COMPARATIVE STUDY OF PLACENTAL ABNORMALITIES IN NORMAL AND SPECIFIC HIGH RISK PREGNANCIES BY USING DOPPLER, HISTOLOGY AND THEIR FETAL OUTCOME

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CERTIFICATE

This is to certify that the dissertation titled **COMPARATIVE STUDY OF PLACENTAL ABNORMALITIES IN NORMAL AND SPECIFIC HIGH RISK PREGNANCIES BY USING DOPPLER,HISTOLOGY AND THEIR FETAL OUTCOME** is the bonafide work done by **Dr.P.VASANTHI** between May 2009 to October 2010 during her M.D O.G Course at INSTITUTE OF SOCIAL OBSTETRICS AND GOVT KASTURBA GANDHI HOSPITAL, MADRAS MEDICAL COLLEGE , Chennai.

DEAN

MADRAS MEDICAL COLLEGE

DEAN
Madras Medical College
Chennai-600 003

13/12/2010

DIRECTOR

ISO-GOVT.KGH

Director of Social Obstetrics
Institute of Social Obstetrics and
Govt Kasturba Gandhi Hospital
For women and children,
Triplicane, Chennai-600 005

DECLARATION

I **Dr.P.Vasanthi** solemnly declare that the dissertation titled **'COMPARATIVE STUDY OF PLACENTAL ABNORMALITIES IN NORMAL AND SPECIFIC HIGH RISK PREGNANCIES BY USING DOPPLER, HISTOLOGY AND THEIR FETAL OUTCOME'** has been prepared by me. This is submitted to the Tamilnadu Dr. MGR Medical University, Chennai in partial fulfillment of the rules and regulations for MD Examination in Obstetrics and Gynaecology. This has not been previously submitted by me for the award of any degree or diploma from any university.

Place : Chennai

Date : 13.12.2010

P.Vasanthi

(P. VASANTHI)

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CONTENTS

INTRODUCTION

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- Placenta is a vital lifeline between mother and fetus through which nutrients, oxygen, antibodies and hormones pass.
- Improper placentation and placental function could be potentially danger to the health of the mother and the fetus.
- High risk pregnancies are a small segment of the obstetric population, that produces the majority of maternal and infant mortality and morbidity
- Upto 40% of high risk mothers experience placental damage.

Placenta is one of the most remarkable organ in the sense that it has a very short life and yet very critical for continuation of the pregnancy and thus life of the baby in utero. For nine months it is effectively the lung, the gut and the kidney of the fetus.

The term ‘Placenta’ is believed to have been introduced in 1559 by **RAELDUS COLUMBUS** who used this Latin work which actually means ‘Circular lake’.

In (1937), **NOSEMAN** defined Placenta as that portion of the fetal membranes that was in apposition with (or) fused to the uterine mucosa for the transfer of oxygen and metabolites between the maternal and fetal blood.

As the Placenta grows and ages, histological changes suggest an increase in the efficiency of transport to meet the metabolic requirements of the growing fetus. A variety of changes in the morphology of the placental villi in normal and abnormal pregnancies have been reported.

The pathological changes in the Placenta are not by large specific to a particular disorder and therefore a variety of disorders may show similar changes. Final picture is often very complicated and no particular complication of pregnancy produces specific morphological changes within the placenta, which allow one to make a specific morphological diagnosis. **FOX(1964) AND WIGGLESWORTH(1964)** have attributed all changes to diminished uteroplacental blood flow.

In this study, an attempt has been made to correlate various placental villous changes to the fetal outcome in normal and specific high risk pregnancies.

BACKGROUND FOR THE STUDY

BACKGROUND FOR THE STUDY

The scientific interest in the Placenta derives not only from its enormous diversity of form and function, but also from the varied histopathological changes in different disease entities. Much effort is being put into understanding the Placental changes and their effects on the fetus. Placenta is the primary site of pathology responsible for many common forms of fetal risk in particular the group of disorders which can be broadly described as **'intra uterine deprivation'** and which is manifest amongst other features of fetal growth retardation.

The college of American Pathologists (1991) recommends routine pathological examination of the Placenta with certain obstetrical and neonatal conditions. Conversely the American College of Obstetricians and Gynaecologists (1991) feels that there are insufficient data to support these recommendations.

BENIRSCHKE (1991) observed that physicians generally are uncomfortable with the task of examining the Placenta yet it is a task they should willingly undertake. Submitting this organ to a reasonably knowledgeable look and touch can provide much insight into prenatal life. "…apart from this, available data suggests that examination of Placenta may be very useful in medico legal cases" – **CYNTHIA, G. KAPLAN** (1996). Perinatologists have long appreciated the importance of placental findings in the understanding of perinatal outcome.

INDICATIONS FOR PATHOLOGICAL EXAMINATION OF PLACENTA

CAROLYN M SALAFIA (1992) describes 18 maternal factors, 11 delivery complications and 4 neonatal conditions as indications and criteria for HPE of placenta following delivery. They are given below:

Maternal Indications	**Delivery complications**	**Neonatal Indications**
1. Auto Immune diseases	1. Preterm delivery	IUGR
2. Cardiac lesions	2. Post datism	Low APGAR
3. Blood disorders	3. Placenta praevia	Congenital anomalies
4. Diabetes	4. Abruptio placenta	Newborn ICU admission
5. Hypertension	5. Intra uterine/neonatal death	
6. DES exposure	6. Passage of meconium	
7. Thyroid disorders	7. Membrane rupture > 12 hrs	
8. Gynaecological abnormality	8. Fetal heart rate abnormality	
9. Viral syndrome	9. Chorioamnionitis	
10. STD	10. Retained placenta	
11. Previous demise (or) still birth	11. Poor biophysical profile	
12. 2 (or) more abortions		
13. Infectious diseases		
14. Infertility		
15. Drugs use/abuse		
16. Abnormal ante partum lab studies		
17. Preterm labour		
18. Hydramnios		

PLACENTA

PLACENTA

The placenta is developed from two sources. The principal component is **fetal which develops from chorion frondosum, maternal** component consists of decidua basalis. **Development of the placenta according to RANDL S. KUHLMANN (1996)** consists of **previllous or lacunar stage and villous stage.**

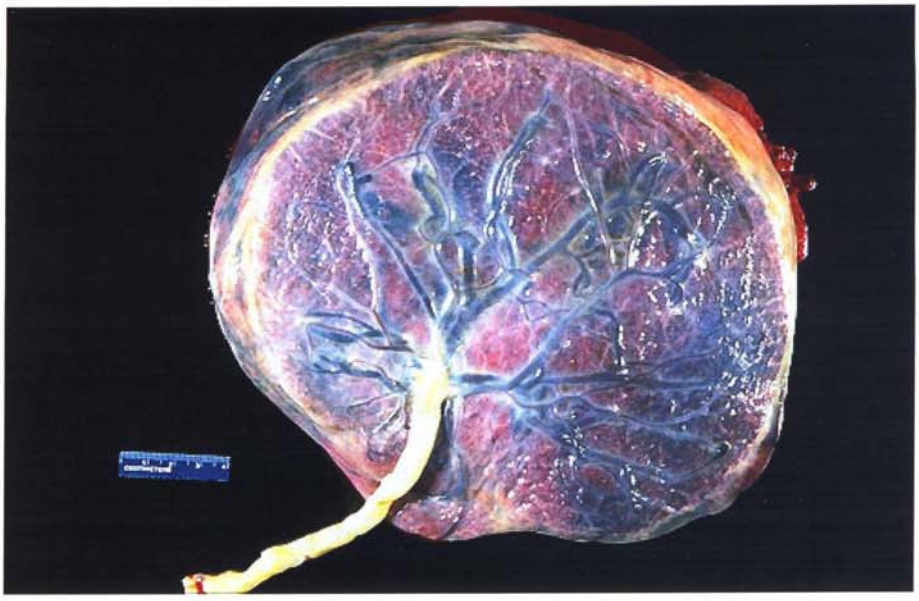
DEVELOPMENT OF PLACENTA – RANDALL S. KUHLMANN

During the first 2 weeks of Placental development (7-12 days after fertilization) invasion of the endometrium occurs. Complete endometrial invasion is believed to be completed by 12-14 days following fertilization. At this time the inner cytotrophoblast and outer syncytiotrophoblast is formed. This stage is referred to as the **previllous** or **lacunar stage.**

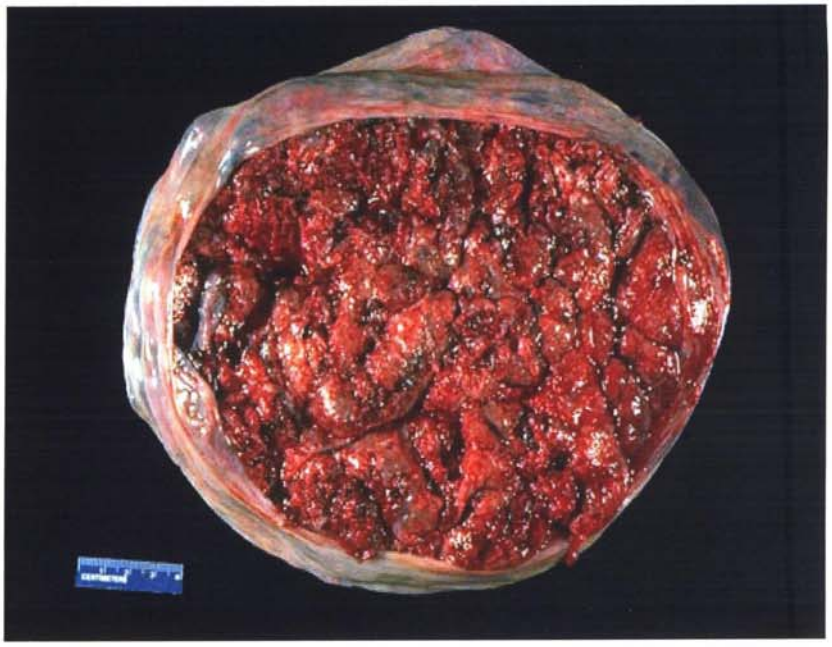
The third week after fertilization is referred to as villous stage. At this time, the primary villi develop mesodermal cores. Through inductive process, these **primary villi** become **secondary villi.**

Mesoderm undergoes further differentiation giving rise to the arteriocapillary venous chain, hence forming the **“tertiary villi”**. This vascular network is established by 20 days after fertilization.

PLACENTA FETAL SURFACE



PLACENTA MATERNAL SURFACE



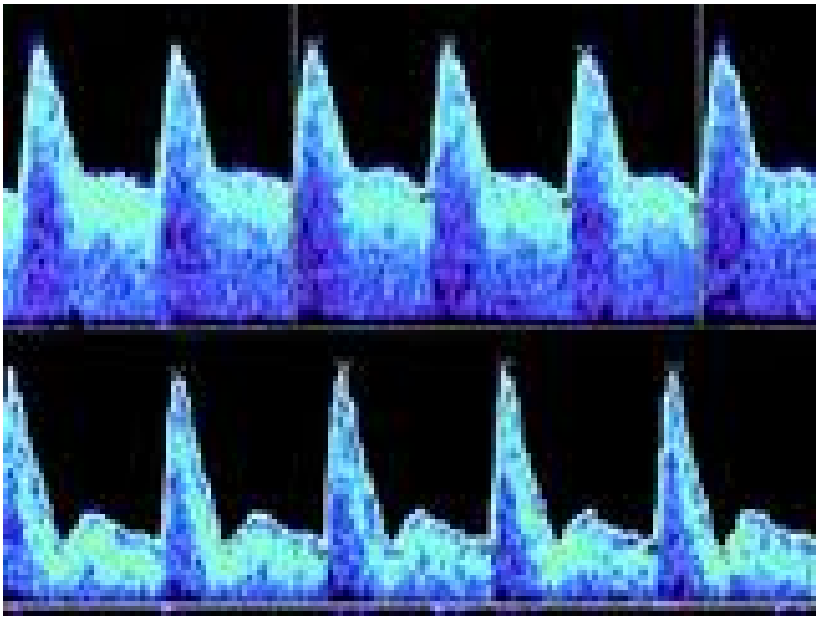
Further development of this network includes cytotrophoblastic invasion of the syncytio trophoblastic layers resulting in the formation of the stem or anchoring villi and the branching villi.

The anchoring villi are specialized stem villi that are connected to the basal plate. The branching villi are ultimately responsible for maternal-embryo exchange. Continuous differentiation results in the formation of the villous fetal circulation.

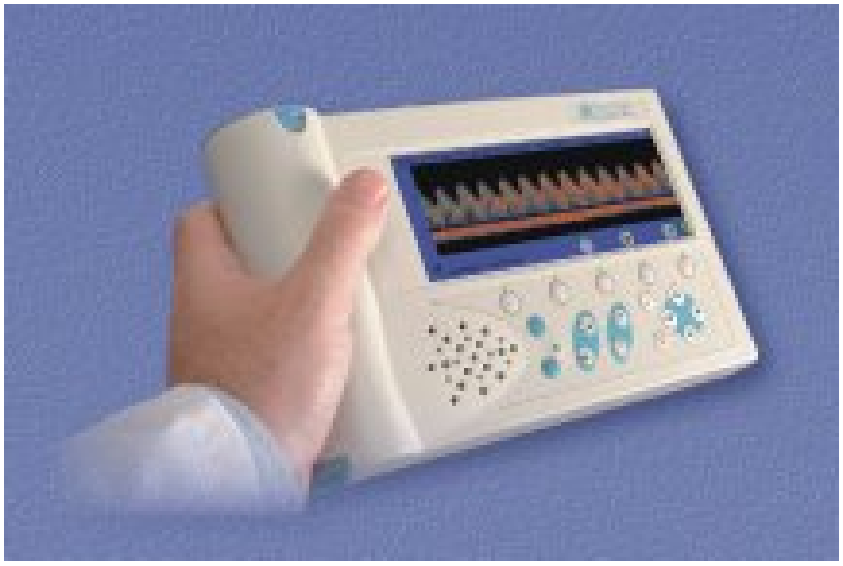
Normal development of the uteroplacental arteries depends on two types of extratrophoblastic invasion. Invasion of the decidua and myometrium by the stromal trophoblast forms the placental bed giant cells. The second type invasion occurs when the endovascular trophoblast migrates into the spiral arteries which eventually transforms them into uteroplacental arteries. The endovascular trophoblastic invasion of the spiral arteries is thought to occur in two waves.

