.3	Anterior, grade III maturity.	3.2 kg ± 475 g	10 to 11	Adequate	nil	Normal	0.6	Abnormal	1.3	Normal	0.9	Abnormal	1	Normal
79	106566	21	Primi	Eclampsia	30 weeks	155	24 weeks 1 day	5.9	22	18.6	4.4	Posterior, grade I maturity	824 ± 94 g	11 to 12	Adequate	6 weeks	Increased	0.6	Abnormal	1.5	Abnormal	1	Abnormal	3.2	Raised
80	107788	36	Multipara	Preeclampsia without severe features	37 weeks 6 days	156	33 weeks 6 days	8.4	30.6	28.6	6.6	Anterior, grade I maturity	2.1 ± 213 g	3 to 4	Oligohydramnios	4 weeks	Normal	0.6	Abnormal	1.5	Abnormal	0.8	Abnormal	1.7	Raised
81	112002	21	Primi	GDM	36 WEEKS 1 DAY	135	37 WEEKS 4 DAYS	9.1	32.7	33.9	7.3	Anterior, grade III maturity.	3.2 kg ± 475 g	13 to 14	Adequate	Nil	Normal	0.7	Normal	1.3	Normal	0.6	Normal	1.1	Normal
82	129901	20	Multipara	GDM	35 WEEKS 1 DAY	143	34 WEEKS 6 DAYS	8.4	30.4	31.6	6.8	Posterior, grade III maturity	2.8 ± 381g	11 to 12	Adequate	Nil	Normal	0.7	Normal	1.5	Normal	0.7	Normal	1	Normal
83	134491	28	Primi	Pre-eclampsia with severe features	39 weeks	133	35 weeks 3 days	8.7	31.6	30.1	6.9	Left lateral, grade III maturity	2.5 ± 370 g	4 to 5	Oligohydramnios	4 weeks	Normal	0.7	Normal	1.4	Normal	0.8	Abnormal	1.6	Raised
84	135661	20	Primi	GDM	42 weeks	157	40 WEEKS 2 DAYS	8.9	32.1	32.2	7.8	Posterior, grade III maturity	3.7 ± 464g	14 to 15	Adequate	Nil	Normal	0.8	Normal	1.7	Normal	0.4	Normal	1	Normal
85	135411	25	Primi	Pre-eclampsia with severe features	38 weeks 3 days	151	36 weeks 3 days	8.7	32.2	32.1	7.3	Anterior, grade III maturity	2.9 ± 350 g	10 to 11	Adequate	Nil	Normal	0.7	Normal	1.5	Normal	0.5	Normal	1.8	Raised
86	143435	20	Primi	Eclampsia	36 weeks 5 days	140	33 weeks 2 days	8.4	30.5	27.9	6.3	Fundal posterior, grade II maturity	2.0 ± 300 g	7 to 8	Adequate	3 weeks	Normal	0.8	Normal	1.4	Abnormal	1	Abnormal	3.2	Raised
87	144163	24	Multipara	Eclampsia	38 weeks 3 days	150	34 weeks 3 days	8.7	32.3	27	6.7	Fundal grade III maturity	2.4 ± 383 g	4 to 5	Oligohydramnios	4 weeks	Increased	0.6	Abnormal	1.2	Abnormal	1	Abnormal	2.5	Raised
88	145993	27	Multipara	GDM	35 weeks 2 days	140	36 weeks 3 days	8.7	32.2	32.1	7.3	Anterior, grade II maturity	2.9 ± 350 g	10 to 11	Adequate	Nil	Normal	0.8	Normal	1.7	Normal	0.6	Normal	1.2	Normal
89	144645	30	Primi	Preeclampsia without severe features	33 weeks 3 days	144	27 weeks 6 days	7.7	28.3	23.6	5.5	Anterior, grade II maturity	1.1 ± 250 g	4	Oligohydramnios	6 weeks	Increased	0.6	Abnormal	1.5	Abnormal	0.8	Abnormal	1.9	Raised
90	134668	25	Multipara	GDM	41 WEEKS 1 DAY	152	40 WEEKS 2 DAYS	8.9	32.1	32.2	7.8	Posterior, grade III maturity	3.1 ± 464g	14 to 15	Adequate	Nil	Normal	0.7	Normal	1.4	Normal	0.5	Normal	1	Normal

