“Abnormal Umbilical Artery Doppler Velocimetry Correlation With Placental Histopathology in Foetal Growth Restriction”

By: DR SWETHA S. MBBS

Dissertation submitted to the

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR – 563 101

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY (MS)

IN

OBSTETRICS AND GYNAECOLOGY

Under the Guidance of

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I, hereby declare that this dissertation entitled **"ABNORMAL UMBILICAL ARTERY DOPPLER VELOCIMETRY CORRELATION WITH PLACENTAL HISTOPATHOLOGY IN FOETAL GROWTH RESTRICTION**" is a bona fide and genuine research work carried out by me**, DR. SWETHA S**, in partial fulfilment of the requirement for the degree of Masters of Surgery (MS) in Obstetrics and Gynaecology, under the guidance of **Dr. RATHNAMMA P,** Professor, Department of Obstetrics and Gynecology, Sri Devaraj Urs Medical College, Tamaka, Kolar.

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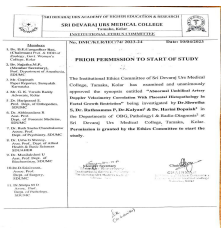

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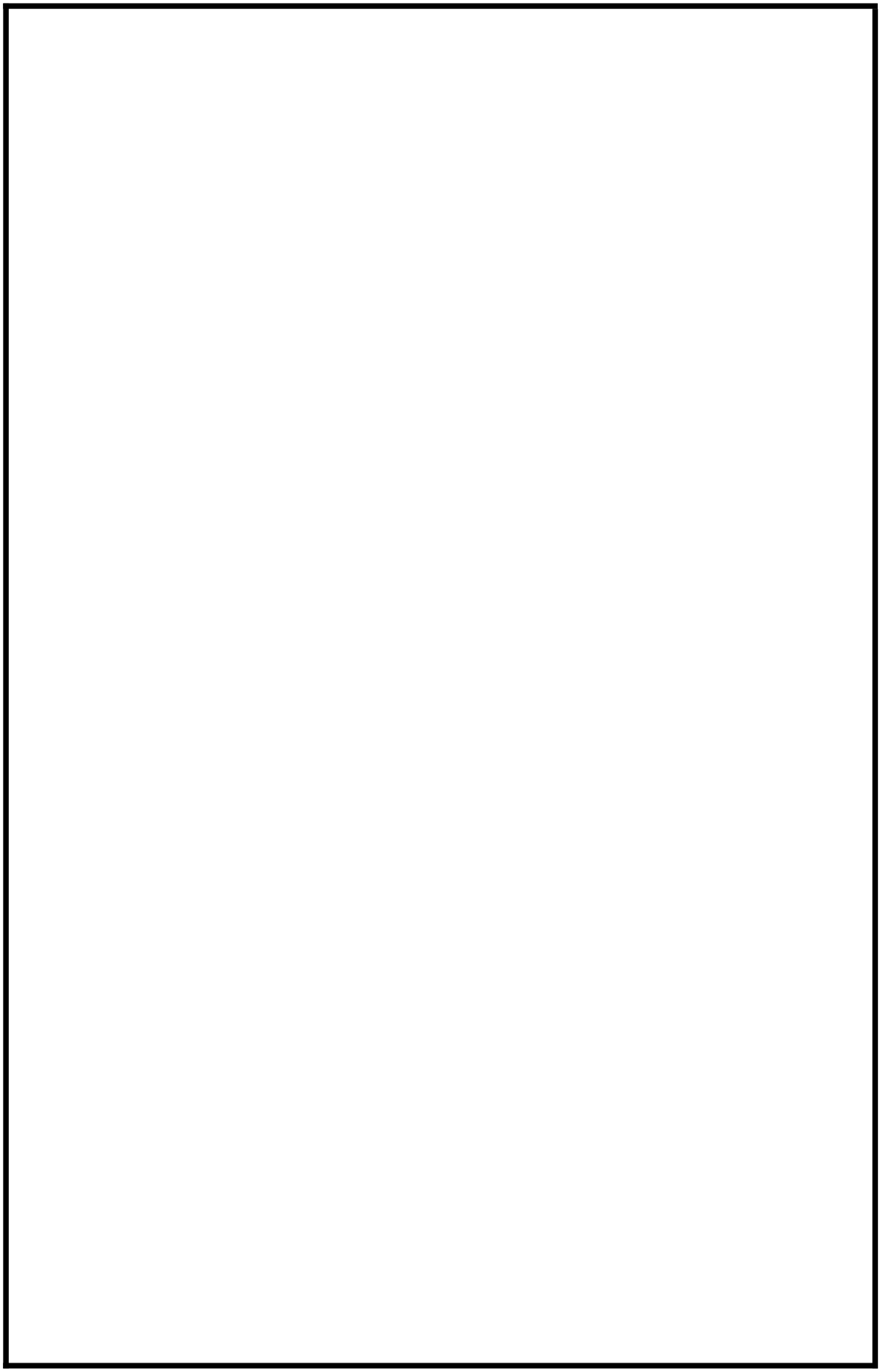

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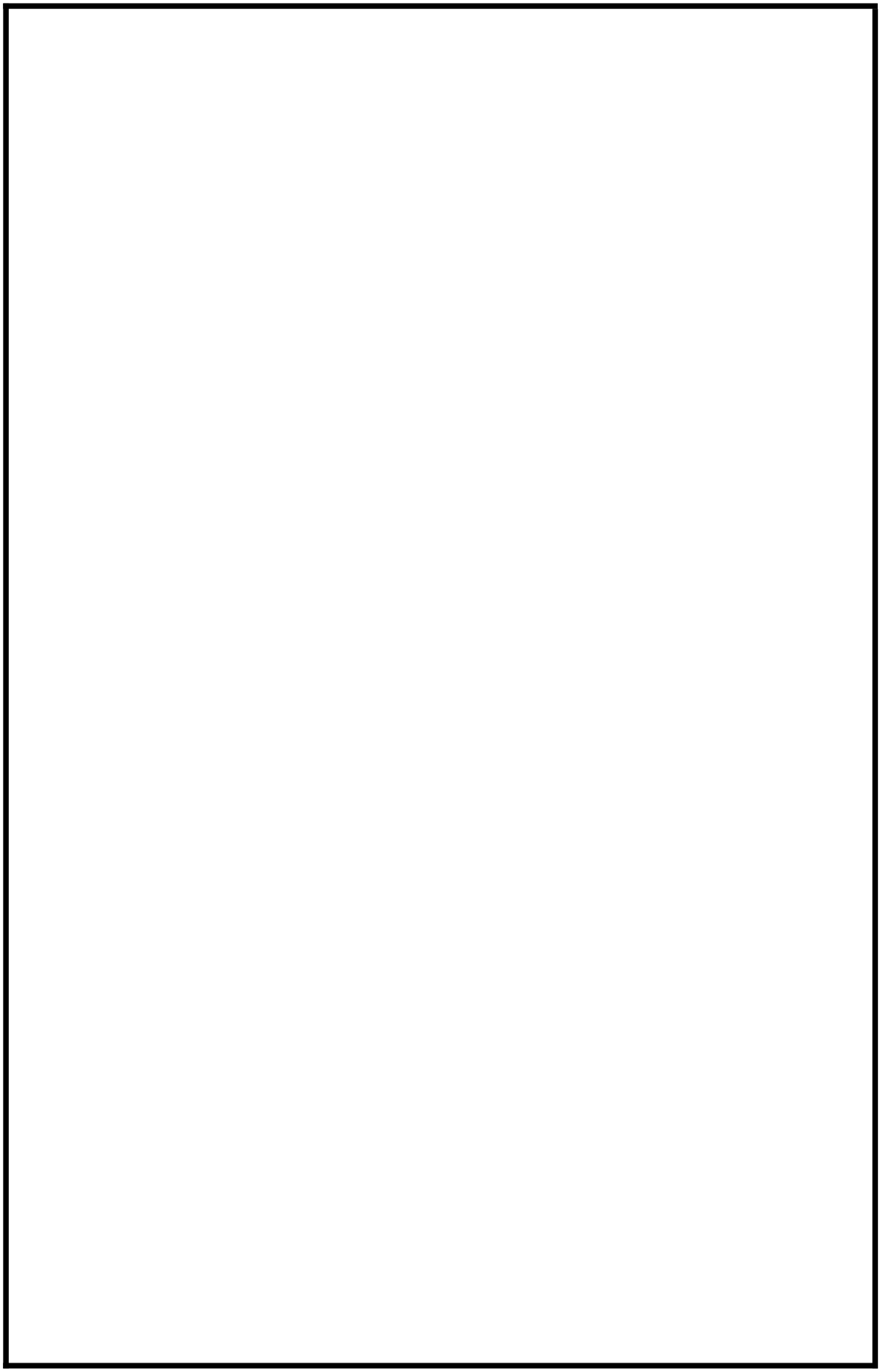

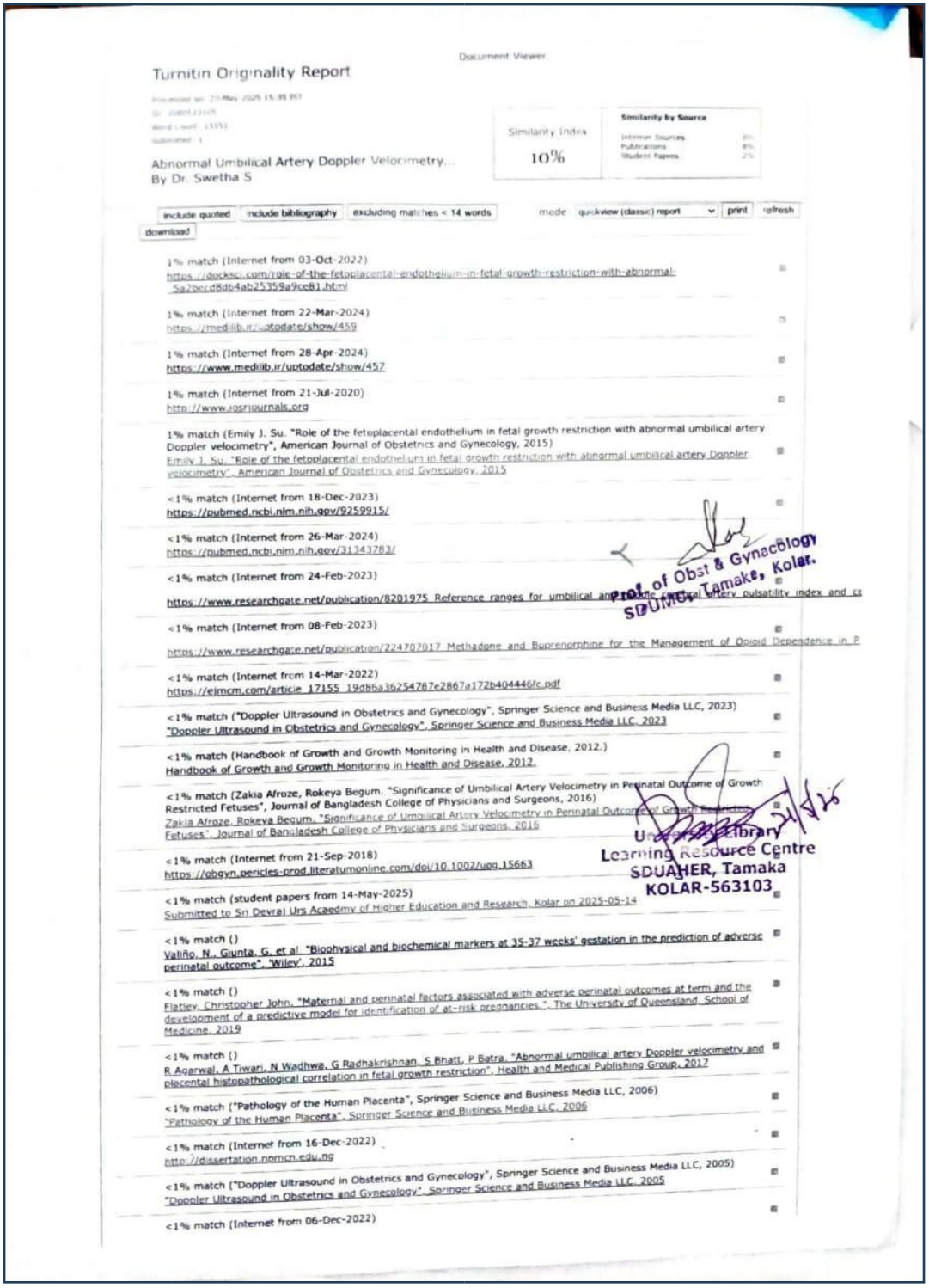

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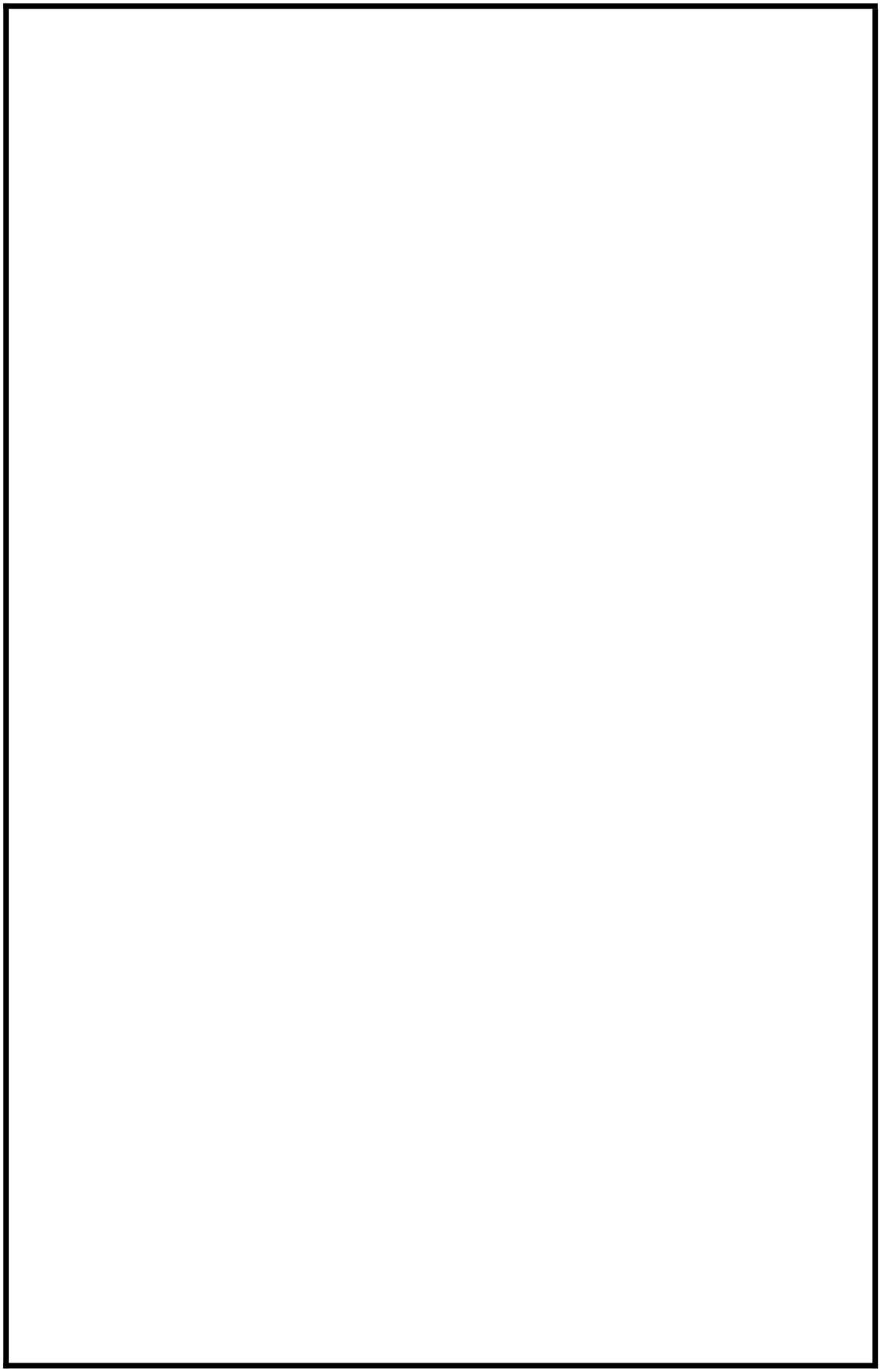