The **'first wave'** is believed to be completed by the 10^{th} week of gestation. The **'second wave'** begins at about 14-16 weeks and is generally completed in 4 weeks. Incomplete development of these two waves is believed to have certain clinical implications. It is postulated that the risk of fetal growth restriction, preeclampsia and general pregnancy failure are all increased when these developmental waves are incomplete.

NORMAL AND ABNORMAL DOPPLER VELOCIMETRY



COLOUR SPECTRAL DOPPLER



DOPPLER ULTRASONOGRAM:

Doppler ultrasonogram is a new non invasive technique that we have used in this study to determine the qualitative aspects of uteroplacental circulation. A variety of Doppler ultrasound modes are used in the diagnostic instruments. These are

1) Continuous wave Doppler (CWD)
2) Pulsed wave Doppler (PWD)
3) Duplex scanner
4) Two Dimensional Doppler Colour Flow Mapping (DCFM)

Two Dimensional Doppler Colour Flow Mapping (DCFM)

DCFM produces a colour coded map of Doppler frequency shift superimposed on B mode ultrasound image. Flow towards the transducer is coded in red and flow away in blue. Mosaic patterns of red orange (or) blue green represents flow in several directions suggesting turbulence. It has also been used in studying flow dynamics in fetal heart.

DOPPLER INDICES FOR ARTERIAL FLOW VELOCITY WAVEFORM

S/D RATIO = Systolic Peak Velocity/End Diastolic Velocity

PULSATILITY INDEX = $\frac{\text{Systolic Peak Velocity – End Diastolic Velocity}}{\text{Mean Frequency Shift}}$

$$\text{RESISTANCE INDEX} = \frac{\text{Systolic Peak Velocity} - \text{End Diastolic Velocity}}{\text{Systolic Peak Velocity}}$$

Uterine Artery Flow Velocity waveforms:

Lack of endovascular invasion by trophoblasts into the myometrial portion of the placental bed spiral arteries is a consistent finding in preecclampsia.

Early Diastolic Notch:

In non-pregnant state, uterine artery waveforms exhibits high pulsatility with a rapid rise and fall in frequency shifts during systole and an early diastolic notch. Pregnancy results in marked changes in uterine artery waveform from 26^{th} week onwards.

Normally S/D ratio value does not change throughout the remainder of the pregnancy. Diastolic notch disappears by 20-26 weeks. So the full evolution of uterine artery waveform is complete only after 26 weeks. So abnormal uterine artery waveforms are those with

1) S/D Ratio > 2.8 (the average of right and left uterine arteries)
2) Persistence of early diastolic notch.

The mean averaged S/D ratio for each trimester were I trimester – 5.5,

II trimester – 2.9 , III trimester – 2.1.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Placental study by CAROLYN – M.SALAFFIA

JOSEPH F . YETTER III (1998)

A. GENERAL FEATURES

1. Placental completeness

It is of critical, immediate importance in the delivery room. Retained tissue is associated with PPH and infection. Large vessels beyond edges indicate the possibility that an entire placental lobe (eg. Succenturiate or accessory lobe) may have been retained in placenta accreta, placenta increta and placenta percreta.

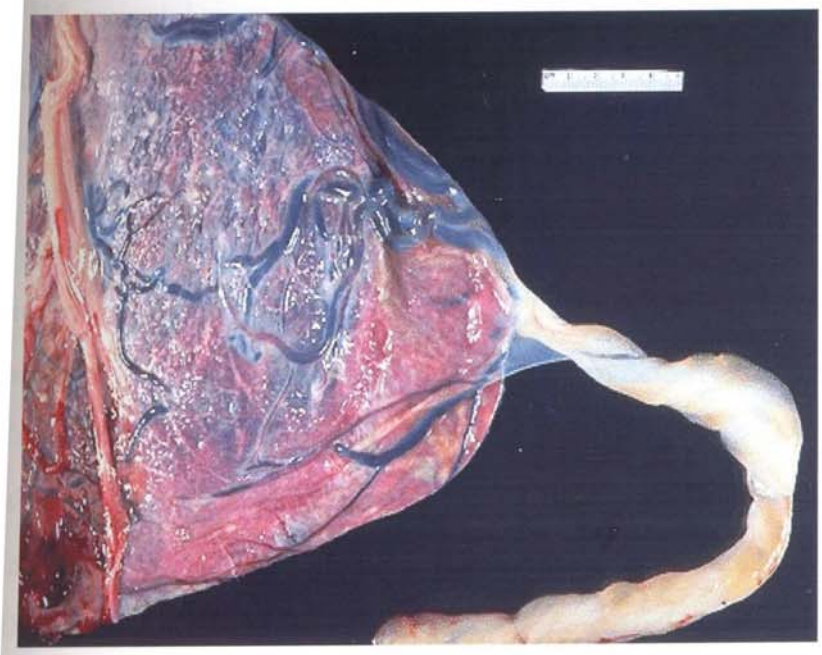
2.Placental Size

Placentas less than **2 cm thick** are associated with intrauterine growth retardation of fetus. Placentas **more than 4 cm thick** have an association with maternal diabetes mellitus, fetal hydrops and intrauterine fetal infections. An extremely thin placenta may represent placenta membranacea, which is associated with a very poor fetal outcome.

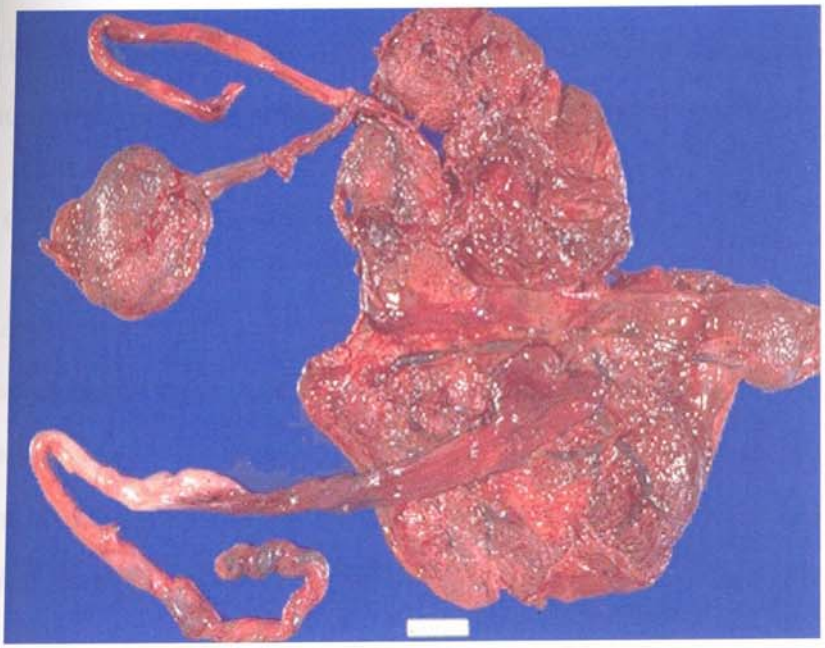
3. Placental Shape

Extra lobes are important primarily due to **retained placental** tissue. Blood may be adherent to the maternal surface of the placenta,

VELAMENTOUS INSERTION OF CORD



SUCCENTURIATE LOBE



particularly at or near the origin. If it distorts the placenta, it may represent an abruption. **Placenta membranacea** – is associated with hemorrhage and poor fetal outcome.

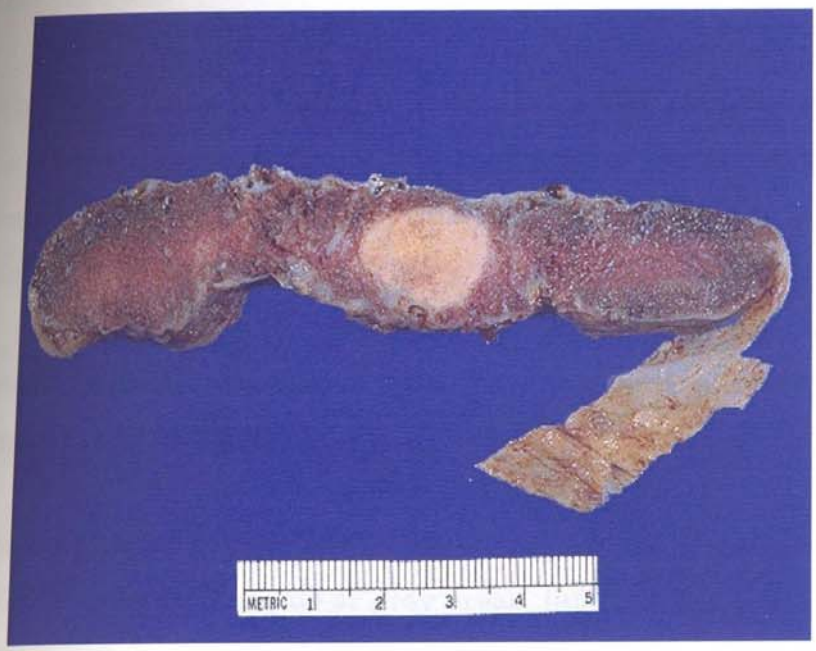
4. Placental Weight Fetoplacental Ratio

A full placental gross evaluation includes weight and dimensions. Although usual term placenta is **22 cm diameter, 2-2.5 cm thick and weight about 470g**, there is considerable variation. Absolute weights < 350 gm> 750 gm in complete term placenta, suggest intrinsic villous pathology that may be revealed only by microscopy.

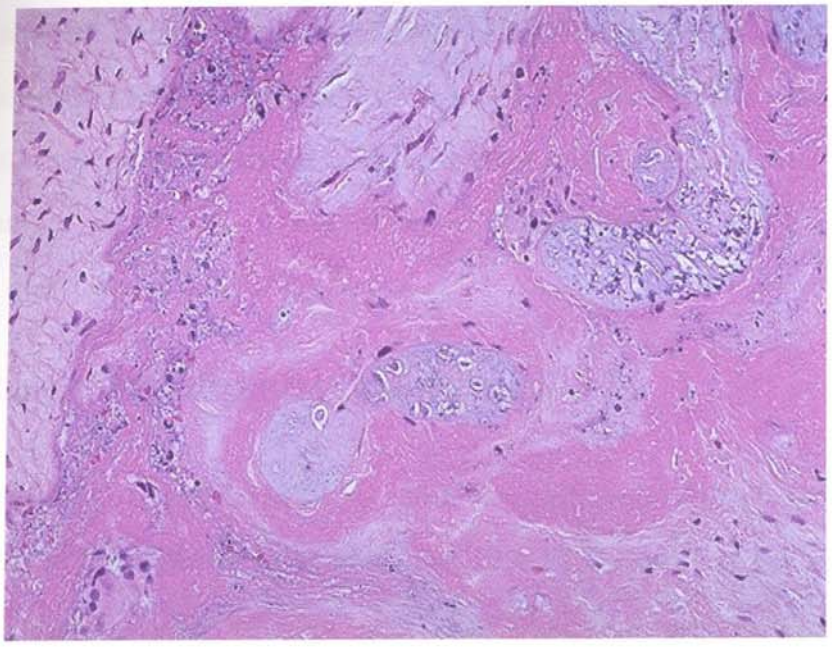
The use of fetal / placental weight ratios offer another measure to assess placental size. The ratio outside normal range for that GA warrants histological examination **(CYNTHIA G. KAPLAN 1996).** According **to NAEYE 1987** a **low** placental weight is associated with maternal uteroplacental vascular insufficiency, congenital anomalies.

An **overweight placenta** was associated with villous edema, maternal diabetes mellitus, maternal, fetal anaemia, hydrops, blood clot with intervillous thrombus or subchorionic bleeding.

INFARCT MACROSCOPIC VIEW



INFARCT MICROSCOPIC VIEW



5. Abnormalities Of Maternal Placental Surface

Favourable perinatal outcome is dependant on good maternal uteroplacental circulation. The 2nd wave of vascular cytotrophoblast invasion may be defective in patients with preecclampsia, SLE, essential hypertension. These manifest microscopically as acute atherosis (Fibrinoid necrosis with lipid deposition) hyperplastic atheroscleromas and necrotizing arteritis of maternal spiral arteries. These vascular lesions cannot be accurately evaluated in remnants of dedidua parietalis as they occur in intramyometrial decidual spiral arteries. Placental site biopsy is required to study these lesions.

A. Infarction

The most dramatic and easily recognized visible sign of maternal uteroplacental vascular insufficiency. It is most common in association with preeclampsia **(34 – 60%)** essential HT, SLE (27.70%) according to **NAEYE 1987**. It also occurs in uncomplicated (25%), prolonged (30%) pregnancy. If infarcts occupy less than 5% of placental mass they are usually unimportant. Infarcts are significant when they are **central** and greater than **20mm** in greater dimension. **Fresh infarcts** are red, dark areas seen in advanced maternal age, PIH, SLE. **Old infarcts** are pale or gray areas. Infarct is associated with significant perinatal mortality and morbidity.

According to NAEYE (1987) true infarction was responsible for 2.4 still births / 1000 births.

Maternal floor infarction is a heavy deposition of fibrin in decidua basalis associated with atrophy of villi. It has a high recurrence rate which is a definitive cause of recurrent early and late pregnancy loss and IUGR. It is found in about 6% on the study of **KAUFFMAN (1996)** with a perinatal mortality of 0.8 / 1000 births.