FLOW	CP Ratio	CP Ratio	Mean UTA PI	Mean UTA PI	Mean UTA RI	Mean UTA RI	FLOW UTERINE ARTERY	Feto-placental insufficiency	Utero-placental insufficiency	MODE OF DELIVERY	OUTCOME	IUD	BIRTH WEIGHT	BIRTH WEIGHT	APGAR SCORE	APGAR SCORE	NICU ADMISSION
Reduced	1.7	Normal	2	Raised	0.75	Abnormal	Normal flow	Present	Present	Normal vaginal delivery	UNEVENTFUL		2.5 Kg	Normal	1 - 7/10, 5 - 9/10		nil
Reversal	0.1	Reversal	1.45	Raised	0.65	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	Normal vaginal delivery	ADVERSE	IUD	830g	Nil	nil		nil
Normal	0.9	Reversal	1.7	Raised	0.7	Abnormal	Notching in left uterine artery	Present	Present	LSCS	ADVERSE		2.1	LBW	1-6/10, 5-8/10	Low	NICU Admission
Reversal	0.1	Reversal	1.6	Raised	0.5	Normal	Early diastolic notching in right uterine artery.	Present	Present	Normal vaginal delivery	ADVERSE	IUD	1.0 Kg	NIL	nil		nil
Reduced	1.4	Normal	1.5	Raised	0.65	Abnormal	Early diastolic notching of bilateral uterine arteries	Present	Present	LSCS	ADVERSE		1.5 Kg	LBW	1 - 7/10, 5 - 9/10		NICU Admission
Absent	0.6	Reversal	1.3	Raised	0.65	Abnormal	Normal flow	Present	Present	LSCS	ADVERSE		1.8 Kg	LBW	1-7/10, 5-9/10		NICU Admission
Normal	1.2	Normal	1.2	Normal	0.52	Normal	Normal flow			Normal vaginal delivery	UNEVENTFUL		2.9 Kg	Normal	1- 8/10, 5 - 9/10		nil
Normal	1.8	Normal	1.05	Normal	0.5	Normal	Normal flow			Normal vaginal delivery	UNEVENTFUL		2.8 Kg	Normal	1 - 8 /10, 5 - 9/10		nil
Normal	2.7	Normal	1.15	Normal	0.4	Normal	Normal flow			LSCS	UNEVENTFUL		2.7 Kg	Normal	1 - 9/10, 5 - 9/10		nil
Reversal	0.8	Reversal	1.5	Raised	0.7	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	Normal vaginal delivery	ADVERSE	IUD	890 g	NIL	NIL		nil
Reversal	0.6	Reversal	2	Raised	0.9	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		2	LBW	1-6/10, 5-8/10	Low	NICU Admission
Reduced	0.8	Reversal	1.6	Raised	0.65	Abnormal	Normal flow	Present	Present	LSCS	ADVERSE		1.9 KG	LBW	1-7/10, 5-9/10		NICU Admission
Absent	0.8	Reversal	1.3	Raised	0.6	Abnormal	Early diastolic notching of bilateral uterine arteries	Present	Present	LSCS	ADVERSE	IUD	1.2 kg	NIL	NIL		nil
Reduced	0.4	Reversal	1.35	Raised	0.65	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		1.7 KG	LBW	1-7/10, 5-9/10		NICU Admission
Reduced	0.8	Reversal	1.25	Normal	0.6	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		1.8 kg	LBW	1-7/10, 5-9/10		NICU Admission
Normal	1.1	Normal	1.15	Normal	0.4	Normal	Normal flow			LSCS	ADVERSE		1.7 KG	LBW	1-6/10, 5-7/10		NICU Admission
Reversal	0.8	Reversal	1.5	Raised	0.45	Normal	Right uterine artery notching noted	Present	Present	LSCS	ADVERSE		2.1 KG	LBW	1-5/10, 5-8/10	Low	NICU Admission