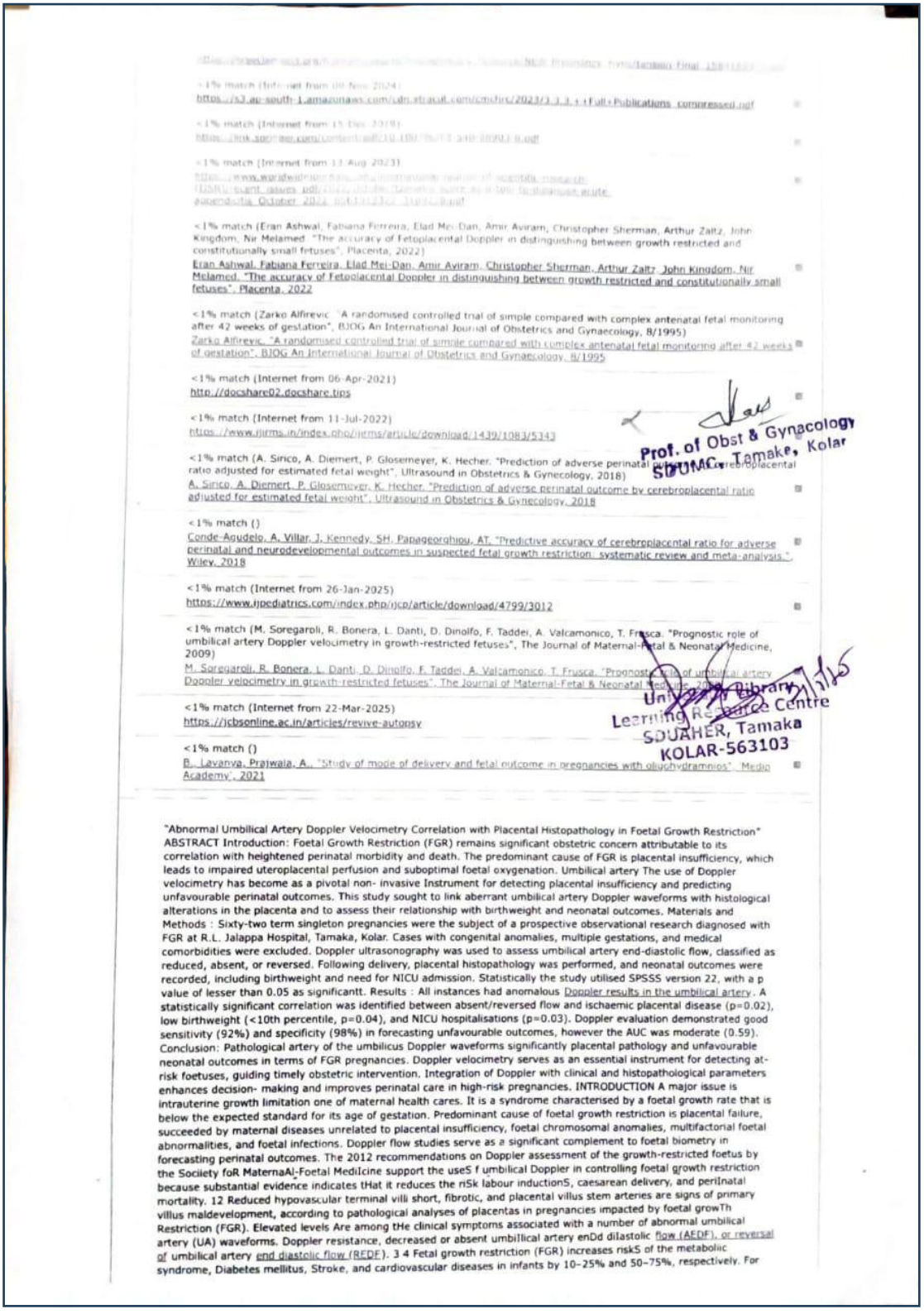

"Abnormal Umbilical Artery Doppler Velocimetry Correlation with Placental Histopathology in Foetal Growth Restriction"
ABSTRACT Introduction: Foetal Growth Restriction (FGR) remains significant obstetric concern attributable to its correlation with heightened perinatal morbidity and death. The predominant cause of FGR is placental insufficiency, which leads to impaired uteroplacental perfusion and suboptimal foetal oxygenation. Umbilical artery The use of Doppler velocimetry has become as a pivotal non- invasive Instrument for detecting placental insufficiency and predicting unfavourable perinatal outcomes. This study sought to link aberrant umbilical artery Doppler waveforms with histological alterations in the placenta and to assess their relationship with birthweight and neonatal outcomes. Materials and Methods : Sixty-two term singleton pregnancies were the subject of a prospective observational research diagnosed with FGR at R.L. Jalappa Hospital, Tamaka, Kolar. Cases with congenital anomalies, multiple gestations, and medical comorbidities were excluded. Doppler ultrasonography was used to assess umbilical artery end-diastolic flow, classified as reduced, absent, or reversed. Following delivery, placental histopathology was performed, and neonatal outcomes were recorded, including birthweight and need for NICU admission. Statistically the study utilised SPSSS version 22, with a p value of lesser than 0.05 as significantt. Results : All instances had anomalous Doppler results in the umbilical artery. A statistically significant correlation was identified between absent/reversed flow and ischaemic placental disease (p=0.02), low birthweight (<10th percentile, p=0.04), and NICU hospitalisations (p=0.03). Doppler evaluation demonstrated good sensitivity (92%) and specificity (98%) in forecasting unfavourable outcomes, however the AUC was moderate (0.59). Conclusion: Pathological artery of the umbilicus Doppler waveforms significantly placental pathology and unfavourable neonatal outcomes in terms of FGR pregnancies. Doppler velocimetry serves as an essential instrument for detecting at-risk foetuses, guiding timely obstetric intervention. Integration of Doppler with clinical and histopathological parameters enhances decision- making and improves perinatal care in high-risk pregnancies. INTRODUCTION A major issue is intrauterine growth limitation one of maternal health cares. It is a syndrome characterised by a foetal growth rate that is below the expected standard for its age of gestation. Predominant cause of foetal growth restriction is placental failure, succeeded by maternal diseases unrelated to placental insufficiency, foetal chromosomal anomalies, multifactorial foetal abnormalities, and foetal infections. Doppler flow studies serve as a significant complement to foetal biometry in forecasting perinatal outcomes. The 2012 recommendations on Doppler assessment of the growth-restricted foetus by the Society foR MaternaAl-Foetal MediIcine support the useS f umbilical Doppler in controlling foetal growth restriction because substantial evidence indicates tHat it reduces the riSk labour inductionS, caesarean delivery, and periInatal mortality. 12 Reduced hypovascular terminal villi short, fibrotic, and placental villus stem arteries are signs of primary villus maldevelopment, according to pathological analyses of placentas in pregnancies impacted by foetal growTh Restriction (FGR). Elevated levels Are among tHe clinical symptoms associated with a number of abnormal umbilical artery (UA) waveforms. Doppler resistance, decreased or absent umbilical artery enDd dilastolic flow (AEDF), or reversal of umbilical artery end diastolic flow (REDF). 3 4 Fetal growth restriction (FGR) increases riskS of the metabolic syndrome, Diabetes mellitus, Stroke, and cardiovascular diseases in infants by 10–25% and 50–75%, respectively. For

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Place: **DR SWETHA S**

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ABBREVIATIONS

1. **FGR** – Foetal Growth Restriction
2. **UA** – Umbilical Artery
3. **AEDF** – Absent End-Diastolic Flow
4. **REDF** – Reversed End-Diastolic Flow
5. **Doppler USG** – Doppler Ultrasonography
6. **NICU** – Neonatal Intensive Care Unit
7. **LSCS** – Lower Segment Caesarean Section
8. **HPE** – Histopathological Examination
9. **AFI** – Amniotic Fluid Index
10. **S/D Ratio** – Systolic/Diastolic Ratio
11. **PI** – Pulsatility Index
12. **RI** – Resistance Index
13. **CPR** – Cerebroplacental Ratio
14. **MCA** – Middle Cerebral Artery
15. **AUC** – Area Under the Curve

Abnormal Umbilical Artery Doppler Velocimetry Correlation with Placental Histopathology in Foetal Growth Restriction”

ABSTRACT

Introduction:

Foetal Growth Restriction (FGR) remains significant obstetric concern attributable to its correlation with heightened perinatal morbidity and death. The predominant cause of FGR is placental insufficiency, which leads to impaired uteroplacental perfusion and suboptimal foetal oxygenation. Umbilical artery The use of Doppler velocimetry has become as a pivotal non-invasive Instrument for detecting placental insufficiency and predicting unfavourable perinatal outcomes. This study sought to link aberrant umbilical artery Doppler waveforms with histological alterations in the placenta and to assess their relationship with birthweight and neonatal outcomes.

Materials and Methods :

Sixty-two term singleton pregnancies were the subject of a prospective observational research diagnosed with FGR at R.L. Jalappa Hospital, Tamaka, Kolar. Cases with congenital anomalies, multiple gestations, and medical comorbidities were excluded. Doppler ultrasonography was used to assess umbilical artery end-diastolic flow, classified as reduced, absent, or reversed. Following delivery, placental histopathology was performed, and neonatal outcomes were recorded, including birthweight and need for NICU admission. Statistically the study utilised SPSSS version 22, with a p value of lesser than 0.05 as significantt.

Results :

All instances had anomalous Doppler results in the umbilical artery. A statistically significant correlation was identified between absent/reversed flow and ischaemic placental disease ($p=0.02$), low birthweight (<10th percentile, $p=0.04$), and NICU hospitalisations ($p=0.03$). Doppler evaluation demonstrated good sensitivity (92%) and specificity (98%) in forecasting unfavourable outcomes, however the AUC was moderate (0.59).

Conclusion:

Pathological artery of the umbilicus Doppler waveforms significantly placental pathology and unfavourable neonatal outcomes in terms of FGR pregnancies. Doppler velocimetry serves as an essential instrument for detecting at-risk foetuses, guiding timely obstetric intervention. Integration of Doppler with clinical and histopathological parameters enhances decision-making and improves perinatal care in high-risk pregnancies.

INTRODUCTION

A major issue is intrauterine growth limitation one of maternal health cares. It is a syndrome characterised by a foetal growth rate that is below the expected standard for its age of gestation. Predominant cause of foetal growth restriction is placental failure, succeeded by maternal diseases unrelated to placental insufficiency, foetal chromosomal anomalies, multifactorial foetal abnormalities, and foetal infections. Doppler flow studies serve as a significant complement to foetal biometry in forecasting perinatal outcomes. The 2012 recommendations on Doppler assessment of the growth-restricted foetus by the Sociiety foR MaternaAl-Foetal MediIcine support the useS f umbilical Doppler in controlling foetal growth restriction because substantial evidence indicates tHat it reduces the riSk labour inductionS, caesarean delivery, and periInatal mortality. [12]

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Fetal growth restriction (FGR) increases riskS of the metaboliic syndrome, Diabetes mellitus, Stroke, and cardiovascular diseases in infants by 10–25% and 50–75%, respectively. For the mother and fetus to exchange gases, nutrients, and metabolites effectively, the placental microvasculature is necessary. The placenta's size is correlated with its ability to allow food transfer, and its bulk, dimensions, and morphology can vary greatly. A correlation between placental morphology and adverse pregnancy outcomes, such as fetal growth restriction (FGR), is shown by a decrease in placental size, surface area, and volume

Foetal development is tightly connected to placental development. Pathological analyses of placenta from pregnancy impacted foetal growth restriction (FGR) revealed primary villus maldevelopment, characterised by reducedD Placental villus Stem arterieEs and smalll, fibroticc, hypovascularly terminal villi. [5]

Clinically, a number of aberrant umbilical artery (UA) waveforms, such as reduced flow, absent end diastolic flow (AEDF), reversed end diastolic flow (REDF), and increased Doppler resistance, are correlated with pathological alterations in the placenta, such as infarcts, retroplacental haemorrhage, accelerated maturation, fibromuscular hyperplasia, villous oedema, and stromal fibrosis. The purpose is to connect Umbilical artery abnormality Doppler velocity with placental histopathology abnormalities.

Objectives of the study:

1. To correlate umbilical artery doppler velocimetry findings occurring in FGR with histopathological findings of placenta.
2. To correlate between umbilical Artery Doppler findings and perinatal outcomes.

REVIEW OF LITERATURE

Antepartum foetal Assessment

The contraction stress tesTt, biophysical profiles, modified biophysical profile, non stresss test, and foetal movements counts are the main techniques used to evaluate the fetus. Together with the biophysical profile and modified biophysicall profilee, the assessmentt of the amniiotic Fluid content and The dopppler velociimetry of The foetal umbilical veins gives more enlightment about the health of the fetus. Despite the fact that these tactics are frequently used, there is insufficient eviIdence to support theiIr best useS AND effectiveness in improviIng perinatIal outcomEes**.**

TECHNIQUES OF FETAL ASSESSMENT

Counting of Foetal movements

Evidences that fetal movements decrease in response to foetal hypoxemia supports the objective maternal assessment of foetal movements, also referred to as "foetal kick counts." . Although there is consensus that individuals exhibiting reduced foetal movement require

further foetal evaluation, randomised studies assessing foetal movement counts as a measure of foetal well-being have not yielded definitive data supporting the efficacy of this intervention. Furthermore, the existing research does not substantiate a definitive threshold for foetal movement or a "alarm limit" that would signify an elevated risk of embryonic demise or harm. [6]

Techniques of Cardiotocographic

Non-stress test

Two observations served as the foundation for thRe non - stress test (NST): first, thaTt the presenTce of twoo or moore foetal heart rate (FHR) accelerationss was often indicative of a negatiive contractiion Stress test (CST); and second, thaat the lack of accelerations on a baselinee Fetal heart rate tracing was related to poorer perinatall outcome. Foetal heart rate accelerations, whether spontaneous or induced (such as by vibroacoustic stimulation), reliably signify adequate foetal autonomic function, the lack of acidosis, and the absence of neurological depression. A meta-analysis of randomised studies failed to definitively establish that prenatal cardiotocography enhances perinatal outcomes, since the quality of the evidence was inadequate or exceedingly low[6].

The primary benefit of the NST compared to the CST is its lack of necessity for an intravenous line, oxytocin, or contractions. A false positive nonstress test (NST) is nonreactive result which is subsequently confirmed with a conventional baackup test, suchh as negative contraction stress test (CST) and a high biophysical profile (BPP) score; a false-negative NST transpires within a week following a reactive test. A downside is that the rates of false positives and false negatives exceed that of the CST. [7]

Test of contraction stress

The CST is based on the fetal reactions to a temporary decrease in oxygen supply during uterine contractions.. The Fetal heart rate may naturally drop in event that fetus suffers from hypoxaemia (foetal arterial pO2 drops below 20 mmHg), which could manifest clinically as late decelerations.. The alteration in foetal heart rate is facilitated by sympathetic and parasympathetic fibres to the cardiac and cerebral arteries, in addition to foetal chemoreceptors and baroreceptors.

The need to give intravenous oxytocin to induce contractions, the fact that some conditions (like placenta previa) make it inappropriate to induce contractions, and the high false-positive rates (i.e., the foetus continues to undergo labour without changes in fetal heart rate requiring intervention) are the main reasons the CST is rarely performed. The False Negative rates, defineed ass the Incidence of foetal demise Within a weeek following a negative teest results, is exceedingly low after a normal test outcome, ensuring adequate foetal oxygenation.[8]

Sonographic methods

Both the biophysical profile and the modified biophysical profile

By assigning points to the following criteria—amniotic fluid volume (AFV), reflexes/tone/flexion-extension movements, fetal breathing movements, and fetal body movementsthe BPP combines the NST with ultrasonographic fetal examination. This test assesses both chronic hypoxia (AFV) and acute hypoxia (NST, respiration, body movement, and tone). There is a strong correlation between the fetal pH and the BPP score.[9]

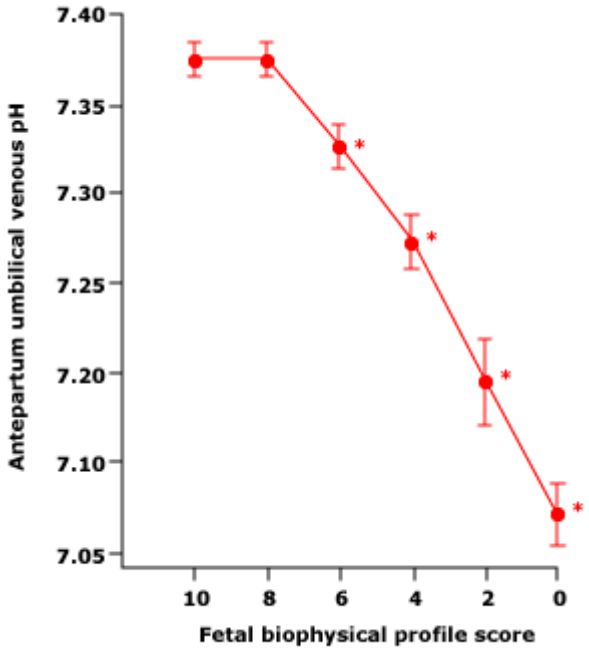

Figure: The relationship between the foetal umbilical venous pH (±2 SD) as assessed by cordocentesis and the foetal biophysical profile score The relationship was inversely, linear, and very significant (R2 0.912; p <0.01).

●Modified BPP (mBPP)

The mBPP comprises the evaluation of AFV, which assesses long-term oxygenation, and the NST, which examines acute Oxygenation. If the deeepest vertiical pockeet of amniootic fluiid is lesss than 2 cm, the NST is non reactive, or both are present, the mBPP is considered abnormal. The assertion that sonographic evaluation of amniotic fluid volume as a supplement to the non-stress test improves sensitivity (i.e., reduces the incidence of false-negative reaction tests) is substantiated solely by low-quality research. According to small retrospective investigation, pregnancy with Reactiive NST And a loo w Amniotic Fluid Index (0 - 5 Cm) were more likely to have meconium passage and a five minute Apgar score of less than 7 at birth than pregnancies with a normal amniotic fluid index. [10]

A research on post term pregnancy indicated that a greater incidence of foetal distress correlated with varied decelerations and reduced amniotic fluid volume, despite a reactive non-stress test; nonetheless, the efficacy of the combIined test waas comparable to that of each test individually. [11]

Assessment of amniotic fluid volume

A normal backup test after a low score results in a false-positive; an antepartum stillbirths within a weeek of a hiigh score results in a false-negative BPP or mBPP.. The BPP and mBPP have high false-positive rates but low false-negative rates.

DOPPLER VELOCIMETRY

Overview

An understanding of bloood floow in the uterus the fetus's reactions to physiological stresses may be gained by measuring the rates of blood flow in the mother's and fetus's arteries. The fetoplacental circulation gradually changes haemodynamically as a result of the placenta's abnormal vascular development, which is evident in preeclampsia. When the placental vascular tree is damaged to 60–70%, the umbilical artery's Doppler indices rise; as a result, the foetal middle cerebral artery's impedance falls and the foetal aorta's Resistance increases,

preferentially directing bloood too thee growing braiin , hearrt. Ultimaately, resistance escalates foetal venous system (ductus venosus, inferior vena cava), resulting in the cessation or reverrsal of end diastolic flow of umbilical artery. These alterations take place throughout a variety of time periods and are linked to fetal acidity. [12]

Unlike the majority of other prenatal screening methods, Doppler-based exams have been thoroughly tested in randomised studies. Depending on the specific vessel under investigation, the velocity waveform data varies.

Umbilical artery

When a foetus exhibits development limitation resulting from uteroplacental inadequacy, Doppler assessments The umbilical arteries are quite beneficial. The waveform pattern of the umbilical artery shows a low-resistance system that is characterized by constant forward blood flow throughout the cardiac cycle. Growth-restricted fetuses have decreased, nonexistent, or eveen reversedd diastoliic floow in the umblical arteery, whereas appropriately growing fetuses show raised diastolic flow in the umbilical artery flow velocity waveforms. The progressive degradation of tertiary villi is associated with the gradual reduction in umbilical artery diastolic flow. Abnormal end-diastolic flow in growth-restricted fetuses is associaated wiith fetal hypoxemia and acidemias, as well as increasedd postnatal morbiditty and death.[13]

Umbilical artery Doppler evaluation tests are recommended by the Ameriican College of Obstetricians and Gynecologists' practice guidelines treating suspected fetal growth restriction, but not for fetuses that are developing normally. When evaluating growth-restricted foetuses, regular foetal evaluations including biophysical profiles (BPP) and/or non-stress tests (NST) should be used in conjunction with umbilical artery Doppler. Using different criteria for "high risk," a comprehensive assessment of 16 randomised studies including almost 10,000 high-risk patients demonstrated that Doppler ultrasonography variably decreased perinatal death (perinatal mortallity: 1.2% versuus 1.7%, ods ratio 0.71, 95% CI 0.52-0.98, number required to treeat 203). [14]

Middle cerebral artery

Doppller measurement of thee foetal miiddlle cerebrral arrtery peak systoolic velocity (MCA-PSV) is thee beest way to identify foetal anemia in high-risk pregnancies, particularly those affected by RhD allloimmuniization.