B. Perivillous Fibrin Deposition

These are firm grey areas of no clinical significance unless extensive. It is present in upto 22% of term placenta, 12% PIH; essential HT and 6% in Diabetes mellitus according to **MOE 1969.** No maternal factor is associated with increased incidence of this lesion. This does not usually contribute to poor perinatal outcome unless **more than 40%** placenta is involved. The clinical significance **of subchorionic fibrin deposition is unknown (FOX 1987)..**

C.Placental Bleeding

Clot especially an adherent clot towards the center of placenta with distortion of shape is associated with abruption. **Marginal** (Fresh clot) hematoma is of no significance if the clot is small.

D. Placental Calcification and Septal Cyst

Calcification is rarely seen before 36 weeks and is not a factor in poor perinatal outcome. **(TINDALL AND SCOTT 1965, BRUNDT 1973)** – there is no association between fetal hypoxia, low birth weight and calcification. Incidence of gross placental calcification is **19% (FOX 1964).**

Septal cyst is seen in 11.20% of full term uncomplicated pregnancies usually in the subchorionic zone. These are of interest due to gross appearance on cross sectioning placenta and association with DM ,Rhesus disease and are of no clinical significance.

E. Choriocarcinoma resembling a fresh infarct, hydatidiform mole are very rare with a normal gestation.

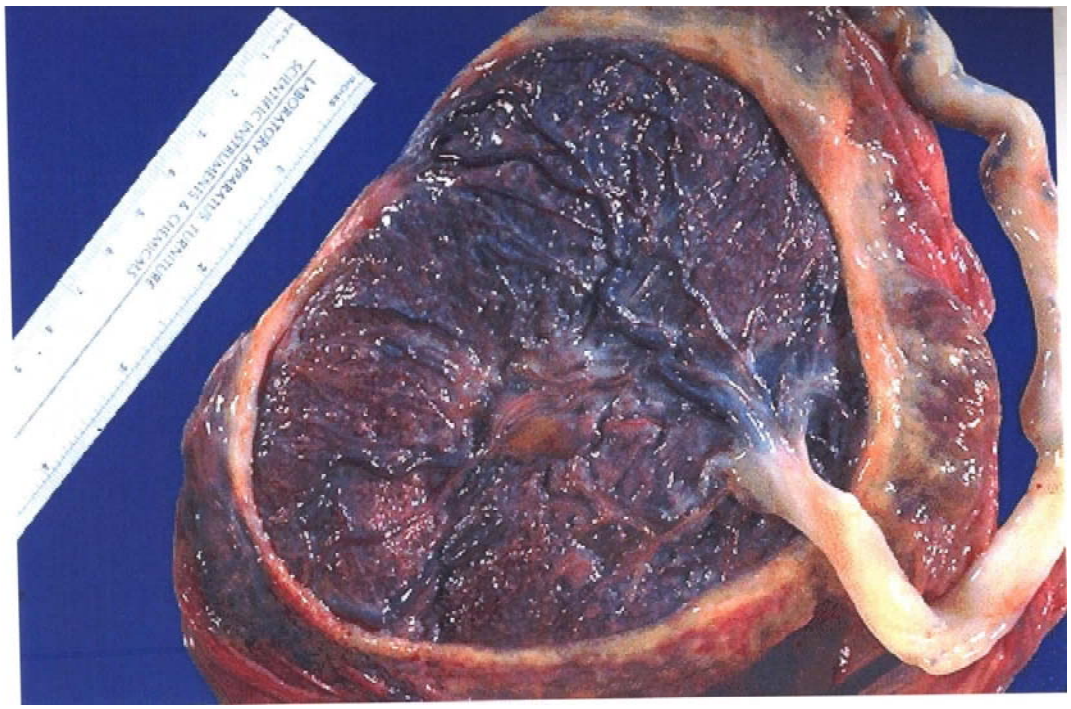
F. Intervillous thrombosis has been reported with Rhesus disease, preeclampsia, ABO incompatibility with no clear association with perinatal outcome.

6. ABNORMALITIES OF FETAL PLACENTAL SURFACE

I. GROSS

i. **Fetal anaemia :** Pale fetal surface is seen in anaemia in newborn, hydrops, hemorrhage, requiring transfusion.

CIRCUMVALLATE PLACENTA



Circumvallate placenta is seen in prematurity, abruption, multiparity, early fluid loss.

ii. **Circummarginate placenta** is of no clinical significance but may be associated with an increase in fetal malformations.

iii. **Amnion nodosum** multiple, tiny, white, yellow nodules is seen in Oligohydramnios, Renal agenesis, pulmonary hypoplasia.

iv. **Squamous metaplasia** multiple, tiny, white, yellow nodules around cord insertion is of no significance.

v. **Amniotic bands** may cause amputation of parts, death.

vi. **Fetus papyraceous and compressus** with deceased twin.

II. Disorders of Fetoplacental Vasculature

I. Fetal Artery Thrombosis

Fetal artery thrombosis is present in 4.5 – 10 % of term placenta and increased in diabetes mellitus according to **DRISCOLL, 1965.** This is usually seen as thrombosis of large surface chorionic plate vessels causing distinct well demarcated pale area of placental parenchyma. Associated histological features include avascular or hypovascular villi devoid of fetal vessel with villous stromal fibrosis and number of syncytial knot. 50 % or more of placenta to be involved is proved to be clinically significant.

II. Chorioangiosis (Villous Hypervascularity)

These are fleshy dark red lesions. If large may be associated with fetal hydrops. It is associated with diabetes mellitus, preeclampsia and RH incompatibility and probably represents a compensatory hyperplasia of uncertain cause. The clinical importance of chorioangiosis is that it is seen in placenta of 5% hospitalized newborns and seen in placenta of normal pregnancies. **25% of affected neonates die or have congenital anomalies according to GEOFFREY ALT SHULER SCORT (1996).**

III. Villous Hypovascularity

This finding refers to small villi with too few vessels per terminal villi. It is a feature of delayed villous maturity resulting from obstruction by thrombus, obliterative endarteritis and post term pregnancy. **(FOX, 1987).**

IV. Villous AVascularity

Usually focal and may be due to absence of primary villous vascularisation and thrombosis. At least 50% of villi need to be avascular to cause adverse perinatal outcome.

V. Obliterative Endarteritis

This is characterized by swelling and proliferation of intimal cells, thickening, reduplication of subendothelial basement membrane which can

result in luminal occlusion. Maternal uteroplacental vascular insufficiency is the primary event in causing the change resulting in low birth weight according to **FOX (1987).** It is associated with preeclampsia, HT, DM. It is primarily due to decreased blood flow into intervillous space contributing to poor perinatal outcome.

VI. Villous Edema

Villous edema is associated with diabetes mellitus, Rh incompatibility, preeclampsia, chorioangiosis, syphilis, toxoplasmosis, cytomegalic inclusion disease.

7. Histological Abnormalities Of Chorionic Villi

FOX 1987 classified as abnormalities of villous maturation and abnormalities of differentiation, changes secondary to reduced uteroplacental blood flow, secondary to decreased fetal villous blood flow.

A. ABNORMALITIES OF VILLOUS MATURATION AND DIFFERENTIATION

I. Accelerated Villous Maturation

Normal villous maturation proceeds from stemvilli **(I trimester)** through intermediate villi **(II trimester)** through **terminal villi** (30 wks to term) according to **KAUFMANN ET AL 1979.** The net physiological

effect is a six fold increase in villous surface area at 20-40 weeks gestation. Accelerated maturation for GA is recognized by decrease in villous size, increase in the number of syncytial knot and inappropriate increase in the number of vasculosyncytial membranes. Such accelaration occurs commonly with PIH, essential hypertension. Cytotrophoblastic hyperplasia and basement membrane thickening also occurs. Placental weight is low.

II. Delayed Villous Maturation

Villous immaturity is recognized by presence of large villi, with stromal density and lack of vasculo sncytial membranes. It is seen in DM, hydrops, anaemia, congenital syphilis and congenital abnormalities of fetus **,** but actually occurs more often in the absence of these disorder. When generalized in nature, it is associated with high incidence of fetal hypoxia and growth retardation. At MAGEA WOMEN HOSPITAL, delayed villous maturity is the only abnormal feature noted in some fresh still birth at or near term. It is an important association of perinatal morbidity and morality which deserves study in placenta from late unexpected fresh stillbirth. It is sometime seen in placenta from preterm infants.

III. Irregular Villous Maturaton

It is seen with chronic villitis and in fetus with abnormal Karyotype.

HISTOLOGICAL ABNORMALITIES OF CHORIONIC VILLI IN CERTAIN DISEASES

Pre-Eclampsia

The most striking and characteristic features of the villi in pre-eclampsia is cytotrophoblastic proliferation and thickening of basement membrane – related to the duration and severity of pre-eclampsia.

- Fibrinoid necrosis of the arterial wall
- Villous odema is sometimes seen
- Usually villi shows normal maturity and small proportion shows evidence of delayed maturation.
- **FISHER SJ et al (2000)** showed that invasive cytotrophoblasts manifest evidence of oxidative stress in preeclampsia. Cytotrophoblasts in preeclampsia have increased xanthine oxidase activity and decreased expression of superoxide dismutase that would shift the local balance in favor of increased reactive oxygen species.Associated finding of peroxynitrite deposition suggests local superoxide / nitric oxide interactions that may reduce vascular responsiveness to normal modulators.

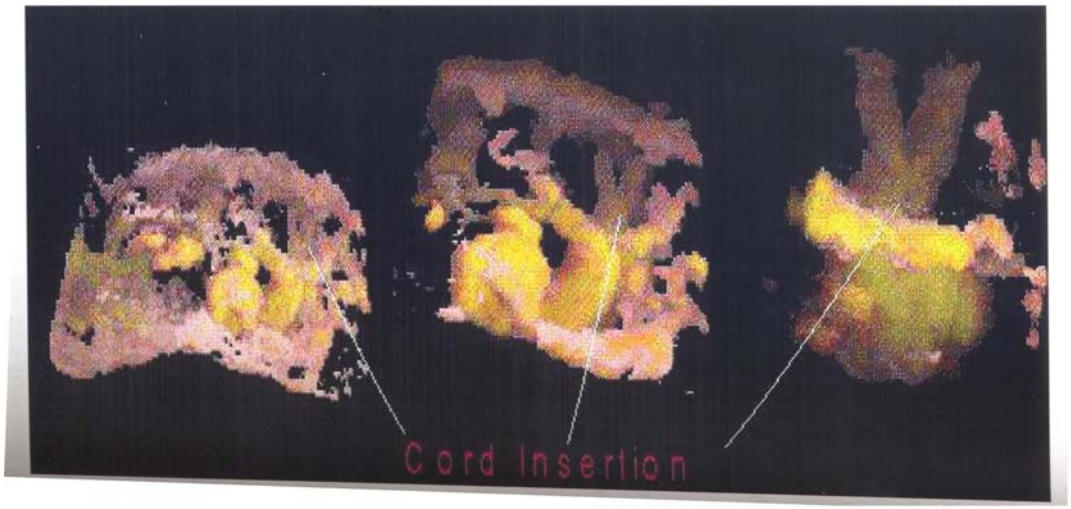
IUGR

25% of placenta tend to be normal in all respects. Further 25% show only evidence of poor fetal perfusion with villous hypovascularity, villous stromal fibrosis and an excessive formation of syncytial knots. 50% show evidence of ischemia. There is evidence of hyperplasia of the villous cytotrophoblastic cells and variable degree of thickening of basement membrane. **SHEPPARD BL, BONNAR J, et al ,1999** showed that cytotrophoblast cells isolated from placentas of IUGR have been shown to express significantly higher levels of plasminogen activator inhibitor-1.This may reduce placental and uteroplacental arterial capacity to lyse fibrin and has been proposed as a mechanism for restricting endovascular conversion and increasing perivillous fibrin deposition.

GESTATIONAL DIABETES MELLITUS

There is generalized **delay in villous maturation,** numerous cytotrophoblastic cells, basement membrane, thickening is seen. Syncytiotrophoblast usually appears normal. There is increased frequency of villous fibrinoid necrosis, edematous villous stroma .**DASKALAKIS G et al (2008)** in his study showed presence of degenerative lesions such as fibrinoid necrosis and vascular lesions like chorioangiosis was apparent, mainly in diabetes, Villous immaturity and presence of NFRBC (Nucleated Fetal Red Blood Cells) as an indication of chronic fetal hypoxia were

CORD INSERTION



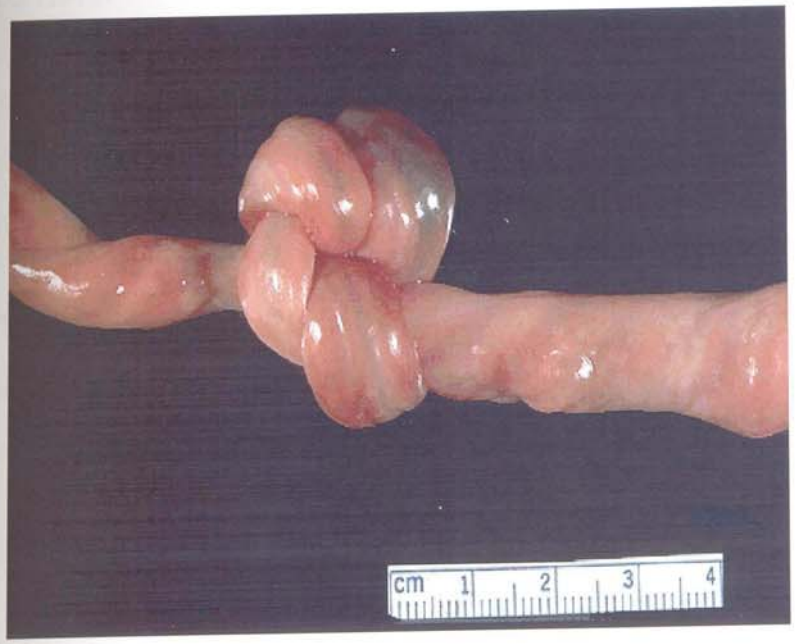
significantly increased in the plaentas of diabetic women.Fetal/placental weight ratio was significantly lower in diabetic women.