Reversal	0.8	Reversal	1.2	Normal	0.65	Abnormal	Early diastolic notching in right uterine artery.	Present	Present	LSCS	ADVERSE		2.2 KG	LBW	1-6/10, 5-8/10	Low	NICU Admission
Normal	1.3	Normal	1.25	Normal	0.5	Normal	Notching in left uterine artery		Present	LSCS	UNEVENTFUL		3.3 Kg	Normal	1-7/10, 5-9/10		NIL
Absent	0.4	Reversal	2	Raised	0.75	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		1.9 KG	LBW	1-7/10, 5-8/10		NICU Admission
Reversal	0.4	Reversal	1.45	Raised	0.65	Normal	Early diastolic notching in bilateral uterine arteries	Present	Present	Normal vaginal delivery	ADVERSE	IUD	1.1 KG	NIL	NIL		NIL
Reduced	0.8	Reversal	1.7	Raised	0.7	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		1.7 KG	LBW	1-7/10, 5-8/10		NICU Admission
Normal	1.2	Normal	0.8	Normal	0.5	Normal	Early diastolic notching in bilateral uterine arteries		Present	Normal vaginal delivery	UNEVENTFUL		3.0 kg	Normal	1-7/10, 5-9/10		nil
Reduced	0.6	Reversal	1.75	Raised	0.75	Abnormal	Early diastolic notching in right uterine artery.	Present	Present	LSCS	ADVERSE		2.2 KG	LBW	1-6/10, 5-8/10	Low	NICU Admission
Absent	0.7	Reversal	0.65	Normal	0.4	Normal	Normal flow	Present		LSCS	ADVERSE		1.6 kg	LBW	1-6/10, 5-8/10	Low	NICU Admission
Normal	1.3	Normal	0.75	Normal	0.5	Normal	Normal flow			Normal vaginal delivery	ADVERSE		3.0 KG	Normal	1-6/10, 5-7/10	Low	NICU Admission
Normal	2	Normal	1.25	Normal	0.5	Normal	Normal flow			Normal vaginal delivery	UNEVENTFUL		2.7 KG	Normal	1-7/10, 5-9/10		NIL
Absent	0.2	Reversal	1.15	Normal	0.5	Normal	Normal flow	Present		LSCS	UNEVENTFUL		2.5 KG	Normal	1-7/10, 5-8/10		nil
Normal	1.2	Normal	0.7	Normal	0.45	Normal	Notching in left uterine artery		Present	LSCS	UNEVENTFUL		3.2 KG	Normal	1-7/10, 5-9/10		NIL
Absent	0.5	Reversal	1.3	Raised	0.65	Normal	Early diastolic notching of left uterine artery	Present	Present	Normal vaginal delivery	ADVERSE	IUD	1 kg	NIL	nil		nil
Reversal	0.4	Reversal	0.65	Normal	0.4	Normal	Normal flow	Present		LSCS	ADVERSE		1.6 KG	LBW	1-7/10, 5-8/10		NICU Admission
Reduced	0.7	Reversal	0.75	Normal	0.5	Normal	Right uterine artery notching noted	Present	Present	LSCS	ADVERSE		1.9 kg	LBW	1-7/10, 5-9/10		NICU Admission
Normal	1.2	Normal	1.1	Normal	0.6	Abnormal	Early diastolic notching of left uterine artery	Present	Present	LSCS	UNEVENTFUL		2.5 KG	Normal	1-7/10, 5-9/10		NIL
Reversal	0.8	Reversal	1.8	Raised	0.7	Abnormal	Right uterine artery notching	Present	Present	LSCS	ADVERSE		2.1 kg	LBW	1-6/10, 5-8/10	Low	NICU Admission
Reduced	0.7	Reversal	0.9	Normal	0.5	Normal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	UNEVENTFUL		2.5 KG	Normal	1-7/10, 5-8/10		nil