MCA Doppler is being investigated as an additional technique to assess growth-restricted pregnancies. This application is predicated the idea that systemiic blood floww in these foetuses is diverted from the periphery to the brain, and that Doppler evaluation of flow velocity in the fetal MCA may reveal this brain-sparing effect.

The cerebroplacentaal ratiio (CPR) is defined as the ratiio of doppler indices of Middle Cerebral artery (MCA) those of the umbilical artery, it is being investigated as a possible predictor of adverse outcomes. Its use in low-risk pregnancies is minimal, and its effectiveness in cases of growth limitation remains ambiguous. [15]

Venous system

Many cardiovascular dysfunctions can cause abnormal venous Doppler readings. This includes abnormalities in heart rhythm and pace, substantial elevations in cardiac afterload, and decreased cardiac compliance and contractility. Venous Doppler velocimetry's therapeutic value is therefore particularly important in foetal conditions that show signs of heart problems and/or severe placental insufficiency. These conditions include twin-twin transfusion syndrome, foetal hydrops, foetal arrhythmia, and foetal development restriction due to placental insufficiency.

Althoough fllow velociity waveform haave beeen recorded foor a number of venoous systems, the ductus venosuses, inferioor vena cava, and umblical vein are the vessels that are moost frequently evalluated in clinical practices. When a pregnancy is above 15 weeks gestation, the umbilical vein continues to circulate blood. Pulsatility in umbilical vein flow may be a sign of cardiac dysfunction linked to increased afterload in pathological circumstances such as foetal growth restriction. Even in situations of extreme growth limitation, the ductus venosus resists changes in flow and regulates the fetus's oxygenated blood. [16]

Uterine artery

In third trimester in Patients With challenging pregnancies the use of uterine artery Doppler has been studied by numerous researchers, although the results are still unclear. As

pregnancy progresses, the uterine arteries' flow impedance frequently decreases. Elevated resistance to uterine arterial blood flow and prolonged high-pressure uterine circulation are the results of Insufficient trophoblaastic invasions modification of maternal spiraal arteriies. Decreased blood flow in mother's placenttal compartment is suggested by eleevated resistannce indiices and persistentance of uteriine artery notchiing during weeks 22 and 24 of pregnancy. These disorders are linked to the onset of preeclampsia, foetal growth limitation, and perinatal mortality. [17]

Doppler ultrasonography of the umbilical artery for foetal monitoring

Frequency alterations induced by sound waves reflected from circulating erythrocytes are used to create Doppler ultrasound waveforms. Numerous elements of circulation, such as the volumee the floow, velocity profile, circulattory impedances (i.e., the resistance to pulsatiile floow), and the presence and direction of flow, can be determined using a Doppler waaveform analysiis. Some hiigh-risk pregnancy, obstetricians can lower perinatal mortality by usiing Dopplerr assessment of umblical artery in combination with additional foetal monitoring methods and suitable interventions. [18]

In pregnancies impacted by Twin anemia-polycythemia sequence and twin-twin transfusion syndrome, hypertension, foetal growth restriction (FGR), the Doppler evaluation of uterine artery flow is an essential component of foetal monitoring.

DOPPLER ASSESSMENT OF UMBILICAL ARTERY IN UNCOMPLICATED PREGNANCIES

The vascullar systems of the foetus gradually grows over the course of a normal pregnancy, guaranteeing enough nourishment and oxygen supply from The placenta facilitates the growth and welfare of the foetus. As umbilical blood flow progressively increases, foetal placental arterial impedance steadily diminishes. The noormal vascuular change indicated as progressive decreease in umblical artery Doppler indices. In middle of 14 and 16 weeks pregnancy, end-diastolic flow (EDF), which is forward flow in umbilical arteryy at end of diastole, is seen. It gradually increases in intensity to provide a steady supply oxygen ,nutrients growing fetus throughout cardiac cycle.

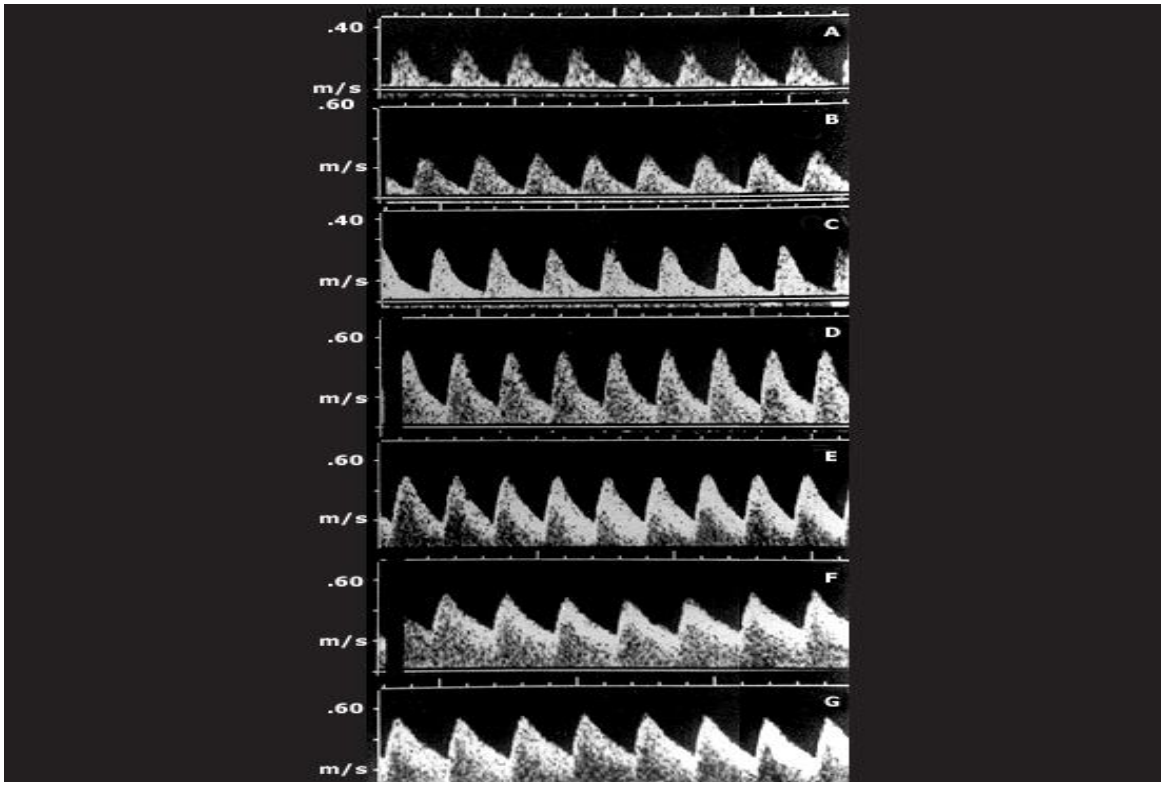

Figure: Gestational age's effect on umbilical arteries As gestational age increases, Doppler frequency shift waveforms are arranged in panels vertically from top to bottom.

16 weeks are needed for A, 20 weeeks for B, 24 weeeks for C, 28 weeeks for D, 32 weeeks for E, 36 weeeks for F, and 40 weeeks for G. Observe the progresssive increasee in ennd-diastoliic velocityy and accompanying decrease in pulsatilityy as gestationn progresses.

Doppler Assessment of Umbilical Artery in Foetal Growth Restriction

Foetal growth restrictiion (FGR) in the end--diastolic flow (EDF)) of the umbilical artery is reduced in chronic placental insufficiency. Clinical evaluation and treatment of FGR are based on the sequential foetal compensatory haemodynamic responses that accompany the resulting foetal oxygen and nutrition deficit. [19]

This is frequently a gradual process: ●A drop in EDF velocity and a rise in UA Doppler indices demonstrate an initial rise in impedance inside the fetoplacental circulation.

The perfusion of vital organs, such as the brain, heart, and adrenal glands, is prioritized during extended periods of deprivation, at the expensse of circulation too muscles, visceraa,

skinn, and otherr non-essential organss. A diminished cerebroplacental ratio (CPR), representing the ratio of middle cerebral artery (MCA) to umbilical artery (UA) Doppler indices, signifies cerebral redistribution. [20]

As fetoplacental vascular impedance rises, the end--diastolic fllow mayy becomee absent (A E D F) or reversed (R E D F), signifying a worsening fetal state. Typically, these alterations in EDF manifest one week prior to a significant decline.

The increasing deterioration of the foetal health is indicated by the lack or reversall of ductuss venosuss atrial wave,, decreased and eventuallly nonexistent fetal cardiac variabilityy and reactivityy, and the cessation of fetal movements and respiration—alll of which are signs of impending foetal distress.

This sequence, while prevalent, is not guaranteed. In several instances, an initially elevated Doppler index may gradually decline as pregnancy advances, potentially signifying a much enhanced prognosis; yet, the baby continues to be susceptible to negative consequences.

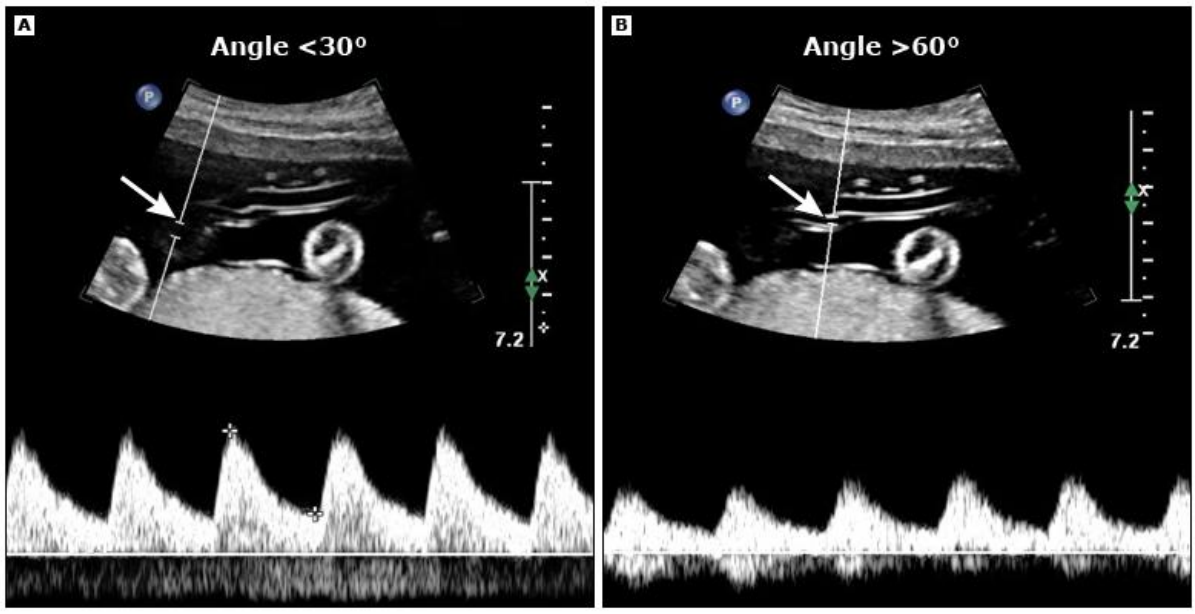

Figure- Normall Dopplerr waveform of the umbilical artery - typical waveform of the umbilical artery. The Doppler affects the Doppler shift, which peaks when the angle is almost 0 degrees and the Dopppler beam paath (shown by the cursor- line) is paralllel to the vessell axis (Panel A). The Doppler shifts will quantify the actual blood flow velocity in this context. Conversely, when the beam path is orthogonal to the vessel axis (at a 90-degree angle), the shift will be minimal, resulting in an underestimation of the velocity (Panel B).

Two Doppler sample site locations are shown by the slant arrows along the same umbilical cord segment.

Interpretation of UA Doppler indices

Doppler indices can be subjectively examined determined by the presence or absence of UA EDF, or statistically evaluated according to their singular threshold value or distribution.

Quantitative interpretation

A S/D ratio of 3.0 or a RI of 0.6 at or after 28 weekss of gestationn are the best singlee-point criteria, according to several doctors, for iidentifying pregnanciess at increased risk of unfavorable outcomess. One commonn technique is to usee the percentile distribution of Doppler values according to gestational age. An abnormal UA Doppler is indicated by a Doppler index (S/D, PI, or RI) beyond the 95th percentile for gestational age.

Selecting the optimal nomogram may prove to be difficult. Significant volatility and poor methodological quality were found in a systematic review of observational studies, highlightingg the need for reliablee referencee rangess 20. More reliable nomogramss, obtained fromm prospective, welll planned longitudinall studiess in diverse and low--risk populations, are commonly used in ultrasounnd reportinng systemss. (23)

Qualitativee interpretationn

Adverse perinatal consequences are linked to Fetoplacental circulatory impairment caused by increased vascular resistance is indicated by the absence and reversal of ennd-diastollic flow velocitiess (A E D F and R E D F) umbilical artery (U A). Risk classification of pregnancy dictates occurrence AEDF or REDF. The prevalence of AEDF or REDF exhibited significant variability, ranging from 0.08 to 2.13 percent, based on a comprehensive study of 42 studiess including overr 18 ,000 predominantly low--risk or unseelected individuals fromm high-income nationss. Between two and fifty-six percent of pregnancies are high-risk. The wide variations are explained by the various risk classification criteria, the seriousness of the under lying obstetrics conditions, like growthh limitation, and the Doppler imaging techniquee, specifically the high--pass filter seting. [22]

According to meta--analysis assessing risk fetall death instances with foetal growthh restrictioon (FGR) beforee 34 weeeks of gestationn, stillbirth rates were 6.8 % for umbilical artery abseent end--diastolic flow (UA A E D F) and 19 % for reversedd end--diastolic flow (R E D F). A greateer frequency of aneuploidy and congenital abnormalities is related with both AEDF and REDF. Based on an analysis of 1,126 cases of A E D F and R E D F from the literature , the stilllbirth and infant death rates were 17 % and 28 %, respectivelyy.

Most death were linked to fetal growth restriction (F G R), pre term birth, congenital defects, and aneuploidyy, specifically trisomy 13 , 18, and 21 .

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Lack of end-diastolic velocity and birth defects

CARDIOVASCULAR SYSTEM	Ventricular septal defect
	Double outlet right ventricle
	Ebsteins anamoly
	Arrythmia – congenital heart block
	Hypoplastic left heart syndrome
Central nervous system	Holoprosencephaly
	Hydrocephaly
	Agenesis of corpus callosum
Urogenital system	Hydronephrosis
	Renal agenesis
Gastrointestinal system	Omphalocele
	Gastroschisis
	Esophageal atresia
Skeletal system	Polydactyly

AEDF can happen sometimes. Even while these foetuses have superior perinatal outcomes compared to those with chronic AEDF, they nonetheless carry a high risk; around 34% of them are delivered because of unsettling foetal abnormalities, and more than 50% have a higher rate of newborn illness.[25]

ASSESSMENT OF EFFICACY

Pregnancy risks that are high

A meta-analysis of randomised trials (16 trials, over 10,000 pregnancies) concerning high-risk pregnancies demonstrated that the utilisation of foetal and umbilical Perinatal mortality

was considerably reduced by Doppler ultrasonography (1.2 % versus 1.7 %, risk ratio [R R] 0.71 , 95 % CI 0.52 - 0.98), with 203 (95% CI 103-4352) treatments required to prevent one fatality.. Supplementary advantages encompassed a reduction in caesarean deliveriies (R R 0.90 , 95 % C I 0.84 - 0.97) and inductions (R R 0.89 , 95 % CI 0.80 - 0.99). Thiss study was unable to identify the high--risk group that would benefit most from umbilicall arteryy (UA) Doppller surveilllance.

. A previous meta-analysis looked at this issue and showed that Doppler surveillance primarily benefited pregnancies with issues linked to pregnancy-associated hypertension and/or fetal growth restriction (FGR). [26]

Due to the differences in the clinical and pathophysiological features of late-onset and early-onset FGR, UA Doppler may have reduced predictive capability for worse perinatal outcomes in FGR pregnancies beyond 32 completed weeks compared to those identified earlier. Nonetheless, the matter continues to be contentious, particularly about the gestational age standard employed for categorisation . Compared to umbilical artery (UA) Doppler indices alone, redistribution of feotal blood flow, as seen by middlle cerebral arteryy (MCA) Dopplerr and the cerebro placental ratio (C P R), may be a better indicator of poorer perinatal outcomes in late-onset foetal growth restriction (FGR). [27]

However, no randomized studies have been conducted to support effectiiveness MCA Doppler , CPR. In a prospective cohoort study (TRUFFLE-2) involving singleeton pregnanciies risk oof foetal growth restriction (FGR) during late preterm gestations, cerebral floow redistribution—defined as an MCA Doppler measurement below the 5th percentile and a high gestational age-specific umbilicocerebral ratio (UCR), the inverse of the cerebroplacental ratio (CPR)—showed the highest relative risk for composite adverse outcomes. The connection remaineed statiistically siignificant after adjustiing biirth weight, gestatiional agee at biirth. A randomised investigatiion is under underway to validate the efficacy of MCA Doppler in conjunction with CPR or UCR in clinical practice, as shown by this study. [28]

The fetoplacental vasculature in FGR

FGR is frequently caused by maternal hypoperfusion of the placenta. Nonetheless, the fetoplacental vasculature is essential to foetal development as it plays a significant role in placental perfusion. A cohort of 34 growth-restricted foetuses illustrates this; 21 of these

pregnancies had normal uterine artery Dopplers, despite the fact that all of them had unusually reduced diastolic flow velocities of umblical artery. Therefore, even when mother's uteroplacental blood floow normal, aberrant umbilical artery Doppler velocimetry and foetal development problems may still occur. [29]

Small placrenta, avasculaar termiinal villli, fibriinoid necrosiis, many villlous infaarcts are common placental pathological characteristics foetal groowth restriiction (FGR). However, whether the umbiilical artery end-diastoliic velocity is absent, reversed, or retained frequently affects other clinical characteristics .Placentas from FGR-affected pregnancies wiith AEDV/REDV far likeely haave marginal corrd insertions than placentas from growth-restricted pregnancies with intact diastolic velocitie, including those with increased S/D ratios.

The quantity of abnormal vessells is significantlly associated with fetoplacental vascular resistance, and Stem Villous arteries froom placenta impacted by FGR with AEDV/REDV demonstraate lumiinal occlusion and concenttric thickening of the intiimal ,mediial walls.[30]

The presence or absence of end-diastolic velocities also affects the microvascular characteristics of FGR placentas. Individuals exhibiting sustained end-diastolic velocities possess capillary beds that are either normal or exhibit increased branching. Conversely, terminal capillaries are slender and elongated FGR placenta exhibiting AEDV/REDV, and branch mature interrmediate villli predominantly absentt. Elevated fetoplacental vascular resistance is a result of this reduction in peripheral villous vasculature.