8. UMBILICAL CORD ABNORMALITIES

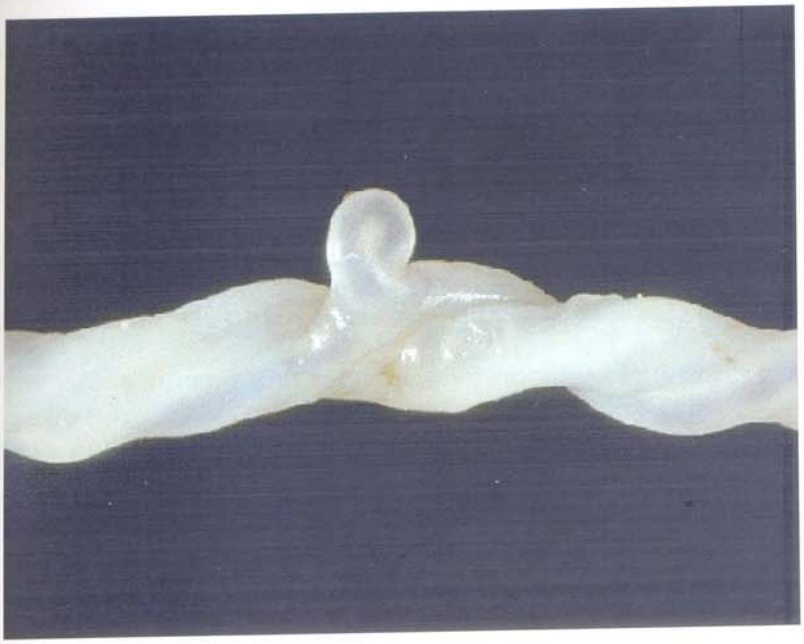
Umbilical cord, the life line of fetus is subject to wide variety of lesions untoward gestational events,whose origin may be structural, mechanical, infections **(RTEPHENE, A. HEIFETZ 1996).** In all the placenta, the length must be measured in the delivery room. True cord length at term < 32 cm or > 100cm warrant further study **(CYNTHIA G KAPLAN 1996).** The typical umbilical cord is long enough to allow the infant to begin nursing before placental delivery. This provides a release of oxytocin to facilitate uterine contractions and both the separation and delivery of the placenta.In part cord length is genetically determined. Cord accidents are well recognized cause of intrauterine fetal death. These cord problems contribute to fetal heart abnormalities.

S**hort cord < 40 cm** is associated with a less active fetus, fetal malformations, myopathic and neuropathic diseases, down syndrome, and oligohydramnios. According to **RAYBURN ET AL 1981.,** is a cause of cord rupture or delayed delivery in 2nd stage. It may also lead to hemorrhage, stricture, malpresentations, abortion, uterine inversion, Werdnig Hoffmann disease.

TRUE KNOT



PSEUDO KNOT



Long cord > 100 cm is associated with fetal hyperkinesis, increased risk of torsion, knots, thrombosis, fetal entanglement, cord prolapse. **Thin cord** and decreased amount of whartons jelly is associated with postmaturity, oligohydramnios, torsion and fetal death.

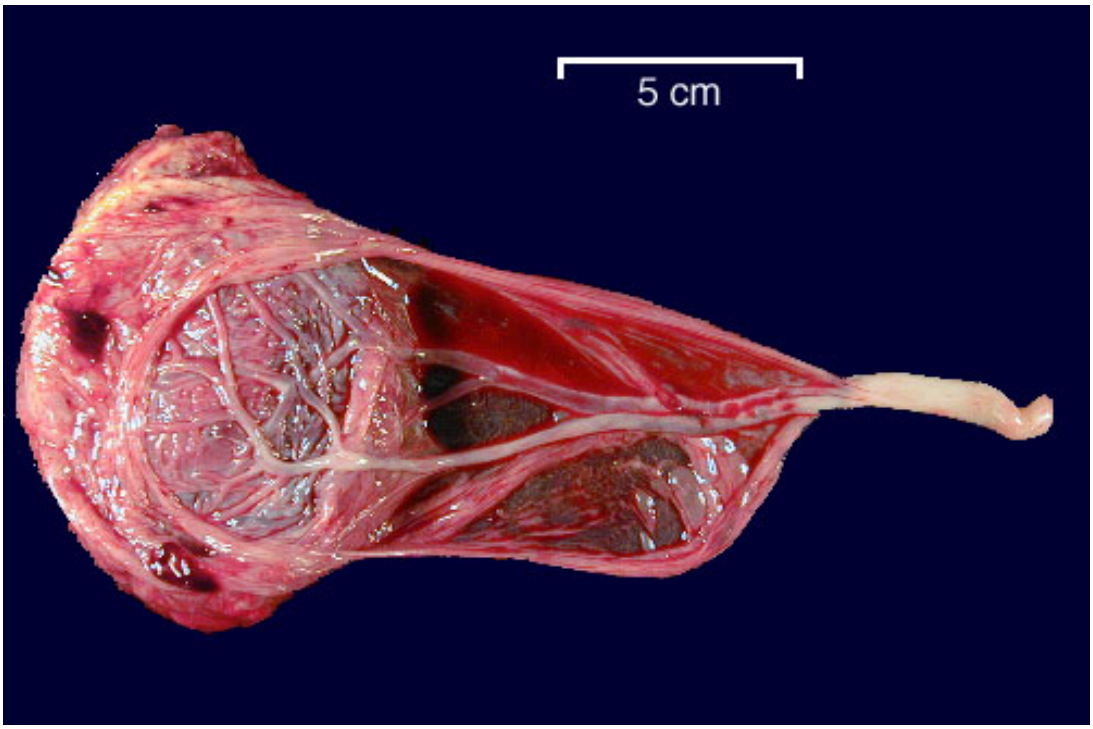
Edema diffuse edema is associated with prematurity, hemolytic disease, preeclampsia, diabetes mellitus, transient tachypnoea of new born. **Focal** edema is seen with **Trisomys,** patent urachus, omphalocoele.

Necrotizing funisitis distinctive segmental resemblance to a barbers pole is seen in syphilis and other acute, subacute and chronic infections.

Velamentous cord insertion – there is greater risk of fetal hemorrhage, thrombosis. Associated with advanced maternal age, Diabetes mellitus, smoking, single umbilical artery, fetal malformations.

Cord hematoma is a rare event, resulting in fetal death from blood loss or compression of cord vessels, more common in late pregnancy.

Cord knots occur in less than 1% of placenta and may account for 8-11% perinatal mortality. **(NAEYE, 1987, FOX 1978).** Cord edema, thrombosis, grooving are morphological changes that indicate knot tightness sufficient to cause obstruction to blood flow.


5 cm

Abnormal number of vessels count the number at more than 5 cm from the placental end of the cord. **Single umbilical artery** has an incidence of 50% of fetal anomalies.. **Two vessel cord, thrombi, congested knots, should be sent for pathological examination (CYNTHIA G. KAPLAN 1996).**

9. PLACENTAL MEMBRANES

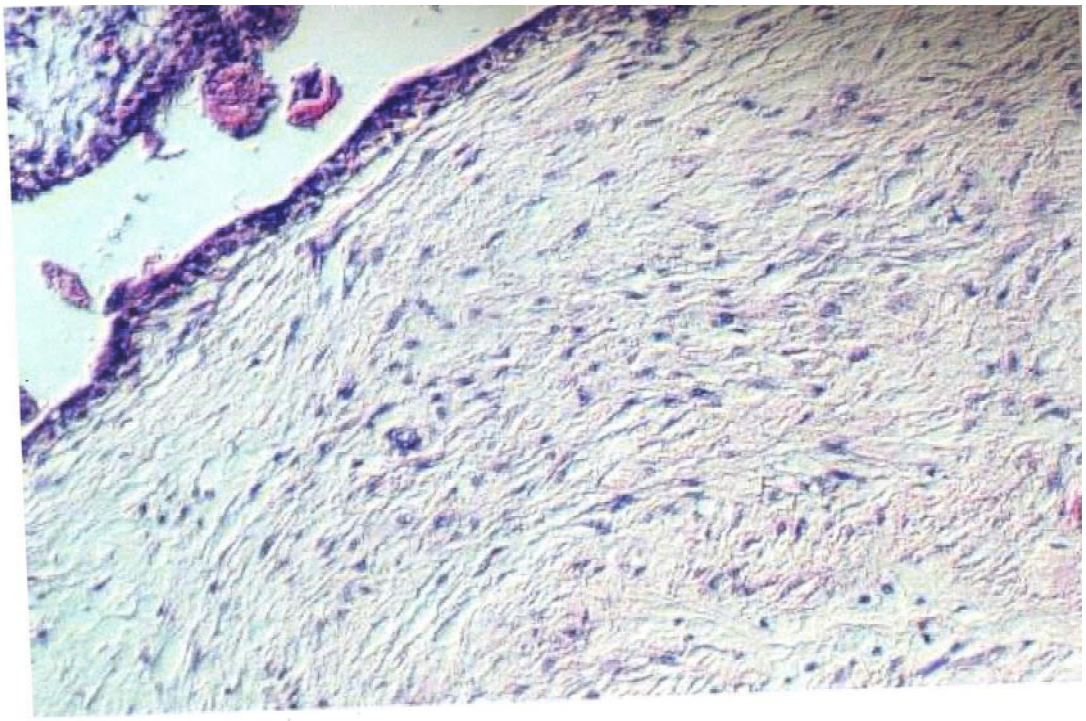
Fetal membranes should be thin grey and glistening. Thick, dull disodoured foul smelling membranes indicate infection.

1. Chorioamnionitis

Acute chorioamnionitis may be recognized grossly by opaque, yellow, malodorous membranes. It is of paramount significance because of its occurrence in atleast 20% of placentas and its clear association with preterm labour, intrauterine hypoxia, fetal infection. It is complicated by stillbirth, prematurity. **(ALT SHULER 1996)**

Most cases are due to ascending bacterial infections that reach the amniotic fluid through the membranes adjacent to cervical os **(BLANK).** The bacteria isolated are generally normal inhabitants of birth canal. If **fecal odour** possible **Fusobacterium** or **Bacteroides** infection. If **sweet** odour possibly **Clostridium or Listeria.**

MEMBRANE - NORMAL VIEW



II. Meconium Staining

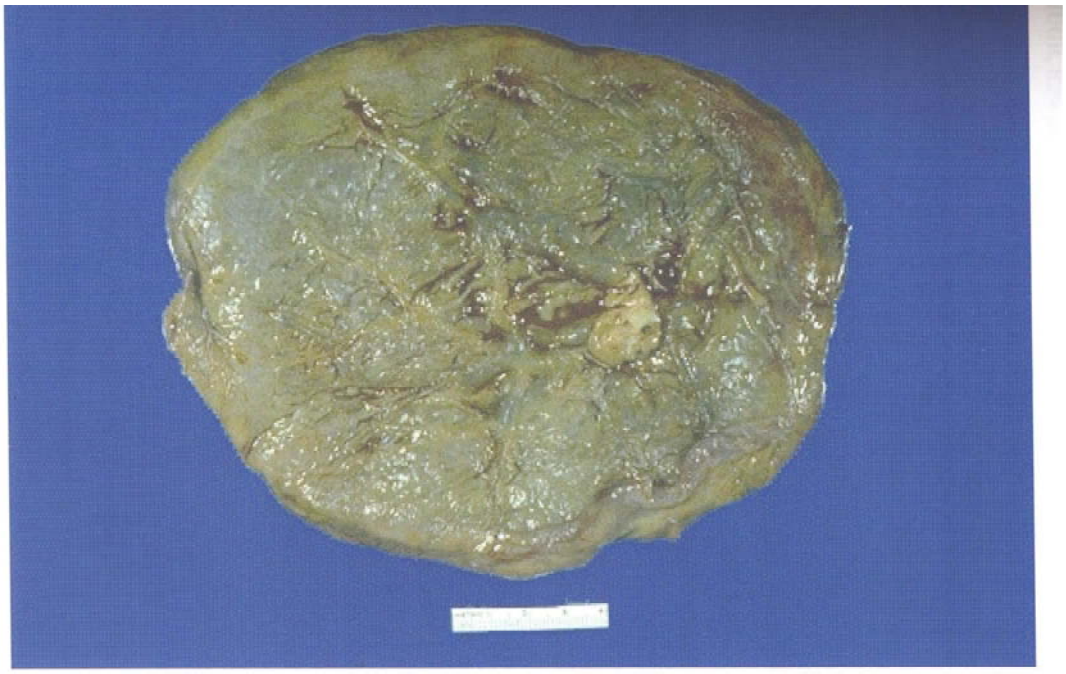
Green odoured fetal membranes are frequently the result of meconium staining. Thick green lime that easily rinses off the membranes is meconium. Meconium staining is present in 20% of deliveries in association with acute chorioamnionitis, PROM, abruptio placenta, cocaine use. It is seen in 61% of post term **(AL STHULER RG. 1996).**

PLACENTAL BIOPSY

- According to **KLIMAN – HJ: PEROTTA – PL, 1995**, a placental biopsy specimen after delivery is reasonably sensitive for diagnosing villous abnormalities that reflect acute and chronic stresses to the placenta. It may be useful to develop a placental biopsy that can be performed safely during pregnancy. Such a biopsy could be the basis for the rational treatment of some diseases of pregnancy.

- According to **JOSEPH, F. YETTER III(1998)**, pathological examination of placenta is indicated in poor pregnancy outcome, Systemic maternal disorders, third trimester bleeding or maternal infection.

MECONIUM STAINED PLACENTA



DOPPLER VELOCIMETRY IN PREECLAMPSIA:

- **FLEISCHER ET AL** in 1986 studied the correlations of severity of preeclampsia with pregnancy outcome. He concluded that when the uterine artery systolic diastolic ratio was more than 2.6 during III trimester, incidence of fetal distress during delivery is high. According to him, about 67% of hypertensive patients with abnormal umbilical blood flow deliver growth retarded babies.