Normal	1	Normal	0.65	Normal	0.4	Normal	Normal flow			LSCS	UNEVENTFUL		2.6 Kg	Normal	1-7/10, 5-9/10		NIL
Reversal	0.7	Reversal	2	Raised	0.8	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	Normal vaginal delivery	ADVERSE	IUD	900 g	NIL	nil		NIL
Normal	1.2	Normal	1.1	Normal	0.6	Abnormal	Early diastolic notching of left uterine artery	Present	Present	LSCS	UNEVENTFUL		2.7 KG	Normal	1-7/10, 5-9/10		NIL
Reduced	0.4	Reversal	1.4	Raised	0.7	Abnormal	Early diastolic notching of left uterine artery	Present	Present	LSCS	ADVERSE		1.6 KG	LBW	1 - 4/10, 5 - 6/10	Low	NICU Admission
Absent	0.6	Reversal	1.4	Raised	0.65	Normal	Early diastolic notching in bilateral uterine arteries	Present	Present	Normal vaginal delivery	ADVERSE	IUD	830 G	NIL	NIL		nil
Absent	0.2	Reversal	0.7	Normal	0.45	Normal	Normal flow	Present		LSCS	ADVERSE		2.3 KG	LBW	1-5/10, 5-7/10	Low	NICU Admission
Reversal	0.4	Reversal	2.55	Raised	0.7	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		2.1 KG	LBW	1-7/10, 5-8/10		NICU Admission
Normal	0.5	Reversal	1.05	Normal	0.55	Normal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		1.5 KG	LBW	1-6/10, 5-8/10	Low	NICU Admission
Absent	0.4	Reversal	1.4	Raised	0.55	Normal	Normal flow	Present	Present	Normal vaginal delivery	ADVERSE	IUD	980 g	NIL	nil		nil
Reversal	0.2	Reversal	1	Normal	0.35	Normal	Normal flow	Present		LSCS	ADVERSE		1.8 KG	LBW	1-7/10, 5-9/10		NICU Admission
Normal	1.4	Normal	1	Normal	0.55	Normal	Normal flow			LSCS	ADVERSE		3.3 KG	Normal	1-5/20, 5-6/10	Low	NICU Admission
Reversal	0.7	Reversal	2	Raised	0.8	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	Normal vaginal delivery	ADVERSE	IUD	900 g	NIL	nil		NIL
Reduced	0.8	Reversal	1.2	Normal	0.5	Normal	Early diastolic notching in right uterine artery.	Present	Present	LSCS	ADVERSE		2.5 KG	Normal	1-6/10, 5-8/10	Low	NICU Admission
Normal	2.2	Normal	1.5	Raised	0.65	Abnormal	Early diastolic notching in bilateral uterine arteries		Present	Normal vaginal delivery	UNEVENTFUL		2.7 KG	Normal	1-7/10, 5-9/10		NIL
Normal	1.4	Normal	1.4	Raised	0.4	Normal	Early diastolic notching in bilateral uterine arteries		Present	Normal vaginal delivery	ADVERSE		2.6 KG	Normal	1-6/10, 5-8/10	Low	NICU Admission
Reduced	0.9	Reversal	1.6	Raised	0.65	Abnormal	Normal flow	Present	Present	LSCS	ADVERSE		1.9 KG	LBW	1-7/10, 5-9/10		NICU Admission
Normal	1.8	Normal	1.7	Raised	0.65	Abnormal	Early diastolic notching in bilateral uterine arteries		Present	LSCS	ADVERSE		1.9 KG	LBW	1 - 4/10, 5 - 6/10	Low	NICU Admission
Reversal	0.8	Reversal	1.9	Raised	0.65	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		2.5 KG	Normal	1-4/10,5-6/10	Low	NICU Admission