Fetoplacental endothelium

The fetoplacental endothelium is at least partially responsible for our understanding of the processes behind these placental pathologic features. By controlling vasomootor tonee, balanciing pro- and antiicoagulant actions, reducing inflammmatory mediator, changing celllular ,nutritional transit, promoting angiogenesiis, the endothelieum playss a crucial role in vascular physiology. 54 Additionally, endothelial cells exhibit tissue-specific behaviours as a result of their local environment. One of the endothelium's main characteristics is its variability.

At term gestation, the human placenta's endothelium covers 15 square meters and reaches a length of around 550 kilometres. The endothelium in the placental vasculature and the

umbilical cord can have rather different phenotypes, even though they are located in the same organ and in continuity. The endothelium of the human umbilical artery and the umbilical vein within umbilical cord are not the same. Following the cultivation of separately isolated endothelial cells from both sources and the implementation of shear stress, human umbilical vein endothelial cells exhibit much lower expression of the vasoconstrictor endothelin-1 than do umbilical artery endothelial cells. This might be one of the mechanisms that help the umbilical vein stay properly open during periods of high flow. [31]

An other example of the variety of endothelial cells found in the placenta is the differentiation between macrovascular umbilical vein endothelial cells and placental microvascular endothelial cells.

In terms of function, placental miicrovascular endotheliial cells secrete moore prostanoids thaan umbilical vein endothelial cells. These include 6-keto prostaglandin F1α, a stable metabolite of the vasodilator prostacyclin, and thromboxane B2, a stable metabolite of the vasoconstrictor thromboxane A2. Compared to umbilical vein endothelial cells, placental microvascular endothelial cells multiply more in response to VEGF. Some researchers assert thaat Human Umbilical vein endothelial cell, the most often employed endotheliall cell type in studies, may not be the ideal model for examining the biology of placental endothelial cells. A more effective model of inquiry would use endothelial cells isolated from a specific region of the placentas (e.g., arteriial vs venoous and Macrovascular versus Microvascular) that is most pertinent specific topic research. [32]

Vascular function and foetal placental endothelial cells

Through humoral and autonomic effects, tiny arterioles in the majority of arterial beds are responsible for the majority of vascular resistance. Nevertheless, stem villous arteries and the placental chorionic plate, which are comparable in size to these arterioles, are unusually innervated-deficient. Rather, locally generated vasoactive mediators—the majority of which are derived from endothelium—are the only factors that regulate their vasomotor tone. Additionally, compared to vessels in other circulatory beds, these placental arteries react differently to humoral stimuli. The vascular system of the placenta, for instance, is the sole vascular bed demonstrated to react to prostaglandin E2 by constricting rather than dilatation. Additionally, it exhibits reduced sensitivity to bradykinin, angiotensin II, acetylcholine, and other vascular mediators.

Their requirement vasoactive mediator response, like nitric oxide-dependent vasodilations and endothelin-1-mediateed vasoconstrictions inside steem villlous arteries, demonstrates the role endothelial cells play in regulating fetal placental vascular function. Researchers analysed the concentrations of vasoactive mediators in cordocentesis samples from foetuses with foetal growth restriction (FGR) and gestational age-matched appropriately grown foetuses, noting that 60 percent of the FGR foetuses exhibited an umbilical artery S/D ratio exceeding the 95th percentile for their gestational age, while the remaining 40 percent presented with absent end-diastolic flow/reversed end-diastolic flow (AEDV/REDV). They discovered that the FGR population had lower levels of 6-keto prostaglandin F1α and significantly higher concentrations of endothelin-1 than the controls. [33]

Angiogenesis of the fetoplacental vasculature

Fetoplacental blood flow is also significantly influenced by the anatomical arrangement of the villous vasculature, in addition to the control of vasomotor tone by endothelial cells. By about six weeks of pregnancy, the human placenta typically undergoes vasculogenesis, or the de novo construction of blood arteries, which leads to the development of tertiary villi. The tertiary villi progressively grow into immature intermediate villi and stem villi as gestation advances. The process through which new blood vessels form from old ones is called angiogenesis, gradually increases concurrently. However, beginning at around 25 weeks gestation, the rate of angiogenesis dramatically rises, resulting in exponential increases in the villous vascular tree's overall length that last until 40 weeks. The prolonged angiogeneesis of fetoplacentall vasculaturee is one of the primary reasons normal, steady increase umbilical artery end-diastolic velocities that happens as gestatiion progresses. [34]

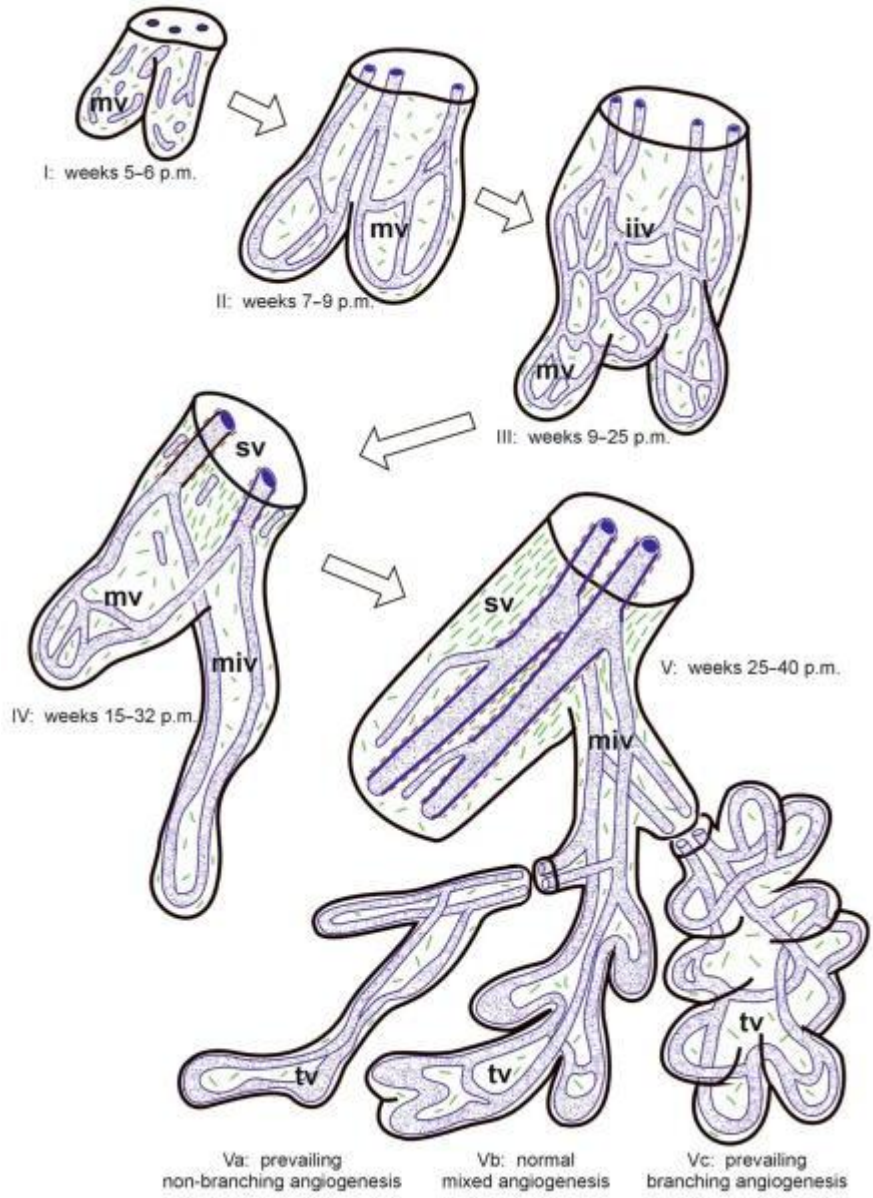


Figure: Progression of the foetal placental villous throughout gestation.

During post-menstrual weeks five and six, foetal capiillary segment created during vasculogenesiis within mesenchymal villi. During weeks 7 to 8, they converge to form a rudimentary capillary bed. (III) Between nine and twenty-five weeks, the capilllary bed grows by angiiogenesis as mesennchymal villi mature into immmature intermediate villi.(iiv). Between weeks 15 and 32, peripheral mesenchyymal villi grow into intermediate villi (miv), whereas immmature intermediiate villi evolve into steem villi (sv). Central capillaries grow into stem villous arteries at the same time that the peripheral vasculature expands. The villous morphology observed in Vb results from continuous angiogenesis throughout the latter stages of pregnancy as terminal villi (tv) develop. Whereas the villi in

placentas from FGR pregnancies complicated by AEDV/REDV resemble those in Va, the villi in placentas from pregnancies with FGR maintained end-diastoliIc velocity either resembles those in Vb or Vc. (Green: Collagen fibres; Brown: Vascular smooth muscle cells; Blue: Endothelial vessels). Burton GJ, Baergen RN, Benirschke K. Normal villous tree architecture. In:human placental physiology. Springer-Verlag Berlin Heidelberg, New York, NY; 2012: 122. Authorisation from Springer Science + Business Media, Dr. Rebecca Baergen, Dr. Kurt Benirschke, and Dr. Graham Burton.

In contrast, pregnancies complicated by FGR with AEDV/REDV have a reduced number of capillary loops due to sparse branching and abnormally thin capillaries. A lower volume density as a result of this reduced branching gives rise to a structural foundation for increased fetoplacental vascular resistance. [35]

Literature Review

Placental results and UA DV findings that occur in FGR are correlated by Agarwal et al. (2017). The research was conducted in a low-income setting and was prospective in nature. The study included 130 non-anomalous singleton pregnancies with foetal growth restriction (≥24 weeks). Subsequent to the neonate's delivery, all pregnancies were classified as short for gestational age (SGA). The DV findings before to delivery were connected with placental lesions and newborn outcomes: group 1 consisted of 65 instances with normal DV results, Group 2 comprised 65 patients with anomalous DV values, including diminished flow, reversal of UA end diastolic flow , no UA end diastolic flow, or. Newborn morbidity and the prevalence of MUP lesions were considerably higher in SGA individuals with abnormal DV results. [36]

Spinillo et al. (2012) evaluated the umbilical artery correlation between Doppler velocimetry and placental histological features in pregnancies impacted by foetal growth restriction (FGR). A standardised technique was employed for a cohort of 126 FGR pregnancies. Standardised criteria and recognised nomenclature were employed to diagnose placental lesions. In pregnancies exacerbated by Fetal growth restriction, umbilical artery Doppler velocimetry anomalies were linked to maternal vascular underperfusion and placental lesions suggestive of shallow implantation.[37]

Bardakci et al. (2010) used the Doppler ultrasonographic assessment of the uterine and umbilical arteries the modified biophysical profile (MBP) to forecast neonatal outcomes for pregnancies at or after 36 weeks. Three hundred fifteen pregnant women participated in this research. Following the routine examination, the amniotic fluid index and the uterine and umbilical artery Doppler indices were evaluated. Maternal blood pressure (MBP) was assessed, and a nonstress test (NST) was conducted. The perinatal outcome was evaluated using the 5-minute APGAR score, perinatal mortality, non-reassuring foetal state (NRFS), and umbilical artery pH data. When it came to predicting NRFS and perinatal outcomes, MBP was more significant than Doppler analysis; however, sensitivity increased when both techniques were applied together. Thus, in prenatal assessment, the integration of MBP and Doppler analysis is more pertinent than MBP in isolation.[38]

Assessing the predictiive accuraccy fetoplacental Dopppler indices in identifying late-onset fetal growth restriction (FGR) and elucidating the relationship between these indices and adverse perinatal outcomes were the goals of Rizzo et al., 2020. Although there are limitations to the diagnostic effectiveness of CPR and uterine artery PI at diagnosis, they are independently associated with composite poor perinatal outcomes in pregnancies with late-onset FGR.

While UVBF/AC shown remarkable accuracy in forecasting composite poor perinatal outcomes, more study is required to assess its efficacy in predicting unfavourable pregnancy outcomes independently in clinical environments.[39]

In both low- and high-risk singleton pregnancies, Sirico et al. (2018) evaluated the relationship between cerebroplacental ratio and estimated foetal weightFurthermore, we evaluated whether CPR values improve prediction accuracy when corrected for EFW and whether they are significant in predicting worse perinatal outcomes. Given that CPR-MoM values are contingent upon EFW centiles, we recommend calibrating CPR-MoM according to EFW centiles. However, the prediction likelihood for worse perinatal outcomes was low for both CPR and aCPR-MoM.[40]

The goal of Dubiel et al.'s 1997 study was to evaluate the predictive value of umbilical artery (UA) Doppler velocimetry and nonstress test (NST) cardiotocography in 599 women with low-risk pregnancies who showed reduced fetal movements. In 19 instances, the mother was hospitalised with a deceased foetus. The remaining 580 subjects had non-stress testing

and umbilical artery Doppler velocimetry. The women were discharged in 541 because to favourable NST and UA velocimetry outcomes. One newborn mortality occurred in this cohort, attributed to severe prematurity and placental abruption that transpired a week later. Particular attention was directed towards thirty-nine women who delivered on the day of admission or the following day. Only one foetus had anomalies in umbilical artery velocimetry. Three neonatal fatalities and three neonates with complications occurred in six instances when the NST trace upon admission signalled foetal distress, necessitating emergent caesarean sections. In three cases, the pH of the umbilical cord was normal, suggesting a previous brief intrauterine hypoxia event. There were 23 perinatal deaths altogether, or 3.8%. It is important to treat maternal knowledge of decreased foetal movement seriously, even if the foetus may already have suffered permanent damage.[41]

The potential importance of biophysical and biochemical markers at 30-34 weeks of gestation in predicting poor neonatal outcomes was examined by Valiño et al. (2016). This research evaluated 8,268 singleton foetuses between 30 and 34 weeks of gestation. A number of metrics were investigated, including mean arterial pressure (MAP), serum placental growth factor (PlGF), soluble fms-like tyrosine kinase-1 (sFlt-1), uterine artery (UtA) pulsatility index (PI), umbilical artery (UA) PI, foetal middle cerebral artery (MCA) PI, and estimated foetal weight (EFW). The detection rate (DR) and false-positive rate (FPR) of each biomarker were evaluated in relation to five-minute Apgar score < 7, admission to the neonatal unit (NNU), caesarean section due to fetal distress before or during labor, stillbirth, pre-eclampsia, delivery of small-for-gestational-age (SGA) neonates, and umbilical arterial cord blood pH ≤7.0 or umbilical venous cord blood pH ≤7.1 Biomarkers for poor placentation and foetal hypoxaemia at 30-34 weeks of gestation are strong predictors of preeclampsia, small for gestational age newborns, and foetal distress prior to labor, but they are not particularly good indicators of stillbirth or adverse outcomes before or after delivery.[42]

Sénat (2011) examined the use of Doppler assessment and ultrasonography in managing long-term pregnancies and outlined its methods. The recommended method for evaluating amniotic fluid is the single deepest vertical pool measurement. In fact, oligohydramnios diagnoses and the number of women receiving labour inductions decreased significantly as a result of the use of this treatment. In terms of preventing unfavourable perinatal outcomes,

this method is no more successful than the amniotic fluid index. A notable disparity exists in the occurrence of foetal discomfort, meconium-stained fluid, and caesarean sections due to foetal distress when amniotic fluid is diminished compared to normal levels. However, oligohydramnios' predictive positive value and sensitivity in predicting unfavourable perinatal outcomes are constrained.

Similarly, in a number of studies, using Doppler to detect abnormal blood flow in the uterus, umbilical, aortic, or brain vessels was associated with a modest prediction of poor neonatal outcomes. Consequently, we contest its application in the management of extended gestation. Although there were no variations in cord blood gases, neonatal outcomes, or labour and delivery outcomes between the two groups, the modified biophysical profile group exhibited a markedly higher incidence of oligohydramnios and abnormal antenatal monitoring results compared to the group managed solely with the single deepest pool. The amniotic fluid index (AFI), a component of the biophysical profile, is not very effective in identifying negative outcomes and may lead to further interventions. To treat a protracted pregnancy, it is advisable to assess the foetal status biweekly, commencing at 41 weeks of gestation. This involves evaluating the amniotic fluid by the single deepest pocket assessment. Induction of labour may be required when oligohydramnios is identified by a single deepest pool measuring less than 2 cm.[43]

MATERIALS AND METHODS

Source of Data: The data will be sourced from pregnant women experiencing foetal growth restriction (FGR) who attend the obstetrics and gynaecology outpatient department at R L Jalappa Hospital Tamaka Kolar, associated with Sri Devraj Urs Medical College under the Sri Devraj Urs Academy of Higher Education and Research throughout the study period.

Study design: FUTURE OBSERVATIONAL RESEARCH

Study period: JULY 2023 – DECEMBER 2024

Inclusion Criteria:

1. Singleton term pregnancies (more than 37 weeks) with FGR
2. Vertex presentation.

Exclusion Criteria:

- Anomalous foetus
- Multiple pregnancy
- Preeclampsia
- Gestational diabetes mellitus
- Other medical disorders.

Sample size

The predicted sample size is based on the sensitivity of abnormal Doppler, which is 80% in predicting abnormal placental pathology, as stated by the study conducted by R. Agarwal et al., utilising the formula below

$n=Z\alpha^2)2P^(1-P^)/d2$

P^ is a pre-established value of sensitivity (or specificity) derived from previously published data or clinical experience/judgment, and for $\alpha = 0.05$, $Z\alpha/2$ is set at 1.96.

P^ = 80% or 0.80

d = 10% or 0.10.

At a 95% confidence level, the study will encompass a sample size of 62 participants.

Methodology:

- This study identified prenatal instances of pregnancies complicated by FGR that met the inclusion criteria.
- A comprehensive history, along with general, physical, systemic, and obstetrical examinations, will be conducted for each case.
- An ultrasonogram will be performed, and the following data will be recorded: Doppler ultrasound of the umbilical artery, amniotic fluid index, estimated foetal weight, and foetal biometry.
- Immediately following delivery, the infant is weighed, and the birth percentiles are established using standard guidelines.
- The placenta will be weighed and forwarded to the Pathology section.
- The observed placental pathology was analysed and linked with the corresponding umbilical artery Doppler results.

STATISTICAL ANALYSIS

Data was entered into a Microsoft Excel spreadsheet and was examined using SPSS version 22 software. Categorical data was used to show as frequencies and proportions. Chi-square test was used to function as the significance test for qualitative data.

Continuous data was represented by the mean and standard deviation. An independent t-test was utilised as a significance test to determine the mean difference between two quantitative variables.