THALER ET AL in 1992 reported that an increased uterine artery resistance index (RI) without a notch poorly correlates with adverse fetal outcome. He concluded that hypertensive pregnant women were divided into four groups based on the presence of absence of a uterine notch or uterine artery RI. The presence of both was associated with most severe complications. In his study, perinatal mortality was 21% and 74% of fetuses were growth retarded.

Schulman H in 1987 – studied the clinical implications of Doppler ultrasound analysis of the uterine and umbilical arteries – Am.J. Obstet gynecol. 156:889-893

DEUTINGER ET AL believed that S/D ratio plateaued at 24 weeks. Retention of the early diastolic notch is thought to represent persistence of inherent total high impedance of uterine artery circulation.

ARISTIDOU ET AL noted that the uterine artery notch was a good predictor of poor perinatal outcome.

KOFINAS ET AL found that the perinatal outcome correlated best with placental uterine artery, the mean index using both uterine arteries next best, and the non placental uterine artery the poorest predictor.

Persistence of the uterine notch indicates severe hypertensive disease and its presence in III trimester is associated with increased rate of IUGR, caesarean delivery for fetal distress and preterm delivery.

The difference in main uterine artery waveform indices between normal and pathologic pregnancies is probably greater than at other sites of uterine artery. Measurement of the main uterine artery may be more reproducible and allows standardized longitudinal follow up, because it is a reflector of total subplacental resistance, remains the most clinically important parameter.

The prevalence of the perinatal outcome measured will have the biggest impact as will the definition of abnormal uterine Doppler velocimetry. The data suggests that the presence of the notch is the most important criterion.**SAGOL S, OZKINAY E et al (1999)** showed that increased uterine artery resistance parellels histologic evidence of impaired trophoblastic migration.

AIM OF THE STUDY

AIM OF THE STUDY

i. To compare the placental abnormalities in normal and specific high risk pregnancies by using Doppler and Histology.

ii. To study the possible, probable correlation with fetal outcome.

MATERIALS AND METHODS

MATERIALS AND METHODS

SETTING

This study was carried out at the Institute of social obstetrics and Govt Kasturba Gandhi Hospital, Triplicane, Chennai.

STUDY DESIGN

It is a prospective study of morphology and histology of placenta in randomly selected normal and specific high risk pregnancies and its correlation with fetal outcome.

PERIOD OF STUDY

From May 2009 – October 2010

MATERIALS AND METHODS

500 pregnant women between 21-35 yrs of age between gestational age of $\geq$ 36 wks, coming to antenatal OP at KGH are selected.

Each women is analysed in detail with

1) Age
2) Parity
3) Associated Medical / Obstetric Complication
4) Gestational Age

They are classified into 2 groups.

Group I - Normal cases (270)

Group II - Specific High risk cases (230)

Preeclampsia – 80

Gestational Diabetes Mellitus – 80

IUGR – 70

History, Physical Examination, Obstetrical Examination, Basic investigations, Hb% , urine analysis, blood grouping typing, pertaining to individual patients are carried out.

They are offered the following test.

Uterine Artery Doppler Ultrasonogram:

Following are mainly looked

1) Systolic Diastolic Ratio
2) Early Diastolic Notch
3) Pulsatility Index
4) Resistance Index.

They are followed till delivery once in 2 weeks and at the time of delivery, placenta is studied both grossly and histologically after formalin fixation. Fetal outcomes in these cases are observed.

INCLUSION CRITERIA

For Normal Pregnancies:

- Primigravida between the age of 21-35 yrs, ≥ 36 weeks of gestation.
- With singleton gestation
- No associated medical / obstetric complication.

For specific high risk pregnancies

- Primigravida between the age of 21-35 years, ≥ 36 weeks of gestation, with singleton gestation with the following high risk factors.
- Preeclampsia, mild, and severe
- Gestational Diabetes Mellitus
- Intrauterine growth restriction.

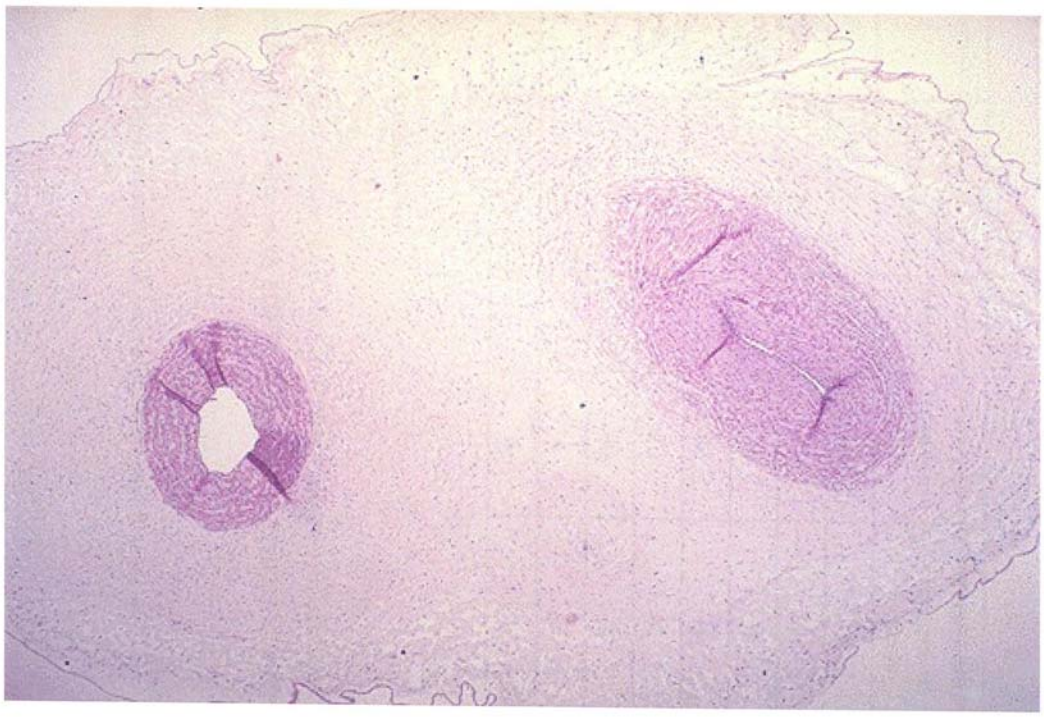
EXCLUSION CRITERIA

1) Pregnant women < 20 yrs, > 35 yrs.
2) Multiple pregnancy
3) Anemia complicating pregnancy
4) Placenta previa, Abruptio placenta.
5) HIV, HBSAg, Tuberculosis, syphilis complicating pregnancy
6) Pregnant women other than primigravida
7) Other known medical / obstetrical complication not mentioned in high risk Inclusion criteria.

EXAMINATION OF PLACENTA

Each placenta was washed with tap water and drained off its blood. On initial examination of placenta, any gross abnormality of shape, morphometric measurements like size, weight, measurement of cord length and site of cord insertion were noted. For examination of the membranes, a segment of membrane was cut rolled from the margin and pinned for HPE. Cut surface of cord was examined and number of vessels recorded. Each placenta was then trimmed of membranes, weighed and was then examined for calcification and infarction.

SINGLE UMBLICAL ARTERY

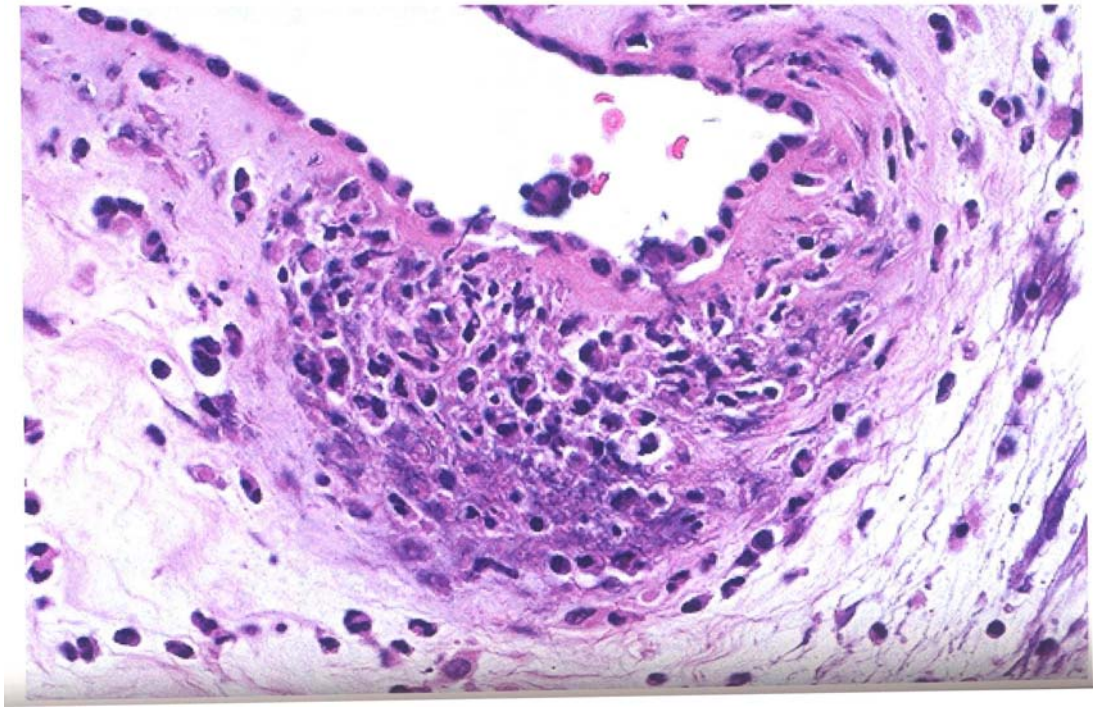


- The placenta was dipped in 10% formalin solution for fixation for a period of 48 hrs. They were then cut at every 2 cm interval into vertical strips. 5µ thin sections were cut from each block and stained with haematoxylin and eosin. Two bits from cord were taken. Each section from placenta was examined with light microscope, first low power and then under high power for villous pathology. Atleast 100 villi are studied.

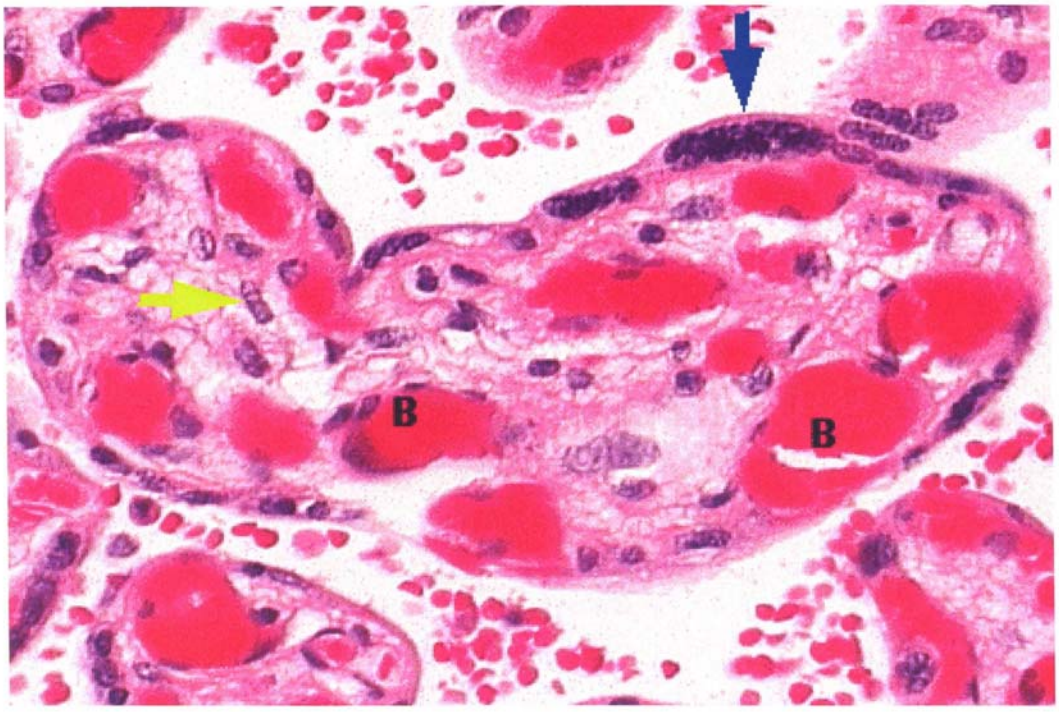
MORPHOLOGY

- Infarction
- Calcification
- Meconium staining
- Septal cyst
- True knot
- Single umbilical artery