Normal	1.5	Normal	0.65	Normal	0.4	Normal	Normal flow			LSCS	ADVERSE		3.6 KG	Normal	1-5/10, 5-6/10	Low	NICU Admission
Normal	1.1	Normal	1.15	Normal	0.3.8	Normal	Normal flow			Normal vaginal delivery	UNEVENTFUL		3.0 KG	Normal	1-7/10, 5-9/10		NIL
Absent	0.5	Reversal	1.6	Raised	0.65	Normal	Early diastolic notching in right uterine artery.	Present	Present	Normal vaginal delivery	ADVERSE	IUD	879 kg	NIL	NIL		Nil
Normal	2.2	Normal	1.5	Raised	0.7	Abnormal	Early diastolic notching in right uterine artery.		Present	LSCS	ADVERSE		3.5 KG	Normal	1-5/10, 5-7/10	Low	NICU Admission
Normal	1.7	Normal	1.15	Normal	0.4	Normal	Normal flow			LSCS	UNEVENTFUL		3.3 Kg	Normal	1-7/10, 5-9/10		nil
Normal	1.2	Normal	1.2	Normal	0.4	Normal	Normal flow			Normal vaginal delivery	UNEVENTFUL		2.9 KG	Normal	1-7/10, 5-9/10		nil
Normal	1.2	Normal	1.25	Normal	0.5	Normal	Normal flow			LSCS	UNEVENTFUL		3.1 KG	Normal	1-7/10, 5-9/10		Nil
Normal	1.7	Normal	2	Raised	0.75	Abnormal	Early diastolic notching of left uterine artery		Present	LSCS	UNEVENTFUL		2.5 Kg	Normal	1 - 7/10, 5 - 9/10		nil
Reversal	0.1	Reversal	1.4	Raised	0.65	Abnormal	Normal flow	Present	Present	LSCS	UNEVENTFUL		2.5 KG	Normal	1-8/10, 5-9/10		NIL
Normal	1.4	Normal	1.7	Raised	0.7	Abnormal	Early diastolic notching of left uterine artery		Present	Normal vaginal delivery	UNEVENTFUL		3.0 Kg	Normal	1 - 8/10, 5 - 9 / 10		nil
Normal	1.2	Normal	0.8	Normal	0.7	Abnormal	Normal flow			Normal vaginal delivery	ADVERSE	IUD	2.9 Kg	NIL	nil		NIL
Normal	0.8	Reversal	1.5	Raised	0.65	Normal	Early diastolic notching in bilateral uterine arteries	Present	Present	Normal vaginal delivery	ADVERSE	IUD	980 Kg	NIL	NIL		NIL
Normal	1.5	Normal	1.27	Normal	0.4	Normal	Normal flow			LSCS	ADVERSE		3.1 Kg	Normal	1 - 4/10, 5 - 6/10	Low	NICU Admission
Reduced	0.9	Reversal	1.55	Raised	0.7	Abnormal	Normal flow	Present	Present	LSCS	ADVERSE		2.0 Kg	LBW	1 - 4/10, 5 - 6/10	Low	NICU Admission
Reversal	0.6	Reversal	1.85	Raised	0.9	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	Normal vaginal delivery	ADVERSE	IUD	980 KG	NIL	NIL		NIL
Normal	0.8	Reversal	1.6	Raised	0.65	Abnormal	Normal flow	Present	Present	LSCS	ADVERSE		1.7 KG	LBW	1-7/10, 5-9/10		NICU Admission
Absent	0.8	Reversal	1.35	Raised	0.6	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		2	LBW	1-4/10,5-6/10	Low	NICU Admission
Reduced	0.4	Reversal	1.35	Raised	0.65	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	UNEVENTFUL		2.8 KG	Normal	1-7/10, 5-9/10		NIL
Reduced	0.8	Reversal	1.3	Raised	0.6	Abnormal	Normal flow	Present	Present	LSCS	UNEVENTFUL		2.5 kg	Normal	1-7/10, 5-9/10		nil