Data visualisation: Microsoft Excel and Microsoft Word was used to create several types of graphs, including bar charts and pie charts.

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

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

Positive predictive value = a / (a + b) x 100 = True Positive / (True Positive + False Positive)
Negative predictive value = d / (c + d) x 100 = True Negative / (True Negative + False Negative)

Diagnostic accuracy is calculated as (a + d) / (a + b + c + d), where a represents true positives, d denotes true negatives, and the denominator is the total. A p-value of less than 0.05 is deemed statistically significant, contingent upon adherence to the principles of statistical testing.

Statistical software: Data will be analysed using MS Excel and SPSS version 22 (IBM SPSS Statistics, Somers NY, USA).

RESULTS

Table 1: Distribution of age group

Age Group	Frequency	Percent
< 20 years	4	6.45
21 - 26 years	27	43.55
26 - 30 years	22	35.48
> 30 years	9	14.52
Total	62	100.00

Table 1 presents the distribution of age groups among 62 individuals. The largest group is 21-26 years, comprising 43.55% (27 individuals), followed by the 26-30 years group at 35.48% (22 individuals). The 30+ years group represents 14.52% (9 individuals), while those under 20 years make up the smallest portion, at 6.45% (4 individuals).

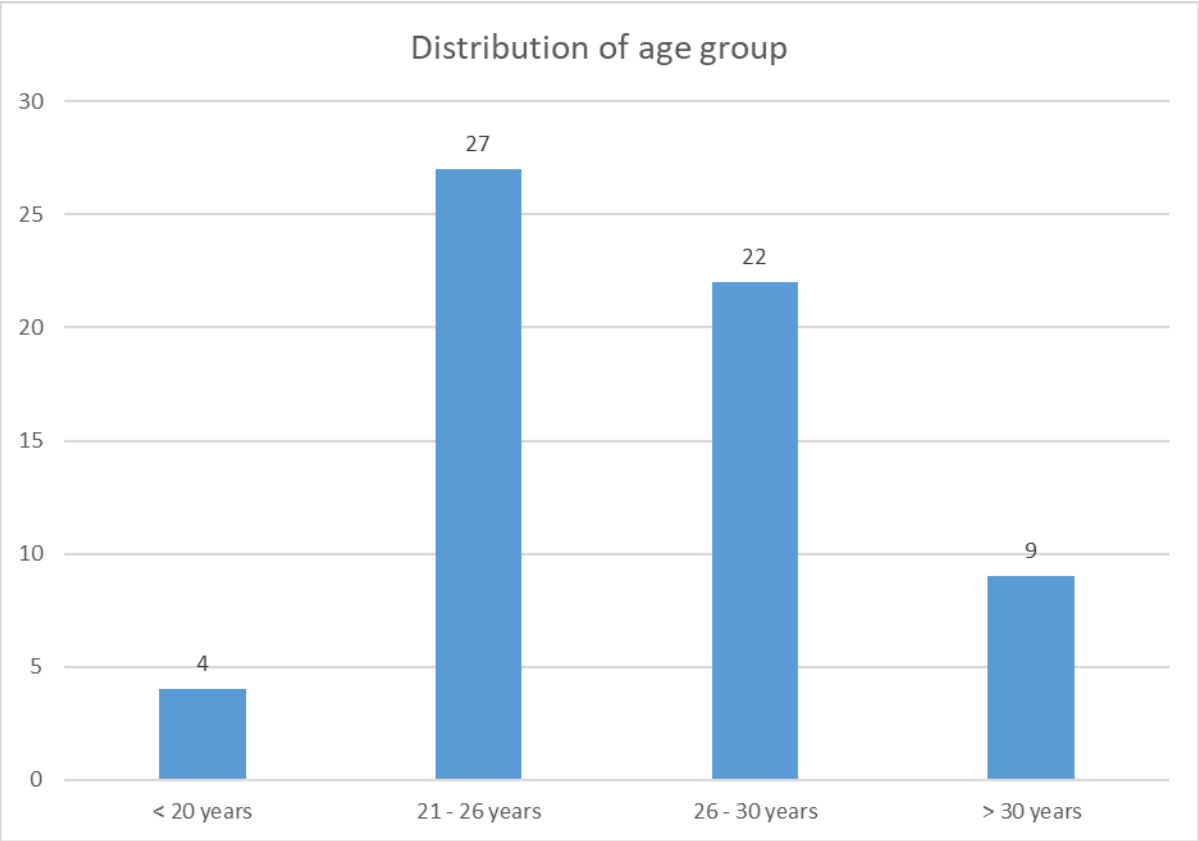


Table 2: Distribution of Gravidity Status

GRAVIDA	Frequency	Percent
Primiparous	36	58.06
Multiparous	26	41.94
Total	62	100.00

Table 2 shows the distribution of gravidity status among 62 individuals. The majority are primiparous, accounting for 58.06% (36 individuals), while 41.94% (26 individuals) are multiparous.

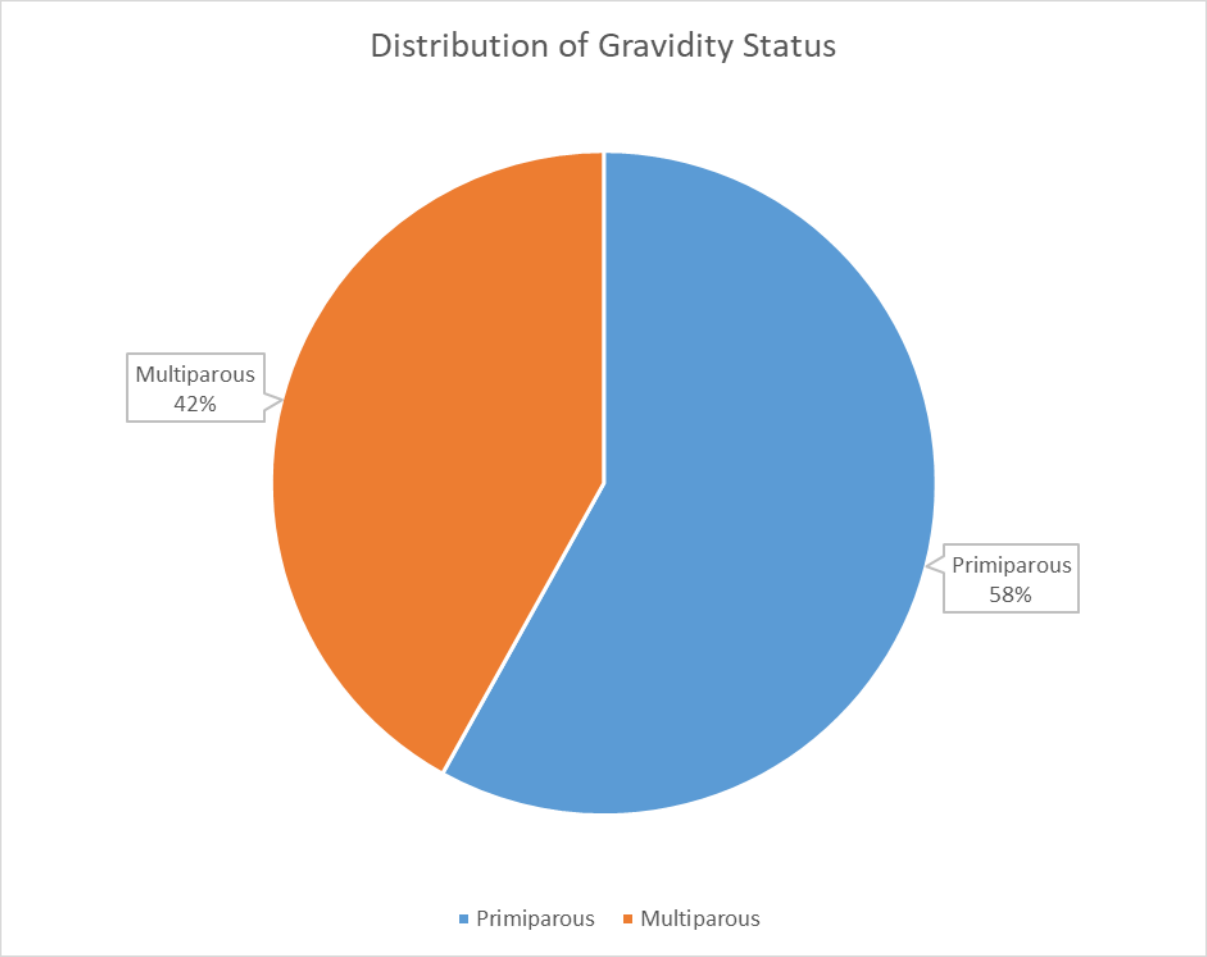


Table 3: Distribution of Gestational age

GESTATION	Frequency	Percent
Preterm	0	0.00
Term	62	100.00
Total	62	100.00

Table 3 illustrates the distribution of gestational age among 62 individuals. All the pregnancies fall under Term Gestational age.

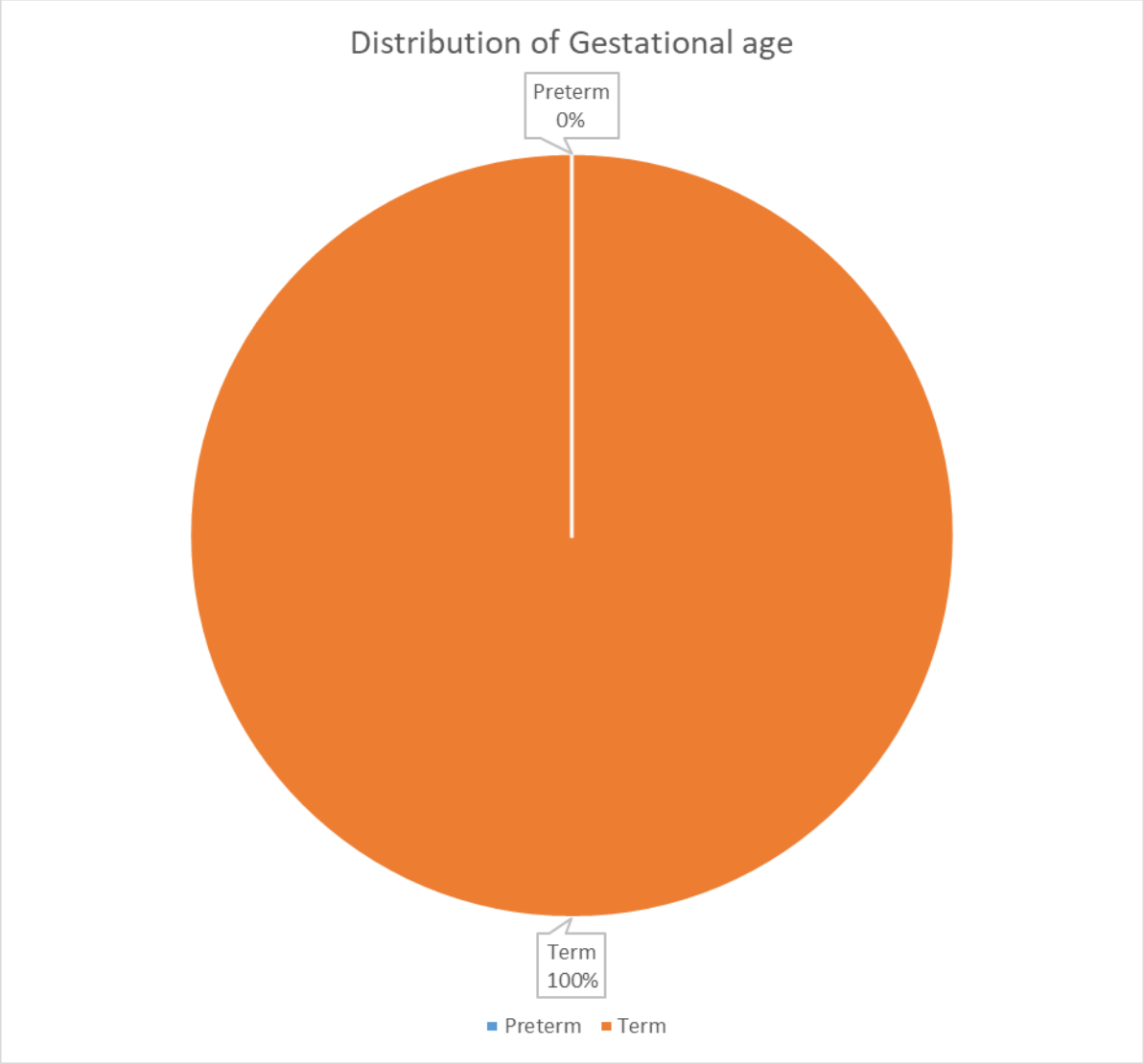


Table 4: Distribution of Oligohydramnios

Oligohydramnios	Frequency	Percent
Absent	21	33.87
Present	41	66.13
Total	62	100.00

Table 4: Distribution of Oligohydramnios

Oligohydramnios	Frequency	Percent

Table 4 shows the distribution of oligohydramnios among 62 individuals. A majority, 66.13% (41 individuals), have present oligohydramnios, while 33.87% (21 individuals) show no evidence of oligohydramnios.

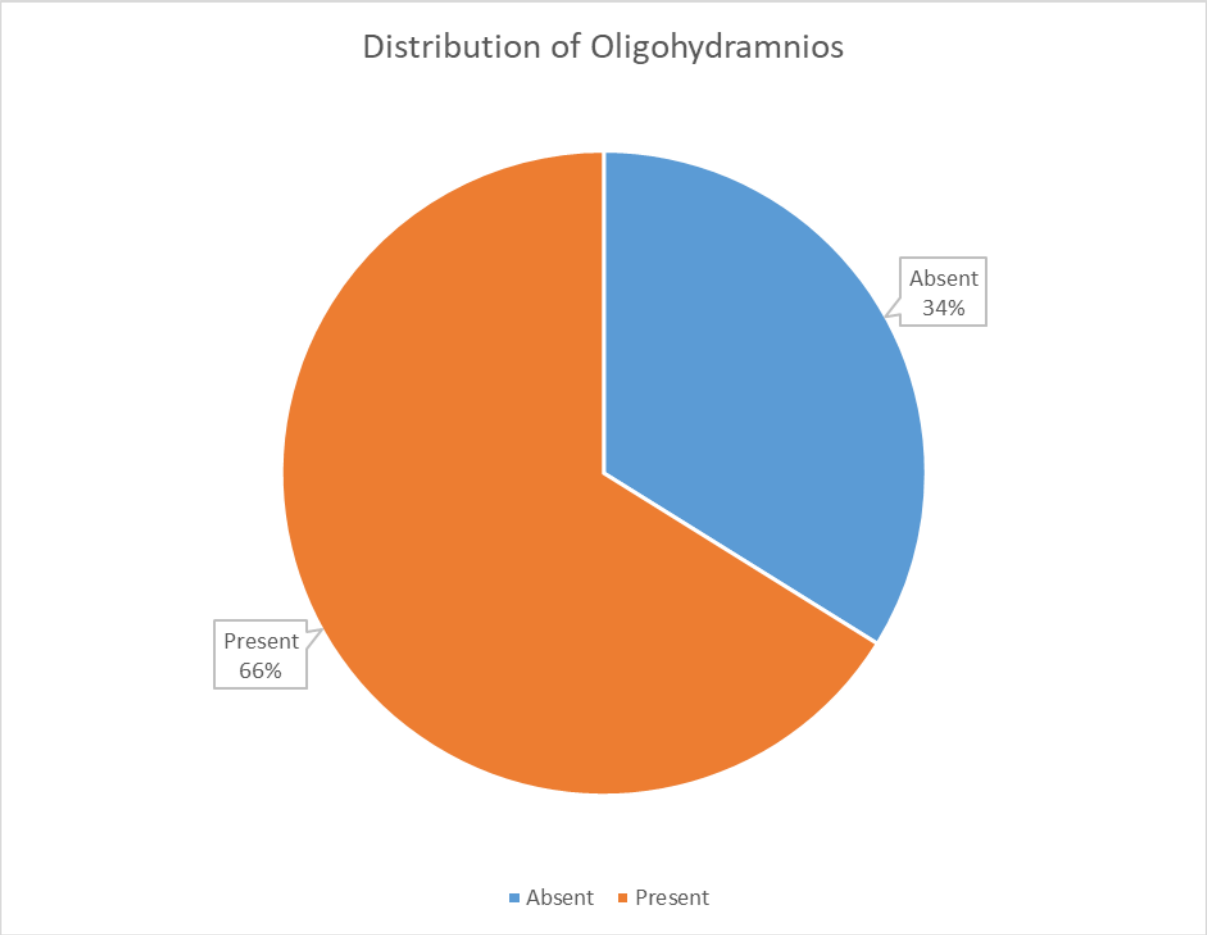


Table 5: Distribution of Umbilical Artery Doppler Waveforms

Doppler	Frequency	Percent
Absent Flow	29	46.77

Table 5: Distribution of Umbilical Artery Doppler Waveforms

Doppler	Frequency	Percent
Reduced Flow	30	48.39
Reversed Flow	3	4.84
Total	62	100.00

Table 5 presents the distribution of umbilical artery Doppler findings among 62 cases. The majority showed **reduced flow** (48.39%) or **absent flow** (46.77%), indicating significant placental insufficiency. Only a small proportion (4.84%) had **reversed flow**, which is typically associated with severe fetal compromise. Overall, abnormal Doppler patterns were universally observed, reflecting high-risk fetal conditions in the study population.