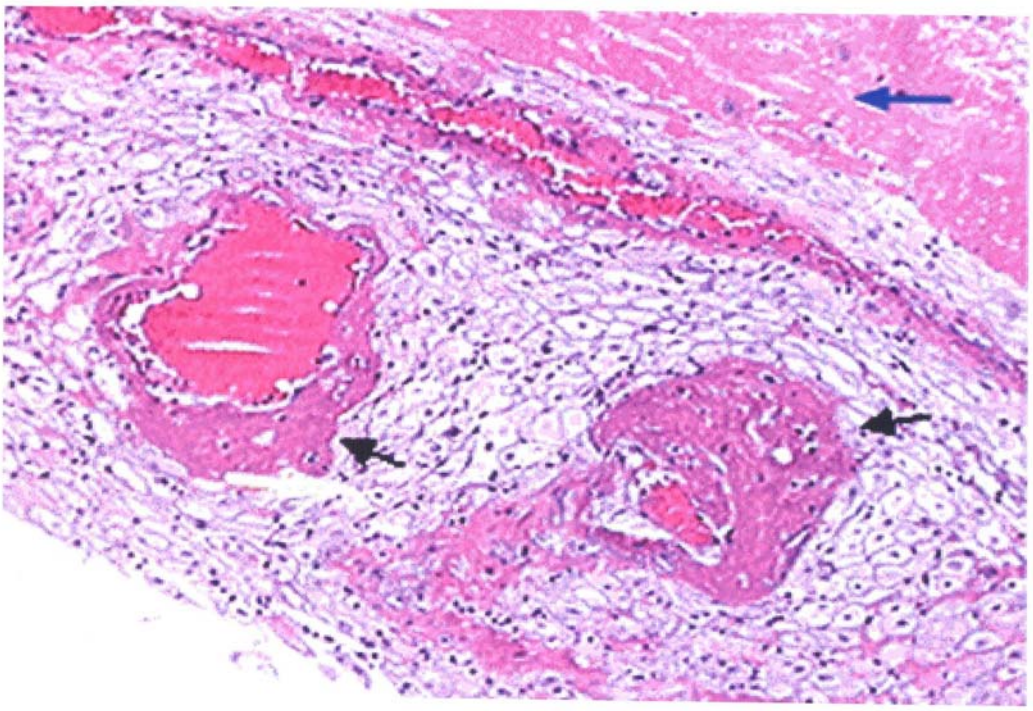
NEUTROPHILIC INFILTRATION OF MEMBRANE



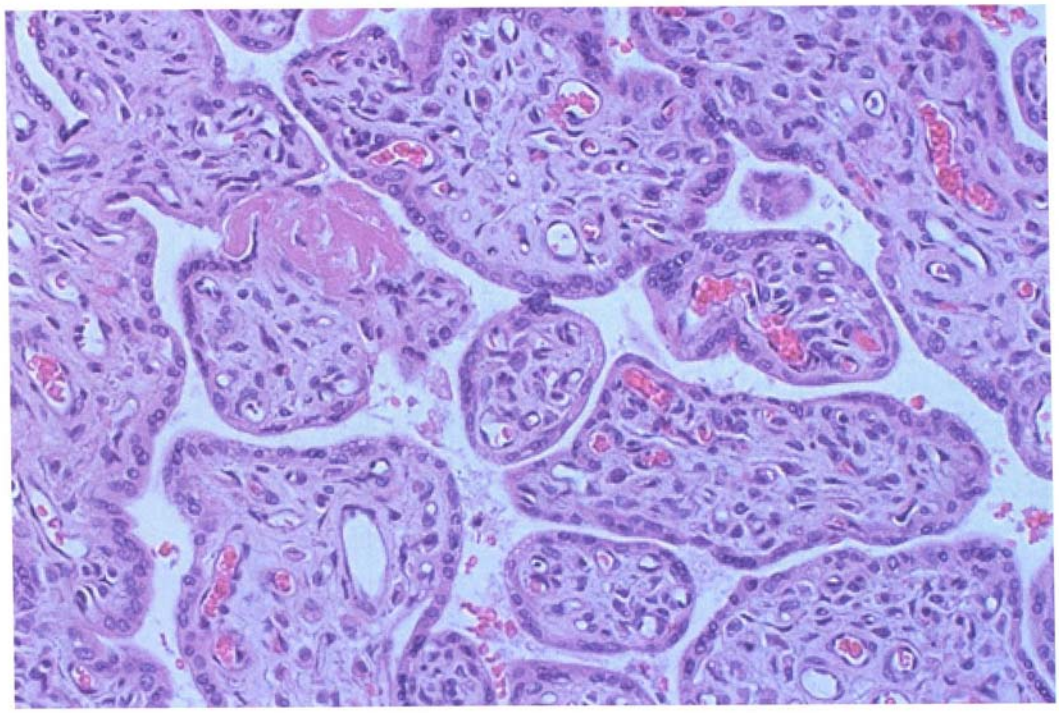
BASEMENT MEMBRANE THICKENING



FIBRINOID NECROSIS



SYNCYTIAL KNOT



BASAL PLATE

- Neutrophilic Infiltration, Abscess
- Infarction
- Fibrinoid changes in vessels.

VILLOUS PATHOLOGY

- Basement membrane thickening
- Syncytial knot count
- Fibrinoid necrosis
- Stromal Fibrosis
- Cytotrophoblastic proliferation
- Calcification
- Infarction

CORD / MEMBRANE

Neutrophilic infiltration, Abscess

FETUS

Babies were followed upto discharge in both groups.

Following data are recorded

- Signs of fetal distress
- Birth weight
- APGAR
- NICU Admission
- Neonatal morbidity and mortality

Correlation of fetal outcome with uterine artery Doppler, morphology and histopathology of placenta was studied using Chi square test, with Yates correction, Fishers exact test in appropriate places.

RESULTS AND ANALYSIS

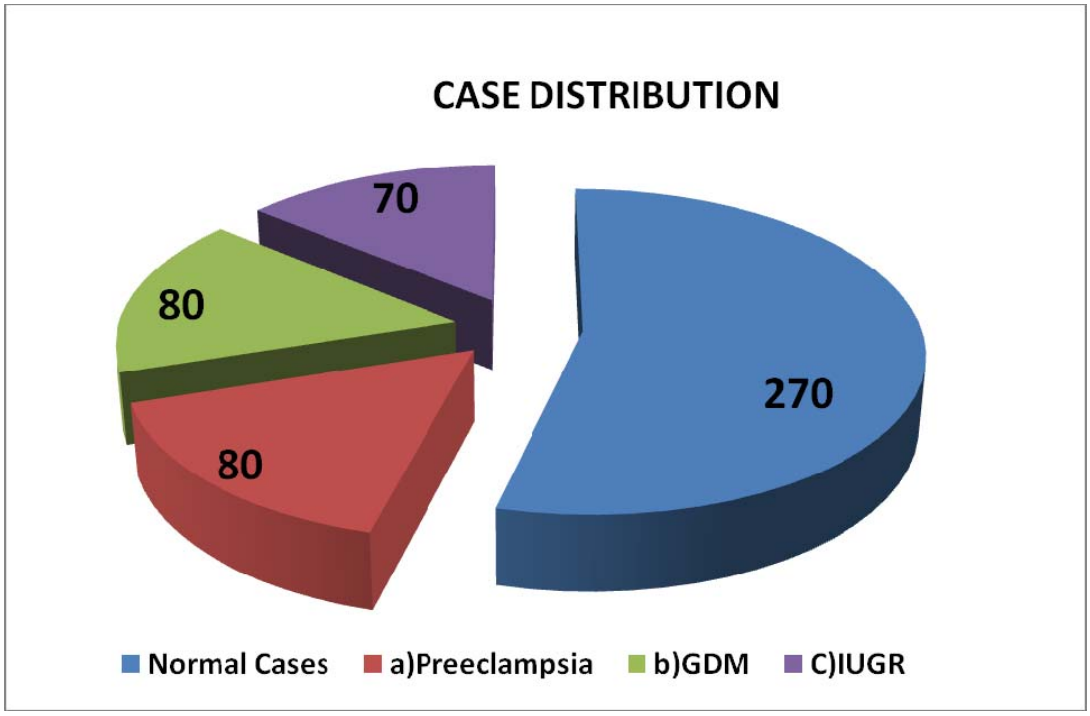

CASE DISTRIBUTION
70
80
80
270
Normal Cases
a)Preeclampsia
b)GDM
C)IUGR

TABLE – I

DISTRIBUTION OF CASES

		Frequency(n)	Percentage %
Group I	Normal Cases	270	54.0
Group II	High Risk Cases		
	a)Preeclampsia	80	16
	b)GDM	80	16
	C)IUGR	70	14
	Total	500	100

Out of 500 cases, 270 were term uncomplicated pregnancies, 230 were pregnancies complicated by various disorders.

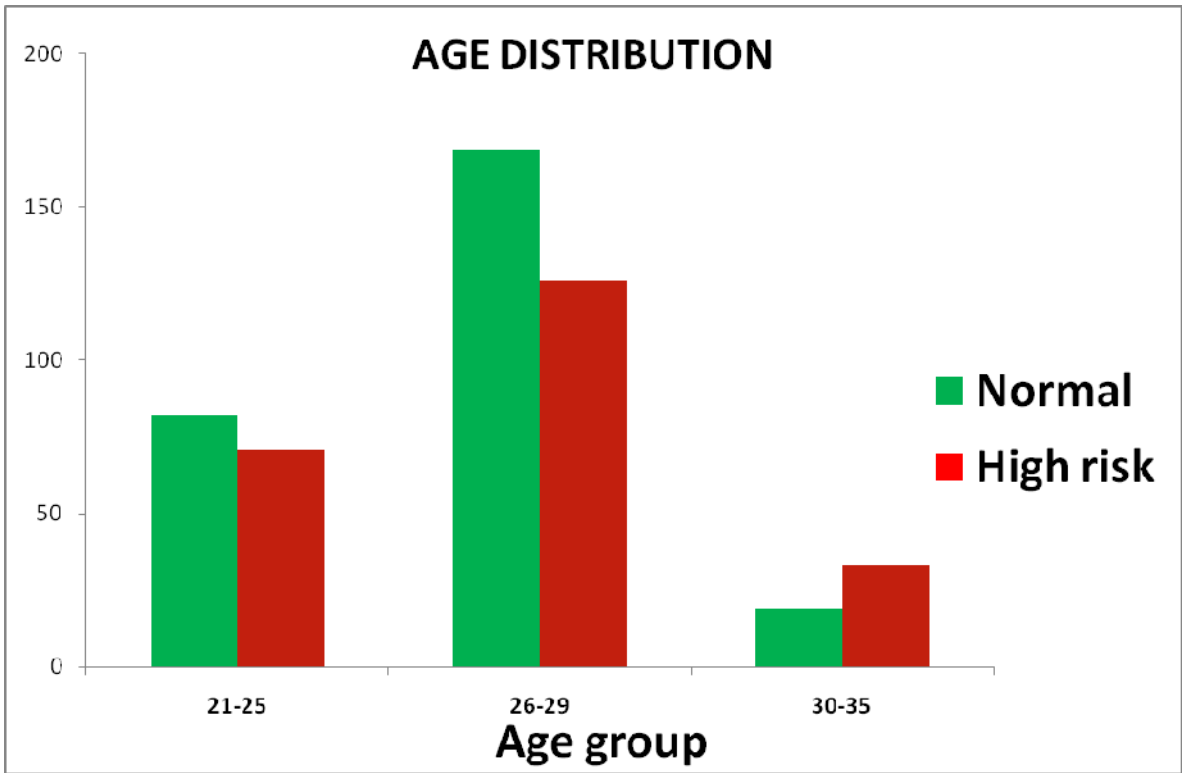


Table-2

AGE DISTRIBUTION

AGE IN	NORMAL	PREECLAMPSIA	GDM	IUGR

YEARS								
	N	%	N	%	N	%	N	%
21-25	82	30.3%	31	38.8%	28	35.0%	12	17.1%
26-29	167	62.6%	38	47.5%	39	48.8%	49	70.0%
30-35	21	7.1%	11	13.7%	13	16.3%	9	12.9%
TOTAL	270	100%	80	100%	80	100%	70	100%

AGE DISTRIBUTION FOR GROUPS

AGE IN YEARS	NORMAL		HIGH RISK	
	N	%	N	%
21-25	82	30.30%	71	38.8%
26-29	167	62.60%	126	47.5%
30-35	21	7.10%	33	13.7%
TOTAL	270	100%	230	100%

This table shows the distribution for age in both groups. Age ranged between 21 – 35 years in both group of patients.

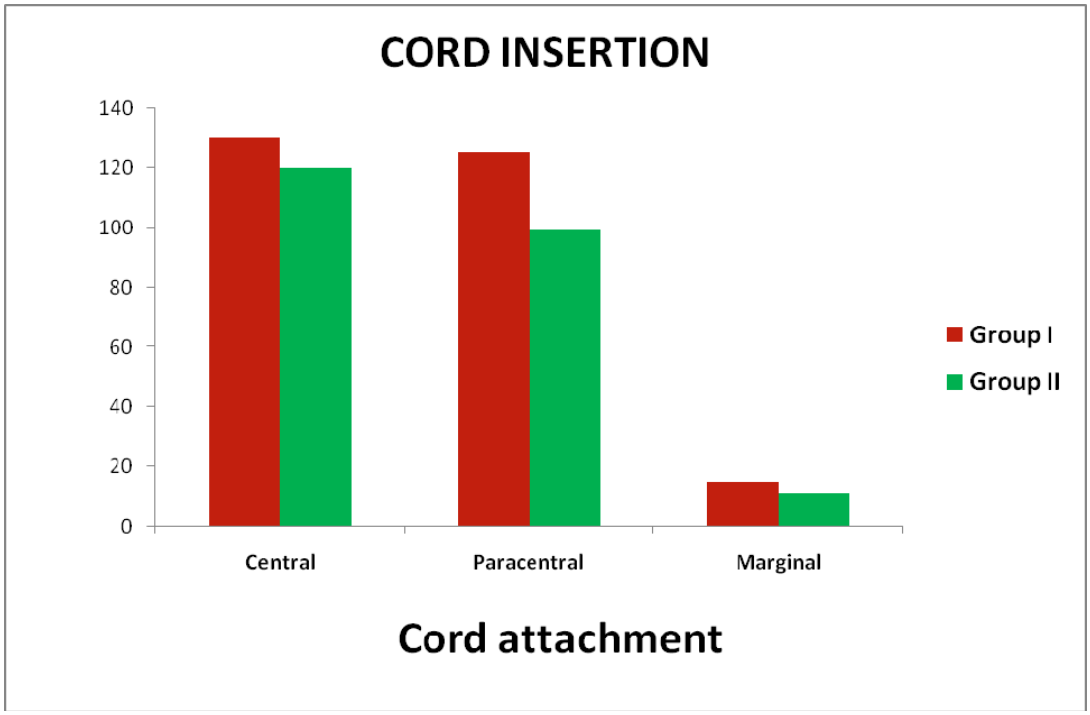

CORD INSERTION
140
120
100
80
60
40
20
0
Central
Paracentral
Marginal
Group I
Group II
Cord attachment

TABLE – 3

GROSS FEATURES

SERIAL NO.	GROSS FEATURES	GROUP I	GROUP II
1	Placental weight (Gms)	450	370
2	Fetal Placental Ratio	5.126:1	3.25:1
3	Cord Length	44.5Cms	46.5Cms
4	Cord Attachment		
	Central	130[48.14%]	120[52.17%]
	Paracentral	125[46.29%]	99[43.47%]
	Marginal	15[5.55%]	11[4.34%]

Above table shows the gross features of placenta in both the groups.