Normal	1.2	Normal	1.15	Normal	0.5	Normal	Normal flow			Normal vaginal delivery	UNEVENTFUL		3.0 KG	Normal	1-7/10, 5-9/10		NIL
Normal	1.7	Normal	0.7	Normal	0.45	Normal	Normal flow			lscs	UNEVENTFUL		2.6 Kg	Normal	1-7/10, 5-9/10		NIL
Normal	0.8	Reversal	1.2	Normal	0.4	Normal	Early diastolic notching in right uterine artery.	Present	Present	LSCS	ADVERSE		2.3 KG	LBW	1-6/10, 5-8/10	Low	NICU Admission
Absent	0.9	Reversal	1.25	Normal	0.6	Abnormal	Early diastolic notching of left uterine artery	Present	Present	LSCS	ADVERSE		1.8 Kg	LBW	1-7/10, 5-9/10		NICU Admission
Normal	1	Normal	1.55	Raised	0.7	Abnormal	Normal flow		Present	Normal vaginal delivery	UNEVENTFUL		2.6 Kg	Normal	1-7/10, 5-9/10		NIL
Normal	0.6	Normal	1.85	Raised	0.9	Abnormal	Bilateral uterine arteries - early diatolic notching		Present	Normal vaginal delivery	UNEVENTFUL		3.0 KG	Normal	1-7/10, 5-9/10		Nil
Absent	0.5	Reversal	1.6	Raised	0.8	Abnormal	Early diastolic notching in right uterine artery.	Present	Present	Normal vaginal delivery	ADVERSE	IUD	644 G	NIL	NIL		Nil
Reversal	0.8	Reversal	1.35	Raised	0.6	Abnormal	Early diastolic notching in right uterine artery.	Present	Present	LSCS	ADVERSE		2.2 KG	LBW	1-6/10, 5-8/10	Low	NICU Admission
Normal	1.1	Normal	1.2	Normal	0.5	Normal	Normal flow			Normal vaginal delivery	UNEVENTFUL		3.1 KG	Normal	1-7/10, 5-9/10		NIL
Normal	1.3	Normal	1.25	Normal	0.4	Normal	Normal flow			Normal vaginal delivery	UNEVENTFUL		3.0 kg	Normal	1-7/10, 5-9/10		NIL
Reduced	1	Normal	1.15	Normal	0.5	Normal	Normal flow	Present		LSCS	ADVERSE		2.2 KG	LBW	1-5/10, 5-6/10	Low	NICU Admission
Normal	1.7	Normal	0.7	Normal	0.45	Normal	Normal flow			LSCS	UNEVENTFUL		3.9 Kg	Normal	1-7/10, 5-9/10		NIL
Absent	0.8	Reversal	1.2	Normal	0.65	Abnormal	Early diastolic notching in right uterine artery.	Present	Present	LSCS	ADVERSE		2.8 KG	Normal	1-5/10, 5-6/10	Low	NICU Admission
Absent	0.5	Reversal	1.4	Raised	0.65	Abnormal	Early diastolic notching in bilateral uterine arteries	Present	Present	LSCS	ADVERSE		1.8 KG	LBW	1-7/10, 5-9/10		NICU Admission
Reversal	0.4	Reversal	0.65	Normal	0.4	Normal	Normal flow	Present		LSCS	ADVERSE		2.6 KG	Normal	1-6/10, 5-8/10	Low	NICU Admission
Normal	1.4	Normal	1.3	Raised	0.6	Abnormal	Early diastolic notching in bilateral uterine arteries		Present	Normal vaginal delivery	UNEVENTFUL		3.1 kg	Normal	1-7/10, 5-9/10		NIL
Reduced	0.7	Reversal	1.1	Normal	0.55	Normal	Early diastolic notching of left uterine artery	Present	Present	LSCS	ADVERSE	IUD	1.0 Kg	NIL	nil		nil
Normal	1.4	Normal	1.15	Normal	0.5	Normal	Normal flow			LSCS	ADVERSE		3.0 Kg	Normal	1-6/10, 5-7/10	Low	NICU Admission