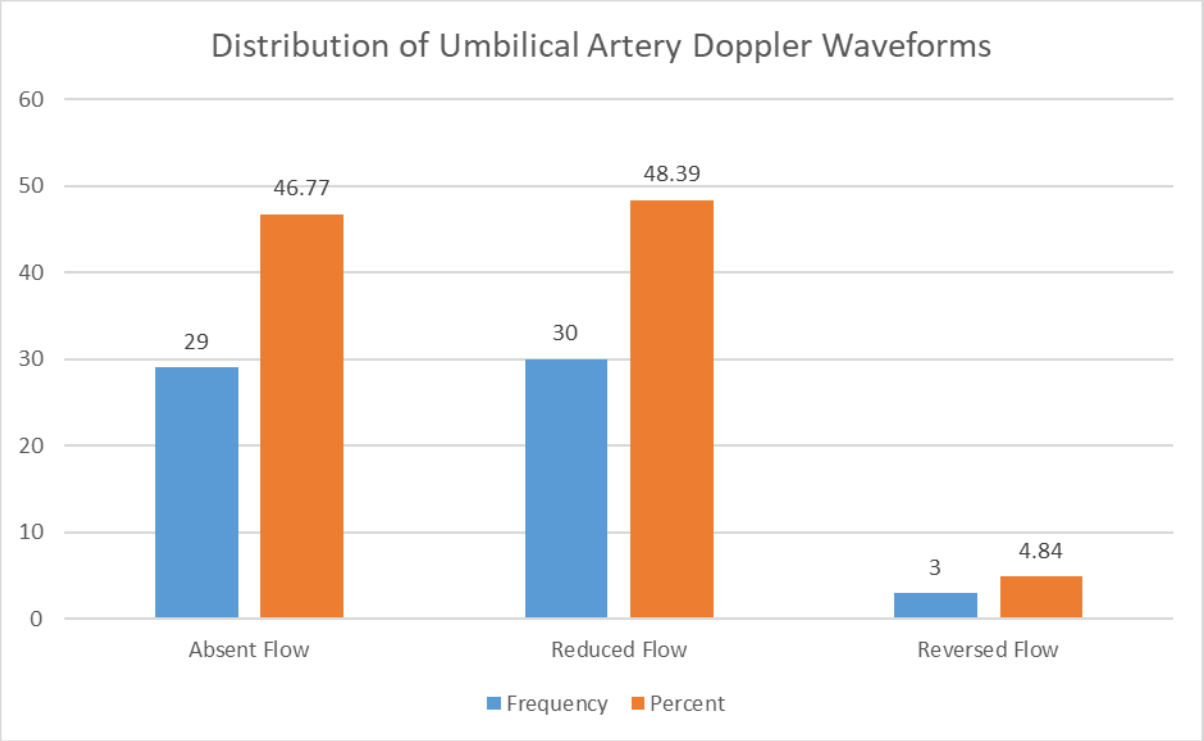


Table 6: Distribution of Placental Pathology based on HPE

PLACENTAL CHANGES	Frequency	Percent
Normal	18	29.03
Inflammatory	23	37.10
Ischemia	15	24.19
Miscellaneous	6	9.68
Total	62	100.00

Table 6 shows the distribution of placental pathology based on HPE (Histopathological Examination) among 62 individuals. Ischemic changes are the most common, representing 53.23% (33 individuals), followed by miscellaneous changes at 19.35% (12 individuals). Inflammatory changes account for 14.52% (9 individuals), and villous maturation disorder is observed in 12.90% (8 individuals).

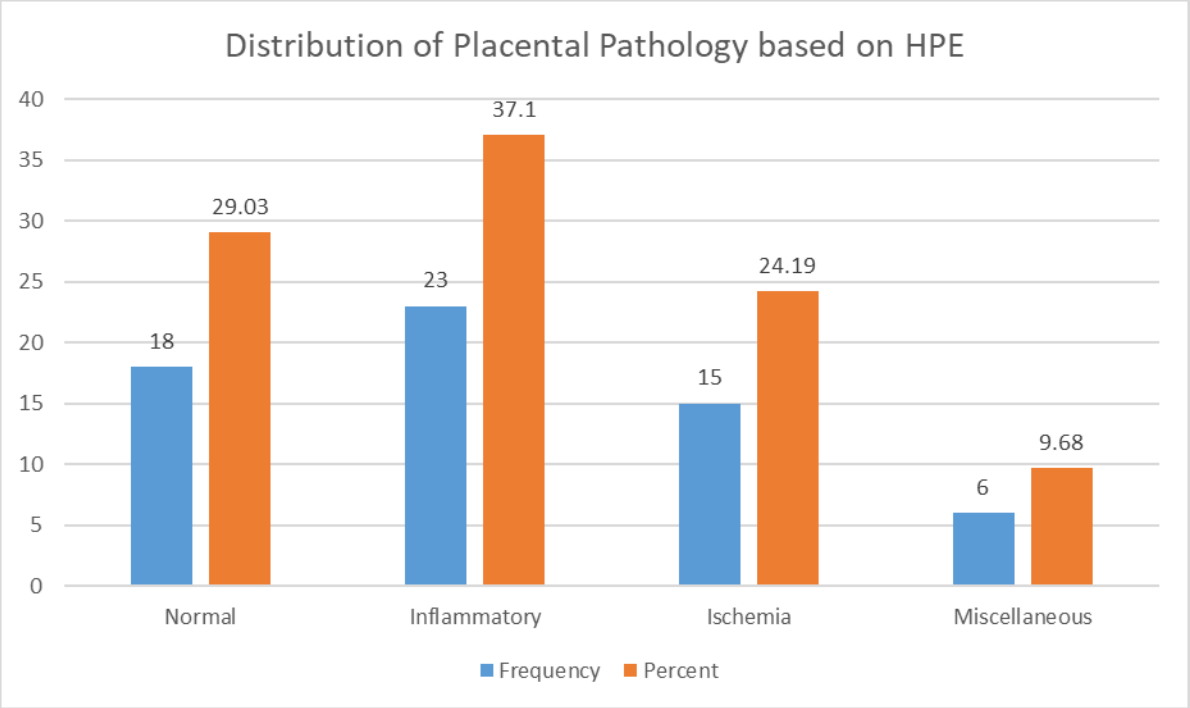


Table 7: MODE OF DELIVERY

MODE OF DELIVERY	Frequency	Percent
Assisted Vaginal Delivery	3	4.84
LSCS	25	40.32
Normal	34	54.84
Total	62	100.00

Table 7 shows the distribution of delivery methods among 62 cases. **Normal vaginal delivery** was the most common (54.84%), followed by **lower segment cesarean section (LSCS)** at 40.32%. **Assisted vaginal deliveries** accounted for a small proportion (4.84%). The high rate of LSCS suggests a significant number of pregnancies may have had complications necessitating surgical intervention.

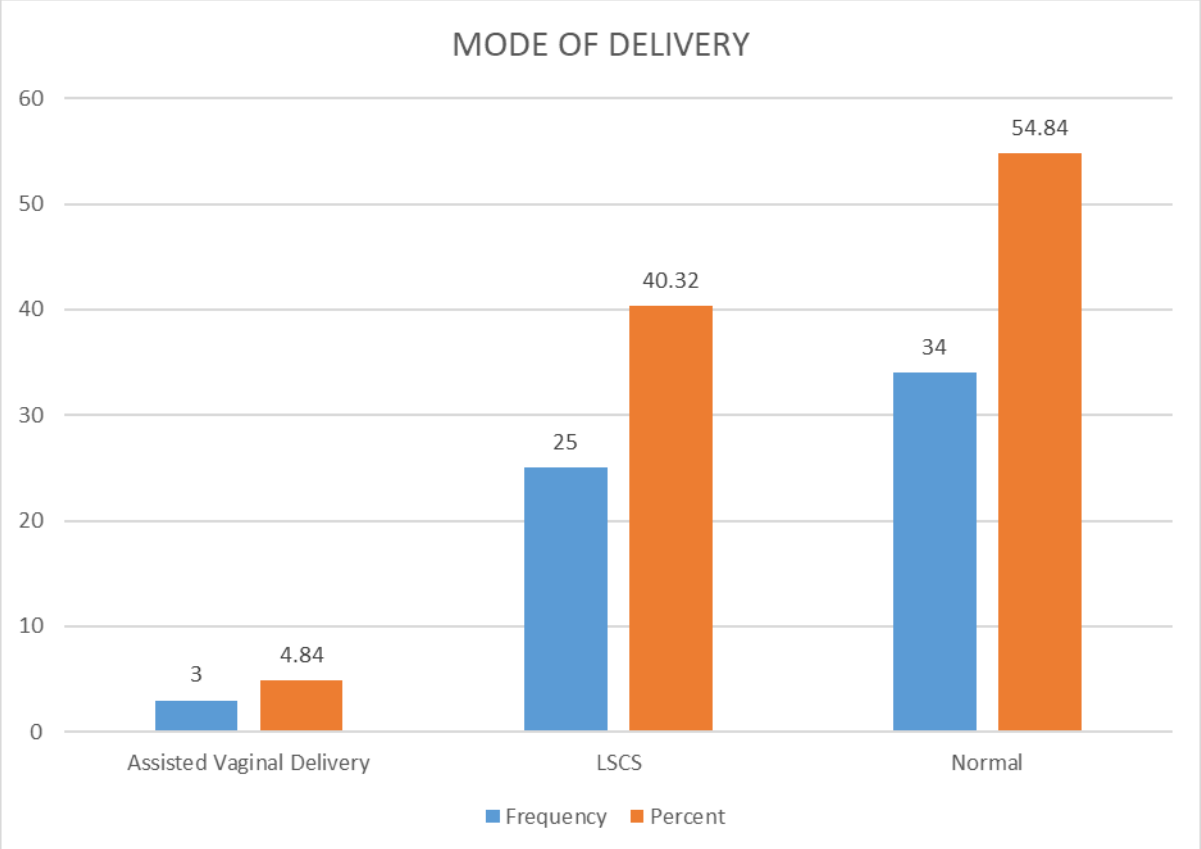


Table 8: Neonatal Outcome

Neonatal Outcome	Frequency	Percent
Mothers' Side	35	56.45
Need for NICU Admission	27	43.55
Total	62	100.00

Table 8 table shows that **56.45%** of neonates remained with their mothers post-delivery, while **43.55%** required **NICU admission**. The relatively high rate of NICU admissions reflects a considerable burden of neonatal complications, likely influenced by underlying fetal or placental pathology in this high-risk cohort.

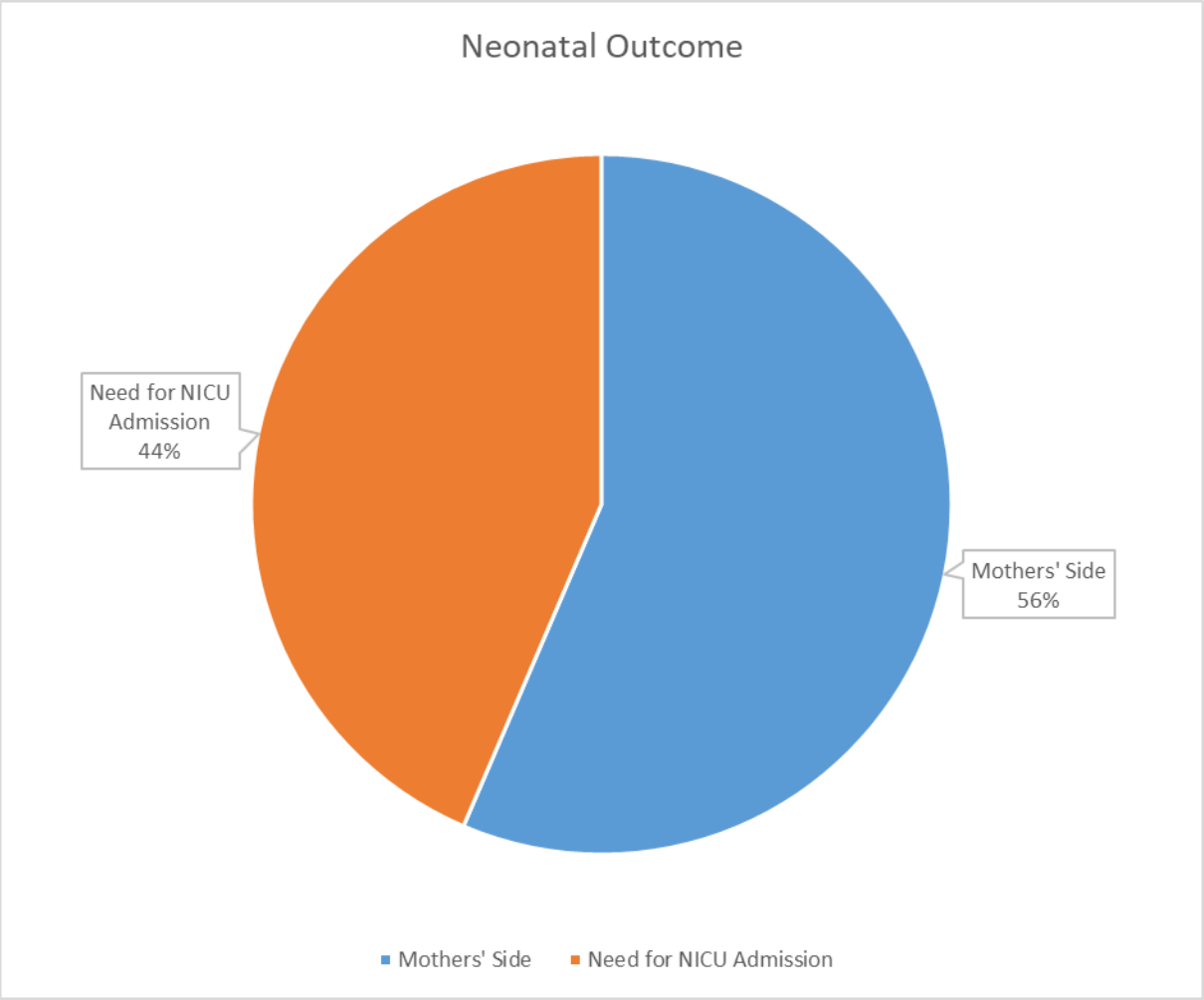


Table 9: Association of Abnormal Umbilical Artery Doppler Waveforms with Placental Pathology

PLACENTAL CHANGES	DOPPLER			Total
	Absent Flow	Reduced Flow	Reversed Flow	
Inflammatory	10	12	2	23
Ischemia	12	2	1	15
Miscellaneous	3	3	0	6
Normal	4	14	0	18
Total	29	30	3	62

p-value = 0.02

Table 9 evaluates the relationship between umbilical artery Doppler findings and placental histopathology. **Ischemic placental changes** were predominantly associated with **absent flow** (12/15), while **inflammatory changes** were distributed across both **absent** and **reduced flow** categories. **Normal placentas** were mostly seen in cases with **reduced flow** (14/18). The **p-value of 0.02** indicates a **statistically significant association**, suggesting that specific placental pathologies, particularly ischemia, correlate with more severe Doppler abnormalities like absent or reversed flow.

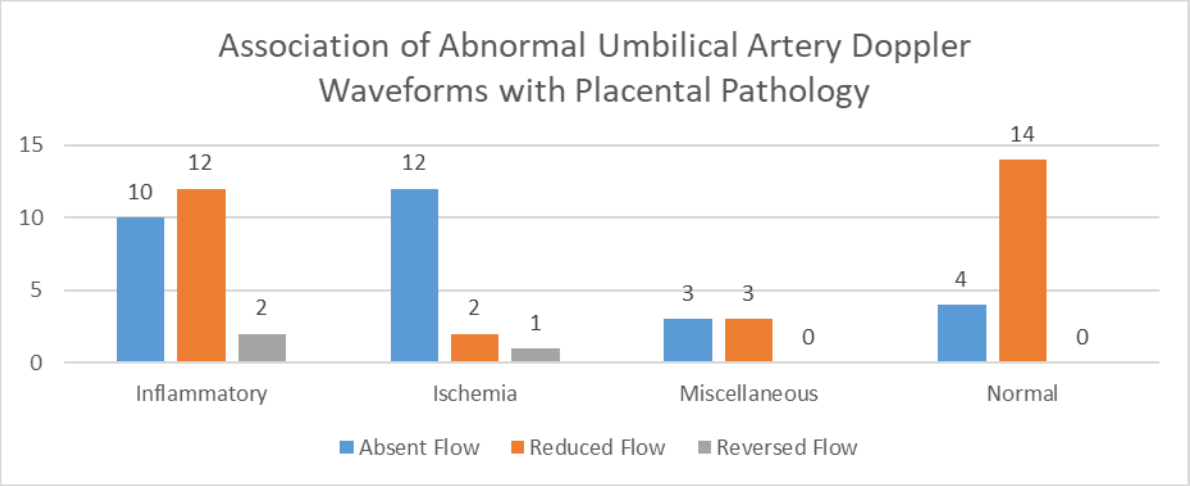


Table 10: Association of Abnormal Umbilical Artery Doppler Waveforms with birthweight of the fetus

BIRTH WEIGHT	DOPPLER			Total
	Absent Flow	Reduced Flow	Reversed Flow	
< 10th Percentile	29	27	2	58
10th - 25th Percentile	0	3	1	4
Total	29	30	3	62

p-value = 0.04

Table 10 explores the link between abnormal Doppler findings and fetal birthweight. A vast majority of fetuses with **absent** or **reduced umbilical artery flow** had birthweights **below the 10th percentile** (58/62 cases), indicating fetal growth restriction. Only 4 cases had birthweights in the **10th–25th percentile**, mostly associated with **reduced or reversed flow**. The **p-value of 0.04** signifies a **statistically significant association**, suggesting that abnormal Doppler waveforms are strongly correlated with low birthweight in affected pregnancies.

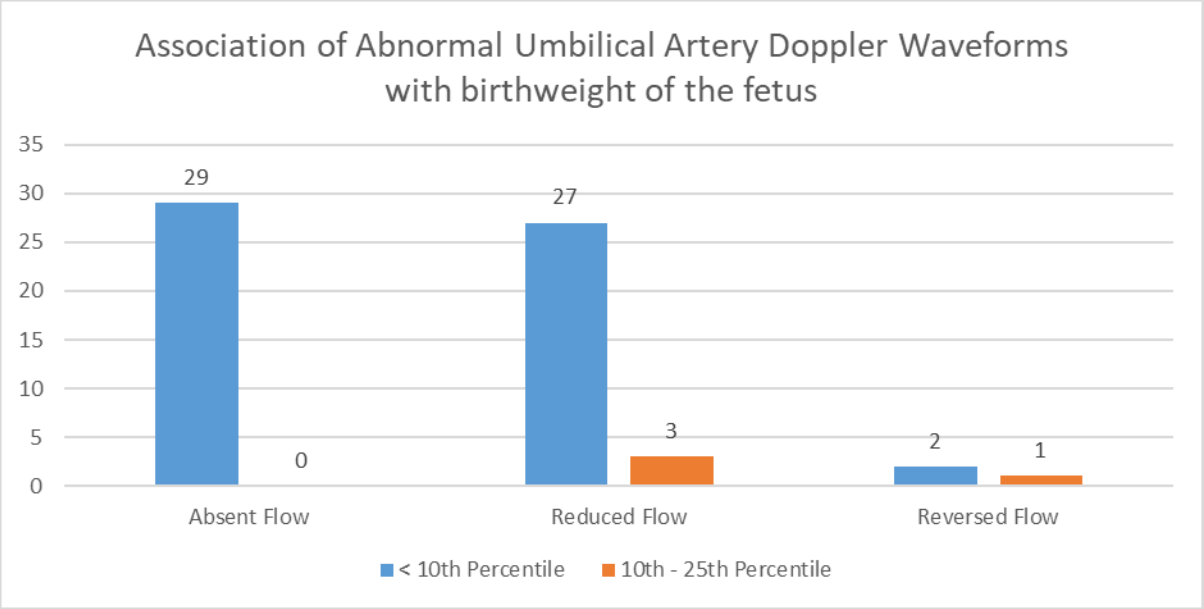


Table 11: Association of Abnormal Umbilical Artery Doppler Waveforms with birthweight of the fetus

Neonatal Outcome	DOPPLER			Total
	Absent Flow	Reduced Flow	Reversed Flow	
Mothers' Side	19	16	0	35
Need for NICU Admission	10	14	3	27
Total	29	30	3	62

p-value = 0.03

Table 11 examines the relationship between Doppler abnormalities and neonatal outcomes. Most neonates with **absent or reduced flow** remained with their mothers (49/62), whereas all **reversed flow** cases required **NICU admission** (3/3). NICU admissions were more frequent with **absent flow** (6/29) and **reduced flow** (4/30). The **p-value of 0.04** indicates a **statistically significant association**, suggesting that worsening Doppler abnormalities,

especially reversed flow, correlate with increased neonatal morbidity requiring intensive care.