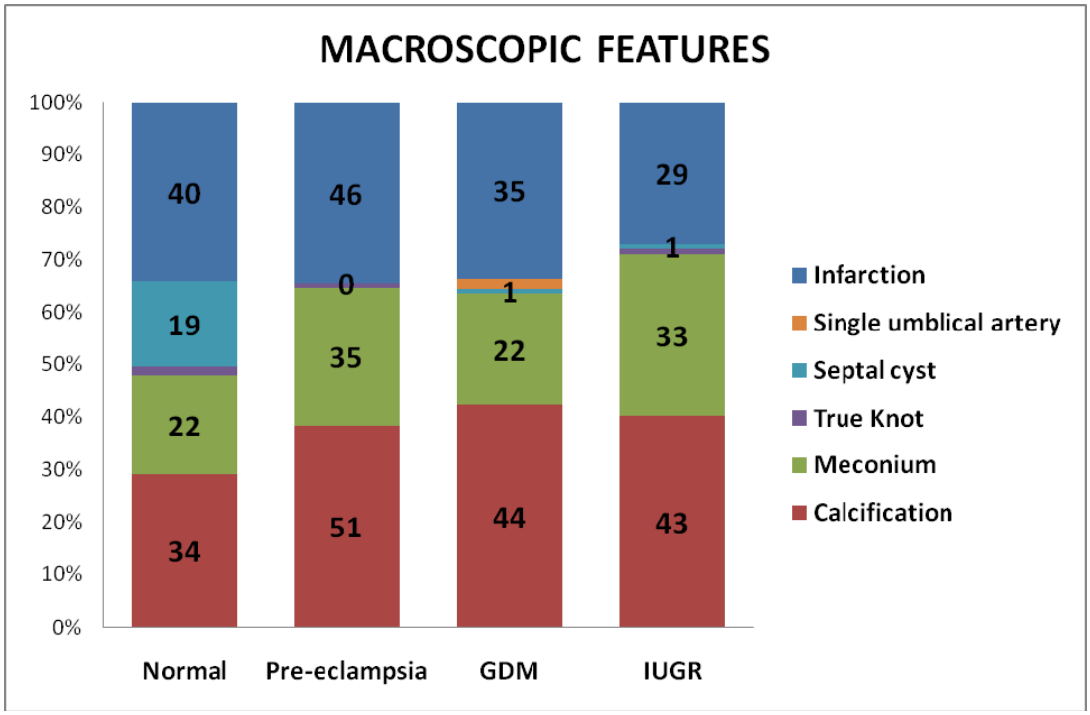

MACROSCOPIC FEATURES
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
40
19
22
34
46
0
35
51
35
1
22
44
29
1
33
43
Normal
Pre-eclampsia
GDM
IUGR
Infarction
Single umblical artery
Septal cyst
True Knot
Meconium
Calcification

TABLE-4

MACROSCOPIC FEATURES OF PLACENTA

Group	Infarction	Calcification	Meconium Stained Membrane	True-Knot	Septal cyst	Single umbilical artery
Normal	40(14.81%)	34[12.59%]	22[8.14%]	2[0.74%]	19[7.04%]	-
Preeclampsia	46[57.5%]	51[63.75%]	35[43.75%]	1[1.25%]	-	-
GDM	35[43.75%]	44[55%]	22[27.5%]	-	1[1.25%]	2[2.5%]
IUGR	29[41.42%]	43[61.42%]	33[47.14%]	1[1.42%]	1	-

Above table shows the macroscopic abnormalities in both the normal and high risk groups.

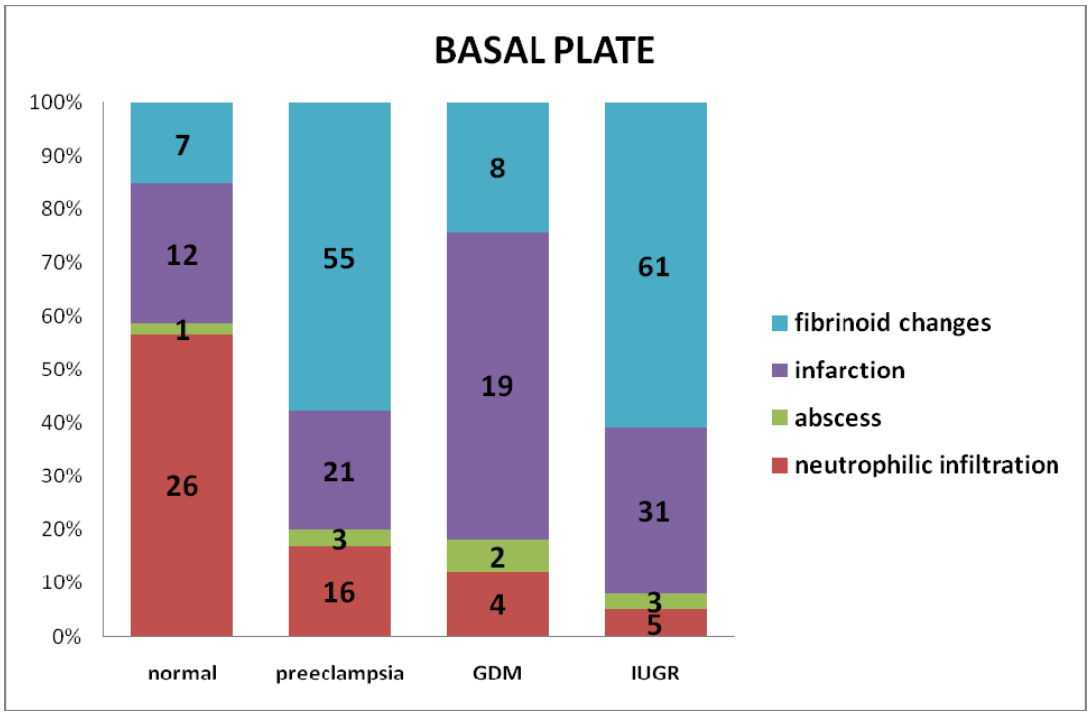

BASAL PLATE
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
7
12
1
26
55
21
3
16
8
19
2
4
61
31
3
5
normal
preeclampsia
GDM
IUGR
fibrinoid changes
infarction
abscess
neutrophilic infiltration

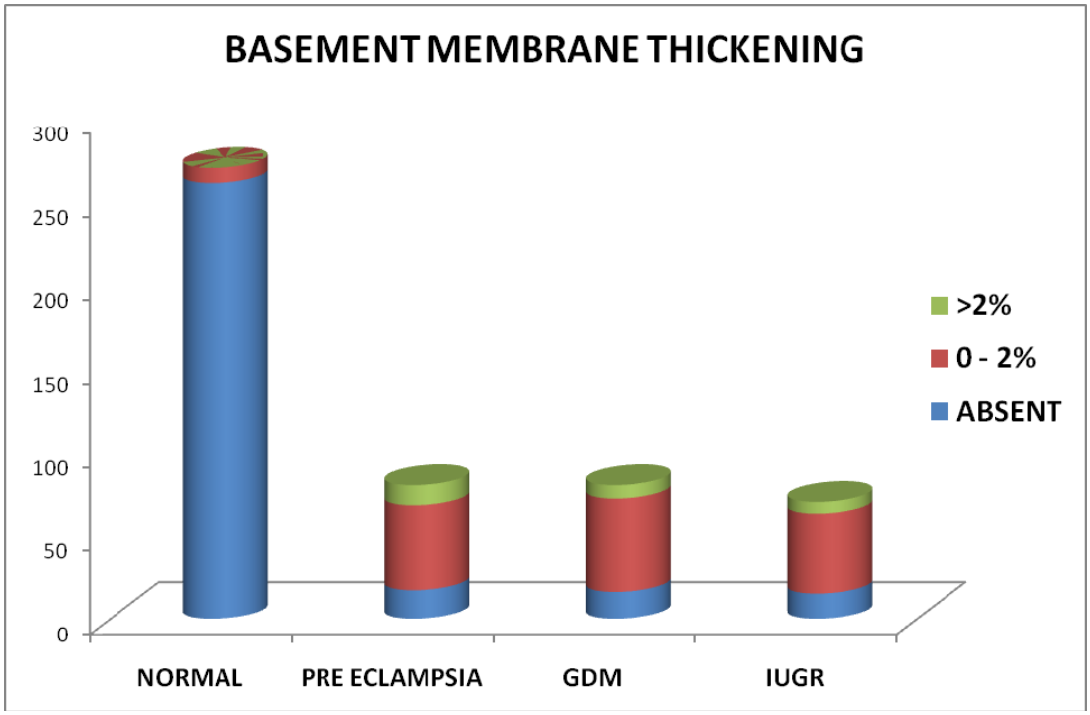
TABLE – 5

HISTOPATHOLOGY OF PLACENTA

BASAL PLATE OR DECIDUA

GROUP	NEUTROPHILIC INFILTRATION	ABSCESS	INFARCTION	FIBRINOID CHANGES
Normal	26 (9.62%)	1 (0.37%)	12 (4.4%)	7 (2.4%)
Preeclampsia	16 (21%)	3 (3.75%)	21 (27%)	55 (69%)
GDM	4 (5%)	2 (2.5%)	19 (25%)	8 (10%)
IUGR	5 (7%)	3 (4.28%)	31 (43%)	61 (87%)

Above table shows the histopathological features of basal plate in both the groups.



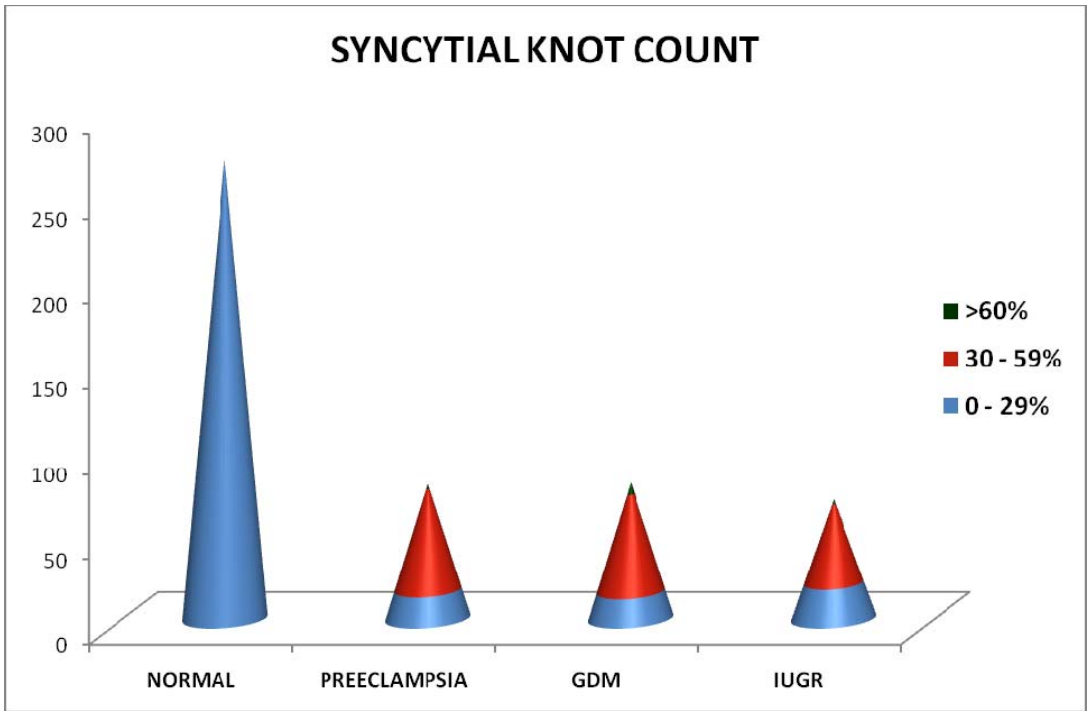


TABLE – 6 VILLOUS PATHOLOGY

6.1 BASEMENT MEMBRANE THICKENING

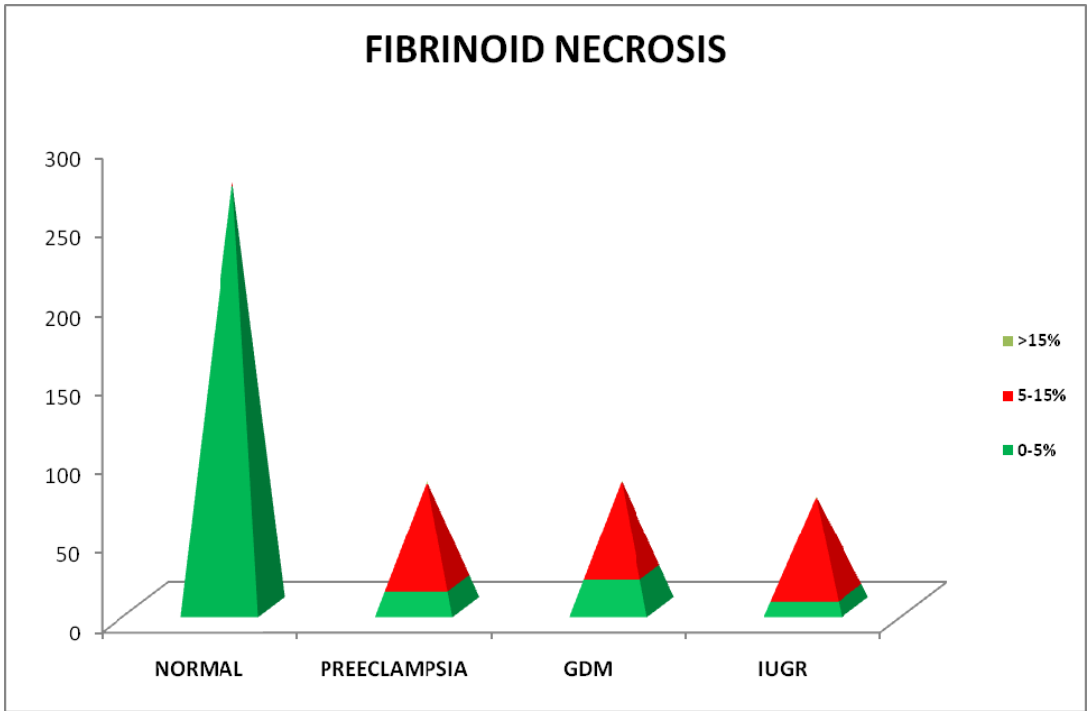
BASEMENT MEMBRANE THICKENING	NORMAL	PREECLAMPSIA	GDM	IUGR
Absent	261 (96.7%)	17 (21.85%)	16 (20%)	15 (21.73%)
0-2%	9 (3.3%)	51 (65%)	56 (70%)	48 (69.56%)
>2%		12 (13.75%)	8 (10%)	7 (10%)