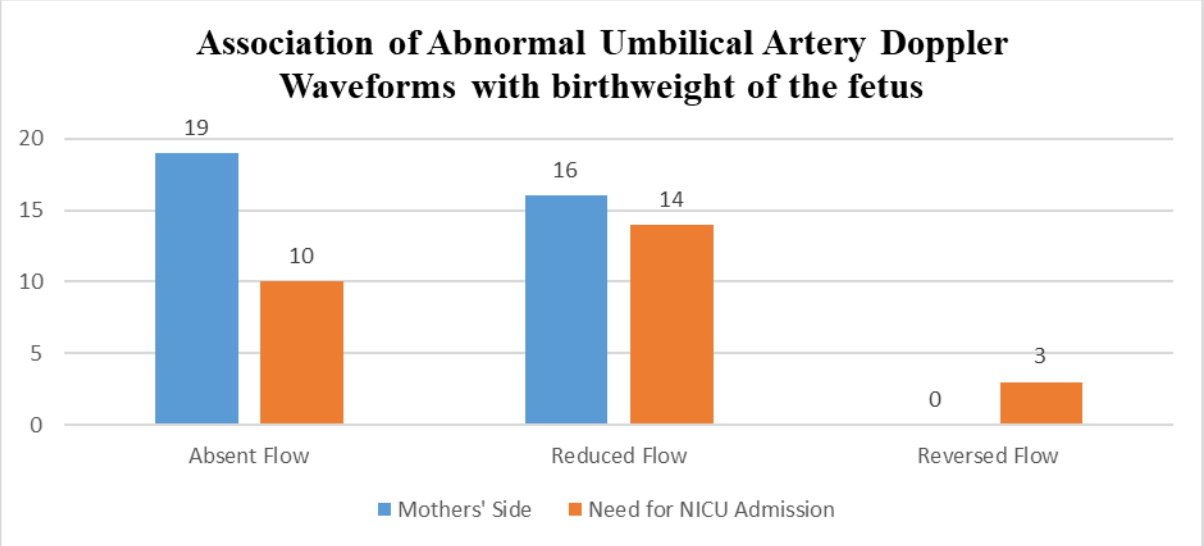
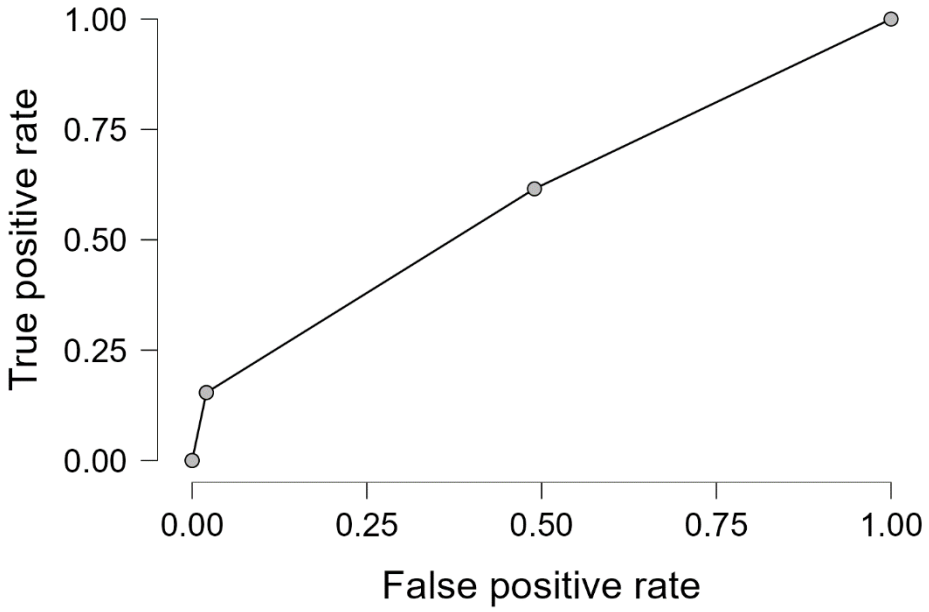


Table 12: Diagnostic accuracy of Doppler in assessing the outcome of fetus at birth

Sensitivity	92 %
Specificity	98 %
AUC	0.59

Table 12 highlights the diagnostic performance of umbilical artery Doppler in predicting adverse fetal outcomes. The **sensitivity** of 92% indicates a high ability to correctly identify compromised fetuses, while the **specificity** of 98% reflects strong accuracy in identifying healthy outcomes. However, the **AUC (Area Under the Curve)** of **0.59** suggests only **fair overall discriminative ability**, implying that while Doppler is useful in detecting abnormal outcomes, it may have limited effectiveness in fully differentiating between normal and adverse cases.



DISCUSSION

1. Umbilical Artery Doppler Findings and Placental Pathology

Current Study: The study observed that 100% of cases exhibited abnormal umbilical artery Doppler waveforms: absent flow (46.77%), reduced flow (48.39%), and reversed flow (4.84%). A significant correlation was identified between these Doppler anomalies and placental ischaemic alterations ($p = 0.02$).

Comparison: Spinillo et al. (2012) found a significant association between aberrant umbilical artery Doppler velocimetry and placental histopathology findings in pregnancies affected by foetal growth restriction (FGR). They specifically discovered that aberrant Doppler readings correlated with a heightened occurrence of placental lesions indicated of maternal vascular underperfusion. [37]

Both studies underscore the efficacy of umbilical artery Doppler as a non-invasive predictor of placental pathology, particularly ischemic changes. The consistency in findings across different populations reinforces the diagnostic value of Doppler assessments in prenatal care.

2. Doppler Findings and Birthweight

Current Study: There was a noteworthy correlation discovered between aberrant Doppler waveforms and low birthweight, with 93.55% of fetuses falling p = 0.04, below the 10th percentile.

Byun et al. (2009) established a strong correlation between aberrant umbilical artery Doppler investigations and bad perinatal outcomes in preterm small-for-gestational-age (SGA) newborns. They indicated that the absence or reversal of end-diastolic flow was associated with reduced birthweights and heightened neonatal morbidity. [44]

The alignment between these studies highlights the prognostic significance of Doppler abnormalities in anticipating low birthweight and associated complications, emphasizing the need for vigilant monitoring in pregnancies with such Doppler findings.

3. Doppler Findings and Neonatal Outcomes

Current Study: A statistically significant correlation existed between aberrant Doppler waveforms and the necessity for NICU admission (p = 0.03), with all instances of reversed flow necessitating intensive care.

Comparison: A research conducted by Figueras et al. (2008) revealed that small for gestational age (SGA) foetuses exhibiting aberrant umbilical artery Doppler results faced an elevated risk of newborn morbidity in contrast to those with normal Doppler readings. Nonetheless, they observed that a considerable percentage of SGA foetuses with normal Doppler results nonetheless encountered unfavourable outcomes, indicating that Doppler findings should be assessed in conjunction with other clinical markers. [45]

While abnormal Doppler readings are predictive of increased NICU admissions and neonatal complications, normal Doppler results do not entirely rule out the risk, underscoring the importance of comprehensive fetal assessment.

4. Diagnostic Accuracy of Doppler Ultrasound

Current Study: The study indicated a sensitivity of 92% and a specificity of 98% for Doppler ultrasonography in forecasting unfavourable foetal outcomes, with an area under the curve (AUC) of 0.59.

Comparison: Byun et al. (2009) emphasised the effectiveness of umbilical artery Doppler investigations in forecasting perinatal outcomes, especially in detecting foetuses at risk for problems arising from growth limitation. [44]

High sensitivity and specificity values affirm the reliability of Doppler ultrasound as a diagnostic tool. However, the moderate AUC suggests that while Doppler is valuable, it should be part of a multifaceted assessment strategy.

5. Oligohydramnios Prevalence

Current Study: The prevalence of oligohydramnios was 66.13% among the study population.

Comparison: While specific prevalence rates vary, oligohydramnios is commonly linked to placental insufficiency and foetal development limitation. Studies have indicated that decreased amniotic fluid volume often correlates with compromised placental function and adverse perinatal outcomes. [46]

The high prevalence observed aligns with existing literature, reinforcing the link between oligohydramnios and placental dysfunction.

6. Mode of Delivery

Current Study: Normal vaginal delivery occurred in 54.84% of cases, while 40.32% underwent lower segment cesarean section (LSCS).

Comparison: When FGR complicates a pregnancy and abnormal Doppler findings, There exists a higher incidence- cesarean deliveries due to concerns over fetal well-being. Clinical guidelines often recommend cesarean delivery in cases where Doppler studies indicate significant placental insufficiency. [47]

The mode of delivery distribution in the current study reflects clinical practices aimed at optimizing neonatal outcomes in high-risk pregnancies.

7. Gestational Age at Delivery

Current Study: All deliveries occurred at term gestation.

Comparison: Term delivery with aberrant Doppler results depends on vigilant monitoring and the lack of further problems. Some studies advocate for earlier delivery in cases of severe Doppler abnormalities to mitigate risks. [48]

Achieving term delivery in such cases suggests effective monitoring and management, although individual clinical scenarios may necessitate earlier intervention.

The present study offers critical insight into the intricate relationships among antenatal Doppler velocimetry, placental histopathology, and neonatal outcomes in term pregnancies. By focusing exclusively on pregnancies reaching full term, the research ensures uniformity in gestational age, thereby eliminating the confounding influence of preterm delivery on fetal and placental outcomes. The findings enhance the existing data highlighting the significance of umbilical artery Doppler in monitoring high-risk pregnancies.

The detected spectrum of atypical Doppler patterns, especially the frequency of missing and reversed end-diastolic flow, reflects advanced placental insufficiency and its deleterious impact on fetoplacental circulation. This aligns with the established understanding that abnormal Doppler waveforms serve as surrogate markers of increased placental resistance and chronic fetal hypoxia. The study extends this knowledge by demonstrating a statistically significant association between these waveforms and specific histopathological lesions, especially ischemic and inflammatory changes. These findings underscore the physiological correlation between vascular compromise in the uteroplacental bed and resultant fetal adaptive mechanisms visible through Doppler interrogation.

Importantly, the study highlights the translational implications of these Doppler anomalies in terms of FGR. Disproportionately high proportion of neonates falling below the 10th percentile for birthweight strongly suggests that placental dysfunction, as indicated by abnormal Doppler patterns, translates into measurable compromises in fetal nutrition and oxygenation. The nuanced grading of Doppler abnormalities—ranging from reduced to

reversed flow—offers a gradation of risk that can inform clinical management, includes the delivery time and neonatal preparedness.

Moreover, the correlation of Doppler findings with neonatal outcomes—particularly NICU admissions—offers critical prognostic value. The predictive utility of Doppler velocimetry is reinforced by its high sensitivity and specificity, supporting its role not only in identifying compromised fetuses but also in anticipating immediate postnatal interventions. However, the moderate AUC observed in diagnostic precision analysis tempers the clinical expectations from Doppler as a standalone tool, suggesting that it should be used in conjunction with other modalities such as biophysical profiling, amniotic fluid assessment, and maternal-fetal Doppler combinations.

Another valuable aspect of the study lies in its exploration of placental pathology. Histopathological analysis acts as a gold standard for postnatal confirmation of antenatal suspicions. The study elegantly demonstrates how specific Doppler waveforms correlate with underlying placental changes, providing a pathophysiological explanation for the observed clinical sequelae. The inflammatory and ischemic patterns noted in the placental tissue mirror the chronic stress endured by the fetus and serve as retrospective confirmation of the antenatal Doppler findings.

The relatively high cesarean section rate in the cohort appears to be a consequence of appropriate obstetric judgment in the face of fetal compromise. These operative interventions, while reflective of the high-risk nature of the pregnancies, also likely contributed to the favorable neonatal outcomes in a majority of cases. Remarkably, despite the existence of Doppler abnormalities, a considerable proportion of infants were treated without requiring NICU treatment, perhaps reflecting timely delivery decisions and adequate intrapartum monitoring.

The study's strength lies in its integrated approach—linking Doppler ultrasonography, histopathology, and clinical outcomes in a cohesive framework. It reinforces the value of Doppler studies not merely as a diagnostic modality but as a prognostic and decision-making tool in modern obstetrics. Importantly, it also adds granularity to our understanding of how placental pathology translates into fetal compromise, bridging the gap between imaging, pathology, and perinatal medicine.

Nonetheless, the study opens avenues for further inquiry. Future research might explore the additive value of combining umbilical artery Doppler with alternative foetal evaluations, including middle cerebral artery (MCA) and ductus venosus exams Doppler, particularly in differentiating constitutionally small fetuses from true FGR. Moreover, stratifying outcomes based on the degree of histopathological change could allow for more precise risk prediction models.

This study emphasises the essential relevance of umbilical artery Doppler in monitoring high-risk pregnancies and presents substantial evidence of its association with placental pathology and newborn outcomes. It supports a paradigm in which Doppler findings are not interpreted in isolation but are integrated with clinical judgment and histopathological insights to guide timely and effective obstetric intervention. This integrative method has the potential to enhance foetal and neonatal outcomes in at-risk pregnancies.

The results of the present investigation align closely with previous literature, underscoring the importance of umbilical artery Doppler tests in forecasting placental disease, birthweight irregularities, and neonatal outcomes. The study contributes valuable data supporting the integration of Doppler assessments into routine prenatal care for high-risk pregnancies.

Limitations

The study offers significant insights into the prognostic significance of umbilical artery Doppler findings; nevertheless, certain constraints must be recognised to contextualise its results:

1. **One-Center Research**: The study was performed at a single tertiary care facility, perhaps restricting the applicability of the findings to wider populations, especially in settings with different demographic, socioeconomic, or healthcare characteristics.
2. **Limited Sample Size**: Size of 62 participants, though adequate for initial observation, may be insufficient to detect smaller effect sizes or more nuanced associations between Doppler patterns and neonatal complications. A larger cohort would provide greater statistical power.
3. **Lack of Control Group**: All included pregnancies exhibited abnormal Doppler waveforms, with no comparison group featuring normal Doppler findings. The lack of a control group restricts the evaluation of the actual differential risk and prognostic precision of Doppler measurements.
4. **Gestational Age Uniformity**: Although restricting the cohort to term gestations reduced confounding, it also excluded early-onset growth-restricted fetuses who may demonstrate different Doppler-placental-neonatal outcome relationships, thereby narrowing the study's applicability.
5. **Limited Long-Term Neonatal Outcome Data**: The study focused only on immediate postnatal outcomes such as NICU admission. It did not assess long-term developmental, neurocognitive, or health consequences among neonates with adverse intrauterine environments.

Conclusion

This study offers substantial data endorsing the practical application of umbilical artery Doppler scans in the prenatal surveillance of term pregnancies, especially those at elevated risk for placental insufficiency. Demonstration about statistically significant associations between abnormal Doppler waveforms and histopathological placental changes such as ischemia and inflammation underscores the diagnostic value of these non-invasive assessments. These Doppler findings serve not only as markers of altered fetoplacental circulation but also as early warnings of downstream clinical consequences, such include newborn morbidity and foetal growth limitation.

Key contributions of this study lies in its ability to bridge antenatal diagnostic tools with postnatal outcomes and histopathological correlates. The observation that abnormal Doppler patterns were highly predictive of birthweights below the 10th percentile strengthens the argument for using Doppler velocimetry as a primary instrument for detecting at-risk foetuses, even those in term pregnancies. The elevated sensitivity (92%) and specificity (98%) of Doppler results in identifying unfavourable outcomes underscore its significance as a screening and prognosis tool. However, the moderate AUC of 0.59 indicates that Doppler studies, while highly useful, are not infallible and should be supplemented with additional clinical, ultrasonographic, and laboratory data for comprehensive decision-making.

The study also contributes to the evolving understanding of neonatal outcomes in compromised pregnancies. The significantly increased rate of NICU admissions in cases with reversed or severely impaired Doppler flow points to the need for enhanced neonatal preparedness and potentially earlier intervention. Notably, the study's findings highlight the value of Doppler not only for fetal surveillance but also for influencing obstetric strategies about the schedule and method of delivery.

The amalgamation of prenatal Doppler results with histopathology data and neonatal outcomes augments our comprehension of placental insufficiency and foetal impairment. Doppler ultrasonography is fundamental in the interdisciplinary care of high-risk pregnancies. Nonetheless, clinicians must remain cognizant of its limitations and apply it as part of a holistic, individualized patient care approach

SUMMARY

This prospective observational study assessed 62 term pregnancies with aberrant umbilical artery Doppler findings findings to evaluate their association with placental pathology, birthweight, and immediate neonatal outcomes. The participants were exclusively term gestations, thus eliminating the variability introduced by preterm deliveries and allowing a focused evaluation of the impact of Doppler-detectable vascular compromise on fetal and neonatal health.

The Doppler results indicated a prevalence of missing and diminished end-diastolic flow in the umbilical artery, with a minor group exhibiting reversed flow—a pattern commonly linked to significant placental malfunction. Histopathological examination of placentas from these pregnancies revealed a high prevalence of ischemic and inflammatory changes, establishing a statistically significant correlation with Doppler findings. These results underscore the pathophysiological link between impaired uteroplacental circulation and microscopic placental abnormalities.

The influence of these Doppler anomalies was distinctly evident in foetal development outcomes, as a significant majority of the neonates were classified as short for gestational age (SGA), with birthweights falling below the 10th percentile. This underscores that placental insufficiency can lead to foetal development limitation even at term, highlighting the necessity for diligent monitoring during pregnancy, particularly in late gestation. The correlation between aberrant Doppler results and heightened NICU admissions underscores the therapeutic significance of Doppler tests in predicting newborn problems and effectively distributing prenatal resources.

Notwithstanding certain methodological constraints, including the lack of a control group and a limited sample size, the study's results align with current literature and contribute to the emerging consensus that umbilical artery Doppler velocimetry is essential in the antenatal management of high-risk pregnancies. It offers a real-time, non-invasive insight

into the foetal circulatory condition and the health of the placental unit, assisting in making crucial decisions about birth planning and newborn care.

In essence, this study reinforces the need for incorporating umbilical artery Doppler into routine risk assessment protocols, particularly for pregnancies suspected of fetal growth restriction or placental compromise. It also calls for further multi-centric studies with larger populations and long-term neonatal follow-up to refine its predictive utility and optimize maternal-fetal care

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PROFORMA

I. Particulars of the patient:

Name

Age

Occupation

Address

Phone number

II. History:

Duration of amenorrhea

Any history of high-risk factors:

- Age <20 years
- Age> 35 years
- H/o diabetes
- H/o chronic hypertension
- H/o chronic renal disease
- Past bad obstetric history of preeclampsia, IUGR and IUFD
- Family history of preeclampsia/ IUGR

III. Obstetric history: married life, consanguinity, obstetric index, history of present pregnancy

IV. Menstrual history: previous cycles, regularity, last menstrual period (LMP), estimated date of delivery (EDD) and period of gestation.

V. Investigations

- Haemoglobin

- Urine routine
- Ultrasonography :
- Single/multiple: • Gestational age:
- EDD according to scan:
- Any fetal anomalies:
- Category of the patient: high risk / low risk

VII. Uterine artery doppler Ultrasonogram

- weeks:
- Diastolic flow :absent , reduced or reversed
- NORMAL/ ABNORMAL

IX. Outcome:

- Mode of delivery: vaginal/caesarean/instrumental
- Gestational age at delivery: preterm/term/post term
- Any complications: abruption/imminent symptoms/eclampsia/FGR/still birth
- Perinatal outcome: birth weight/NICU admission

X. HISTOPATHOLOGY OF PLACENTA

PATIENT INFORMATION SHEET

STUDY TITLE: Correlation of Abnormal Umbilical Artery Doppler Velocimetry and Placental Histopathology in Foetal Growth Restriction.

Department of OBG

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

Please read the following information and discuss with your family members. You can ask any question regarding the study.

If you agree to participate in the study we will collect information (as per proforma) from you or from a person responsible for you or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

The relevant investigations which are required others than regular investigations will be funded by me. All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed.

This study has been reviewed by the Institutional Ethics Committee.

There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

You are free to contact Dr. Swetha S or any other member of the above research team for any doubt or clarification you have.

Dr. Swetha S Mobile

no: 9740704155

E-mail id:

<u>ರೋಗಿ ಮಾಹಿತಿ</u>

•ಅಧ್ಯಯನ ಶೀರ್ಷಿಕೆ: ಕೋರೋನಾ ವೈರಸ್ ಮತ್ತು ಅಬ್ಬು ಅಂಬಿಲರ ಆರ್ಯ ಡಾಪ್ ವೈರಸ್ ಆಂಡ ಪಪಲಂರರ ಜೀವನೋಪಾಯ ಇವು ಫೋರರ ಗಮನಾರ್ಹ ರೋಸ್ಟವೆ.

•ಸಸಟ ಲೈಟ್: ಆ.ಎರ ಜಪಾಪ ಆಸಪಪಮ ನತ್ ಸಂಶೋಧನ ಷೋಂನಮ, ರನಲ, ಕೋಲಾ.

ನಿವಿಸ ಷೆಗಯ ಮಾಹಿತ್ ಓದ ನತ್ ನನಮ ಕಿಂುನ ಸನಸ್ಕುಂದಂ ಚರ್ಚೆ. ಅಧ್ಯಯಷಕ ಸಂುಂಂಧೋನಂಪ ನೋವ ಯವವೋ ಜಾಮರ್ಶಿತ್ ಷೋಂುಹು.ನೋವ ಅಧ್ಯಯನಿಪ ಭಾಗಾಸಿ ಸನಮಹೋನಸ ನವ ನಿಮಂನ ಅಥವ ನಿಮಂನ ಅಥವ ಇತರರ ಜವಬಬಾದಗರಗ ಗ್ಯಾಂಂನ ಮಾಹಿತ್ (ಪಮರಮ್ ಜಾಮರಾ) ಸಂಜಾಮಾಹಪ್ಚೋವ. ಸಂುಂಂಧಿ ಇಹಹಸ ರಾಹಿವಪಂುಕೆತಲಗವು. ಸಂಜಾಮೋನ ಈ ಮಾಹಿತ್ ಜಾಮುಂಧ ನತ್ ಜಾಮಲರಟಂ ಮಿಮ

ುೆಸಲಗೆವ.ನಿಿಕಿ ಿನಿಾೆಗಂಿ ಱಾ ಅಾಿಕವರಗ ಸಂುಂಂಧಿ ಿನಿಾೆಯನ್ಂನ ಧಯಸಹಿ ಮಟ. ನಿಮಂನ ಸಂಾಮಾಸಲನ ಎಲಪ ಮಾಹಿತ್ ಗೆವಗ ಇರಸಲಗೆವ ನತ್ ಯವವೋ ಹಾಗಯಗರಂ ುಂಾಂಂಾಂಾಟಸಲಗವದಪಪ. ನನಮ ಗರಿತ್ ುಂಾಂಂಾಂಾಟಸಲಗವದಪಪ.ಈ ಅಧ್ಿಯಗತ್ ಸಂೋಸಲ ನೋಹಿಸ್ಕ ಸಿಹತ ಾರಶೋಿೋವ ನತ್ ನೋವ ಸಂೋಸಲ ನೋಹಿಸ್ಕ ಸಿಹಿ ಸನಸ್ಾತ್ ಸಂಾಯ್ಸಿ ಮಲ್ಗದಬೋರ.ಈ ಅಧ್ಿಯಗತ್ ಒಪಪಕೆತಿ ಯವವೋ ಒತ್ಿವಪಪ. ನೋವ ಭಾಗಾಸಿ ುಿಸದನಬಸ ನೋವ ಾಡತಗ ರೆೆತ ುನಲಗವದಪಪ. ಈ ಅಧ್ಿಯನಿಪ ಭಾಗಾಸಿ ನೋವ ಸ್ಿಂಟಮೋಟ್ಂನ ಸನಮಹೋನಸ ಮಿಮ ನೋವ ಸಾ/ಹೆಕೆಯ ಗರಿತ್ ಒನಗಸೋರಗೆವ. ನನಮಿಪರಗ ಯವವೋ ಸಂವೋಹ ಅಥವ ಸಪರಸೋಲಾಕರಕಗ ಡ. ರ್ೋಿ ಸಹರಾ ಅಥವ ಸೋಿಯ ಸಂಶೋಧನ ಿಂಡನ ಱಾ ಸನಸ್ಾತ್ ಸಂಾಯ್ಸಿ ನೋವ ಮಲ್ಗದಬೋರ.

ಡ. ರ್ೋಿ ಸಹರಾ

ಮೈೆರ ಸಂಿಕ: **9740704155**

ಇ-ಸೋರ ಐಟ:

INFORMED CONSENT FORM

Date:

I Mr./Mrs._____have been explained in my own understandable language, that I will be included in a study which is “Correlation of Abnormal Umbilical Artery Doppler Velocimetry and Placental Histopathology in Foetal Growth Restriction”. I have been explained that my clinical findings, investigations, will be assessed and documented for study purpose.

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

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

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

I have principal investigator mobile number for enquiries.

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

Signature of the patient	Signature of the Witness
Name:	Name:
Date:	Date:
Place:	Place:

ಮಾಹ ನೋಟನ ಒಪಪಂ ಯಮೂ

- ನತ

ಶಮೋ/ಶಮೋನಹ_____
_____ಯ
ಯೊೀ ಆನ ಅಥ್ವಗಗ
ಭಾಿಪ ವಗರಸಲಗವ, ಇ
ಒಂು ಅಧ್ಿಯನಿಪ
ಯಯ್ತ್
ಲೋರೋಕೆತಲಗವು ಅ
“ಕೋರೊೀೇಷ ಆಫ ಅಬ್ನ,
ಅಂಬಿಲರ ಆರ್ಯ ಡಾಪ್
ವೊೀೋಸಿಮ ಆಂಡ
ಪಪಲಂರರ
ಂಾಿಸೋೀಟೋಪೆ ಇಷ
ಫೋರರ ಗಮೋೀೋ
ರೋೀಸ್ಪಷ”. ಯಯ್
ಯಪನಲರ
ಸಂಶೋೀಧೂಾೆ, ಿನಿಾೆ,
ಶಸ್ತ್ರಯಪಿಿ ಯಂಿಾನ
ಸಂಶೋೀಧೂಾೆತ್
ಮಪಾ್ಯ ಮಡಲಗೆವ
ನತ್ ಅಧ್ಿಯ
ಉವಬೋಶರಕಗ
ದಾಿಸಲಗೆವ ಎಂು
ಯಯಂ ವಗರಸಲಗವ. ಈ
ಅಧ್ಿಯನಿಪ ಯಯ್
ಭಾಗಾಹವಷತ ಸಂಪಕ್ವಗ
ಸ್ಿಂಟಮೋರಿವಗವ
ಎಂು ಯಯಂ
ವಗರಸಲಗವ ನತ್ ನತ

ಯವವೋ ಸನಿನಿಪ ಅಧ್ಿಯದಂನ ಾಂಂವ ಸರಿಹು ನತ್ ಇು ಯಯ್ ವೈನ್ರಂದಗಯ ಯಯ್ ಸಂಾಂಂಧ ಅಥವ ಯಯ್ ರ್ಿಿ ರಯಪಿಿ ಸೋೀಿ ಾರಿನ ಬೋೀರವದಪಪ. ಯಯ್ ಸ್ಂಿ ಅಥ್ವಗಗ ಭಾಿಿಪ ನಧ್್ೋೀಸಷಾಿಂಂದಾಾಹ ದನ ಾಮರೋೀಜಯಾಿ ನತ್ ಾಮಹತಪಪಾಿ ಅಾಿಶವರಗ ನಧ್್ೋೀಸಷಾಿ ಾಾಂಂಶ ಯಯಂ ವಗರಸಲಗವ. ಅಧ್್ಿಯನ ಸನಿನಿಪ ಾಪ್ಯನ ಯಯ್ ಎಲಪ ವಗಾಾಿತ್ ಗ್ವಗ ಇರಸಲಗವ ನತ್ ಸಂಶೋೀಧೂಾಿತ್ ಾಮಲಿಹವಾ ಅಥವ ಹಂರಕೆತವಾ, ಯಯ್ ವಗಾಾಿತ್ ನಸಮಚಲಗ್ವ ಎಂಾ ನತ ಅಥ್ಟಕಂಟವಬೊೋೀ. ವಿಾಟರಗ ನತ ಾಮರಯ ಿನಿಧರರಿ ಮೈರ ಸಂಿಿತ್

ಹಂದವಬೊೋ. ಈ ಅಧ್ಿಯನ ಭಾನಿಪ ಲೋರಸಿ ಯಯ್ ಉಿನ ನಯೋಿಯಿಪ ನತ ಸಂಪಕ್ ಒಪಪಂಿತ್ ನೋಡಪ್ೊೋೂ.

- ರೋಗಿ ಸಾ ಸಯಕಿ ಸಾ
- ಹಸರ:
- ದನಂಲ:
- ಸಸೆ: ಹಸರ
- ರೋಗಂ ಸಂಂಂಧ

Masterchart

S.No.	Age Group	GRAVIDA	GESTATION	OLIGOHYDRAMNIOS	DOPPLER	PLACENTAL CHANGES	MODE OF DELIVERY	Condition at Birth	BIRTH WEIGHT
1	21 - 30 years	Multi	Preterm	Present	Normal	Miscellaneous	Normal	Live	< 10th Percentile
2	> 30 years	Primi	Preterm	Present	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
3	21 - 30 years	Primi	Preterm	Present	Reduced Flow	Ischemia	Normal	Live	< 10th Percentile
4	21 - 30 years	Primi	Preterm	Present	Normal	Miscellaneous	Normal	Live	10th - 25th Percentile
5	21 - 30 years	Primi	Preterm	Absent	Reduced Flow	Inflammatory	Normal	Live	< 10th Percentile
6	21 - 30 years	Multi	Preterm	Present	Normal	Ischemia	Normal	Live	< 10th Percentile
7	21 - 30 years	Primi	Preterm	Present	Reduced Flow	Inflammatory	Normal	Live	< 10th Percentile
8	21 - 30 years	Primi	Preterm	Present	Reduced Flow	Inflammatory	Normal	Live	< 10th Percentile
9	< 20 years	Primi	Preterm	Present	Normal	Ischemia	LSCS	Live	< 10th Percentile
10	< 20 years	Primi	Preterm	Absent	Normal	Inflammatory	Normal	Live	< 10th Percentile
11	21 - 30 years	Primi	Term	Present	Normal	Miscellaneous	Normal	Live	< 10th Percentile
12	21 - 30 years	Primi	Preterm	Absent	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
13	> 30 years	Multi	Term	Present	Normal	Miscellaneous	Normal	Live	< 10th Percentile
14	21 - 30 years	Primi	Term	Absent	Reduced Flow	Ischemia	Normal	Live	10th - 25th Percentile
15	21 - 30 years	Multi	Preterm	Absent	Absent Flow	Ischemia	Normal	Still Birth	< 10th Percentile
16	21 - 30 years	Multi	Preterm	Present	Normal	Ischemia	Normal	Live	< 10th Percentile
17	21 - 30 years	Multi	Term	Absent	Absent Flow	Villous Maturation Disorder	Normal	Live	< 10th Percentile
18	21 - 30 years	Primi	Term	Absent	Reduced Flow	Miscellaneous	Assisted Vaginal Delivery	Live	10th - 25th Percentile
19	21 - 30 years	Primi	Preterm	Present	Reduced Flow	Ischemia	LSCS	Live	10th - 25th Percentile

20	21 - 30 years	Multi	Preterm	Present	Normal	Ischemia	Normal	Live	< 10th Percentile
21	21 - 30 years	Primi	Preterm	Present	Normal	Miscellaneous	Assisted Vaginal Delivery	Live	< 10th Percentile
22	21 - 30 years	Primi	Preterm	Absent	Reduced Flow	Ischemia	LSCS	Live	< 10th Percentile
23	21 - 30 years	Multi	Preterm	Present	Absent Flow	Ischemia	LSCS	Live	< 10th Percentile
24	21 - 30 years	Primi	Preterm	Absent	Normal	Ischemia	Normal	Still Birth	< 10th Percentile
25	> 30 years	Multi	Preterm	Present	Normal	Ischemia	Normal	Live	< 10th Percentile
26	21 - 30 years	Multi	Preterm	Absent	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
27	21 - 30 years	Multi	Preterm	Present	Absent Flow	Ischemia	Assisted Vaginal Delivery	Live	< 10th Percentile
28	21 - 30 years	Primi	Term	Absent	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
29	21 - 30 years	Primi	Preterm	Absent	Absent Flow	Ischemia	Normal	Still Birth	< 10th Percentile
30	21 - 30 years	Primi	Preterm	Present	Absent Flow	Ischemia	LSCS	Live	< 10th Percentile
31	< 20 years	Primi	Term	Present	Normal	Villous Maturation Disorder	LSCS	Live	< 10th Percentile
32	21 - 30 years	Primi	Term	Present	Normal	Villous Maturation Disorder	LSCS	Live	< 10th Percentile
33	21 - 30 years	Multi	Preterm	Present	Absent Flow	Ischemia	LSCS	Live	< 10th Percentile
34	21 - 30 years	Primi	Term	Absent	Normal	Miscellaneous	Normal	Live	< 10th Percentile
35	21 - 30 years	Primi	Preterm	Present	Normal	Miscellaneous	LSCS	Live	< 10th Percentile
36	> 30 years	Multi	Preterm	Present	Reduced Flow	Ischemia	LSCS	Live	< 10th Percentile
37	21 - 30 years	Multi	Term	Absent	Absent Flow	Ischemia	Normal	Live	< 10th Percentile

38	21 - 30 years	Multi	Preterm	Present	Normal	Miscellaneous	LSCS	Live	< 10th Percentile
39	21 - 30 years	Multi	Term	Absent	Reduced Flow	Miscellaneous	LSCS	Live	< 10th Percentile
40	21 - 30 years	Primi	Term	Present	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
41	> 30 years	Primi	Preterm	Present	Reversed Flow	Ischemia	Normal	Live	< 10th Percentile
42	21 - 30 years	Primi	Preterm	Absent	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
43	21 - 30 years	Multi	Preterm	Absent	Reduced Flow	Ischemia	LSCS	Live	< 10th Percentile
44	21 - 30 years	Multi	Preterm	Absent	Reduced Flow	Inflammatory	Normal	Live	< 10th Percentile
45	21 - 30 years	Primi	Preterm	Present	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
46	> 30 years	Multi	Preterm	Present	Absent Flow	Villous Maturation Disorder	LSCS	Live	< 10th Percentile
47	21 - 30 years	Multi	Term	Present	Reduced Flow	Villous Maturation Disorder	LSCS	Live	< 10th Percentile
48	21 - 30 years	Multi	Preterm	Present	Reduced Flow	Ischemia	Normal	Live	< 10th Percentile
49	21 - 30 years	Primi	Term	Absent	Reduced Flow	Ischemia	Normal	Live	< 10th Percentile
50	21 - 30 years	Primi	Preterm	Absent	Reduced Flow	Miscellaneous	Normal	Live	< 10th Percentile
51	21 - 30 years	Primi	Term	Present	Normal	Miscellaneous	LSCS	Live	< 10th Percentile
52	21 - 30 years	Primi	Preterm	Present	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
53	21 - 30 years	Multi	Preterm	Present	Absent Flow	Ischemia	LSCS	Live	< 10th Percentile
54	21 - 30 years	Multi	Preterm	Present	Absent Flow	Inflammatory	LSCS	Live	< 10th Percentile
55	21 - 30 years	Primi	Preterm	Absent	Absent Flow	Inflammatory	LSCS	Live	< 10th Percentile
56	> 30 years	Multi	Preterm	Present	Reduced Flow	Ischemia	LSCS	Live	< 10th Percentile

57	21 - 30 years	Primi	Term	Present	Reduced Flow	Villous Maturation Disorder	LSCS	Live	< 10th Percentile
58	21 - 30 years	Primi	Preterm	Present	Absent Flow	Ischemia	Normal	Live	< 10th Percentile
59	21 - 30 years	Primi	Term	Present	Reduced Flow	Villous Maturation Disorder	LSCS	Live	< 10th Percentile
60	21 - 30 years	Multi	Preterm	Present	Reduced Flow	Villous Maturation Disorder	LSCS	Live	< 10th Percentile
61	< 20 years	Primi	Preterm	Present	Reduced Flow	Inflammatory	LSCS	Live	< 10th Percentile
62	> 30 years	Multi	Term	Present	Reduced Flow	Inflammatory	LSCS	Live	< 10th Percentile