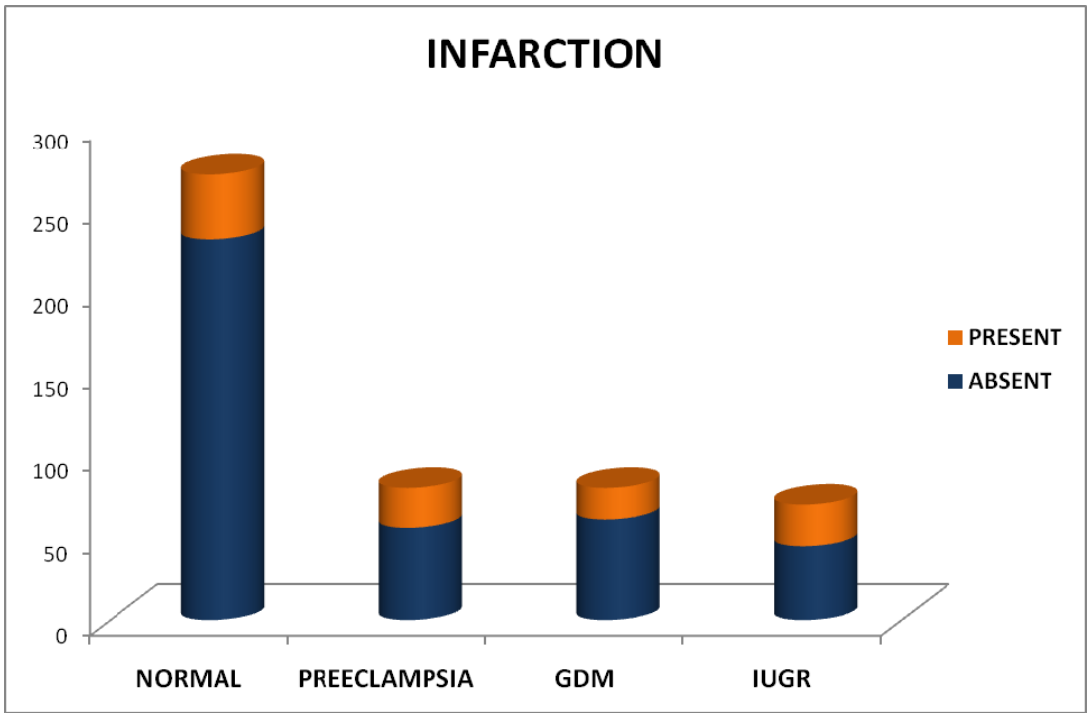
Above table shows basement membrane thickening in normal and high risk cases.

6.2-SYNCYTIAL KNOT COUNT

SYNCYTIAL KNOT COUNT	NORMAL	PREECLAMPSIA	GDM	IUGR
(I) 0-29%	268 (99.25%)	17 (21.17%)	16 (20.1%)	21 (30.43%)
(II) 30-59%	2 (0.75%)	59 (73.36%)	57 (70.69%)	46 (65.21%)
(III) >60%		4 (5.47%)	7 (9.2%)	3 (4.34%)

Above table shows syncytial knot count distribution in Normal and High risk cases.


FIBRINOID NECROSIS
300
250
200
150
100
50
0
NORMAL
PREECLAMPSIA
GDM
IUGR
>15%
5-15%
0-5%


INFARCTION
300
250
200
150
100
50
0
NORMAL
PREECLAMPSIA
GDM
IUGR
PRESENT
ABSENT

6.3-FIBRINOID NECROSIS

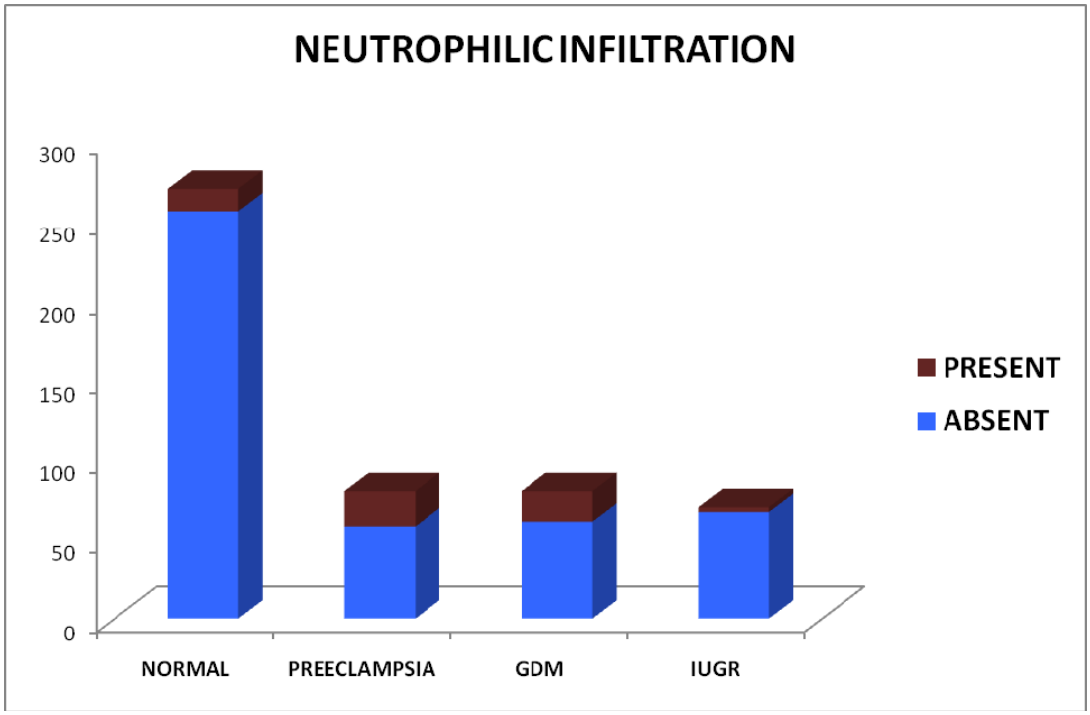
FIBRINOID NECROSIS	NORMAL	PREECLAMPSIA	GDM	IUGR
I) 0-5%	267 (99.2%)	15 (19.73%)	22 (28%)	9 (12.85%)
(II) 5-15%	3 (0.8%)	63 (79.6%)	57 (71%)	60(85.71%)
(III) >15%		2 (065%)	1 (1.2%)	3 (1.4%)

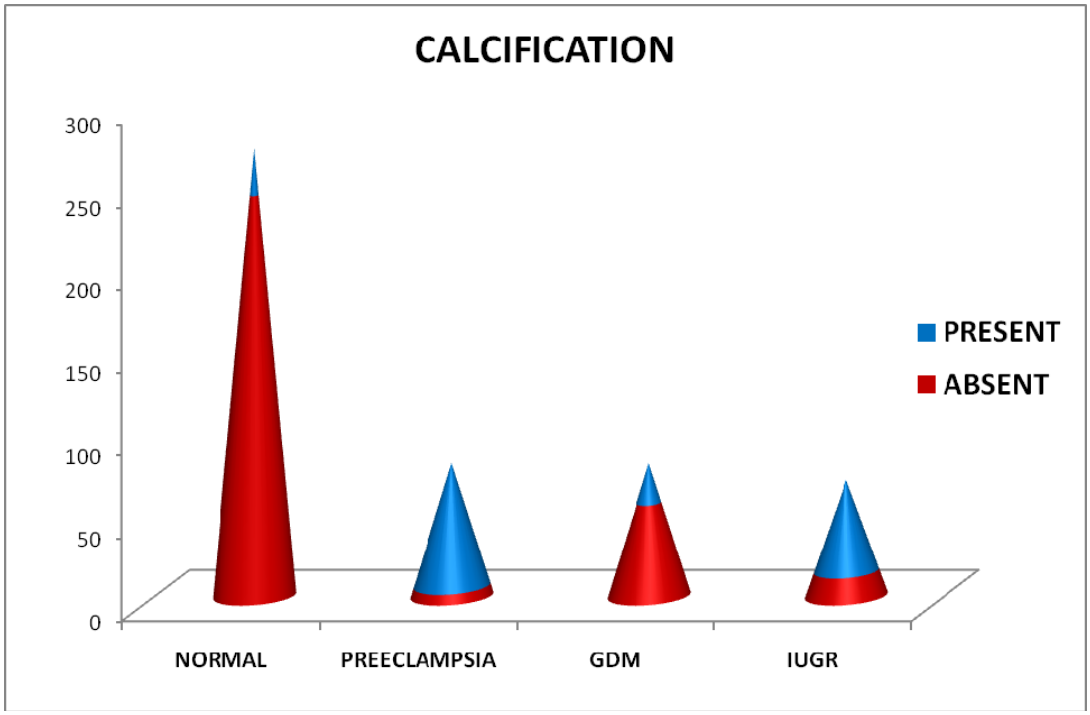
Above table shows fibrinoid necrosis distribution in normal and high risk cases.

6.4-INFARCTION

INFARCTION	NORMAL	PREECLAMPSIA	GDM	IUGR
Absent	231 (85.6%)	56 (70%)	61 (76.25%)	45 (64.28%)
Present	39 (14.4%)	24 (30%)	19 (23.75%)	25 (35.72%)

Above table shows presence and absence of Infarction in normal and high risk cases.


NEUTROPHILIC INFILTRATION
300
250
200
150
100
50
0
NORMAL
PREECLAMPSIA
GDM
IUGR
PRESENT
ABSENT


CALCIFICATION
300
250
200
150
100
50
0
NORMAL
PREECLAMPSIA
GDM
IUGR
PRESENT
ABSENT

6.5-NEUTROPHILIC INFILTRATION

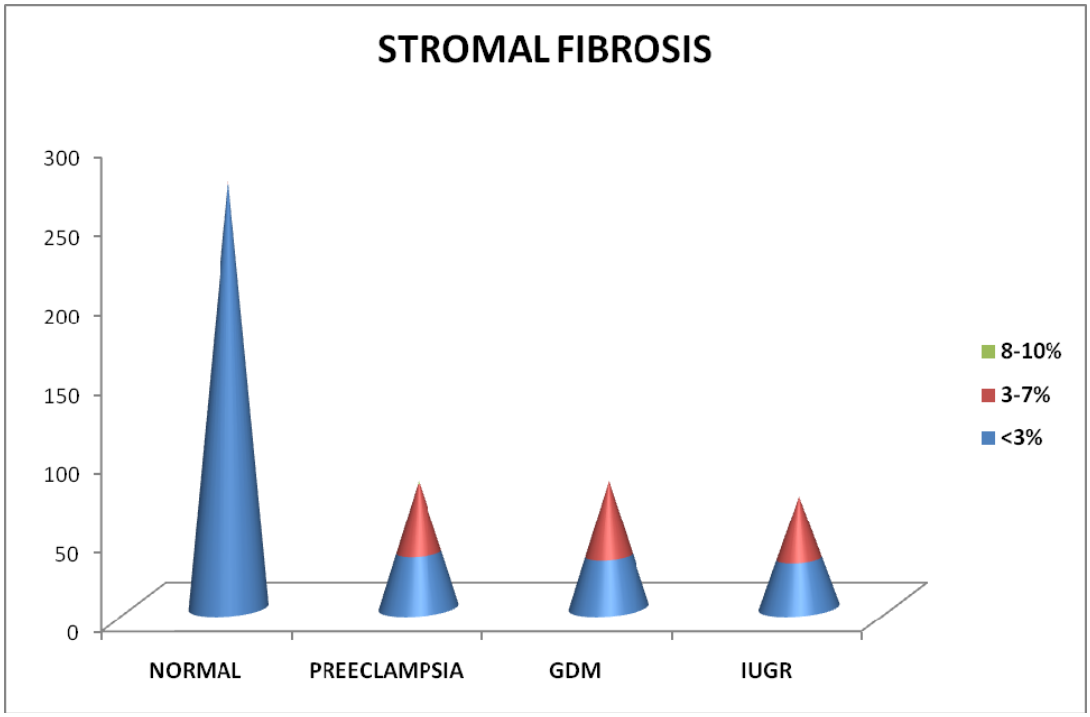
NEUTROPHILIC INFILTRATION	NORMAL	PREECLAMPSIA	GDM	IUGR
Absent	256 (85.6%)	58 (73.25%)	61 (76.25%)	67 (95.71%)
Present	14 (14.4%)	22 (26.75%)	19 (23.75%)	3 (4.29%)

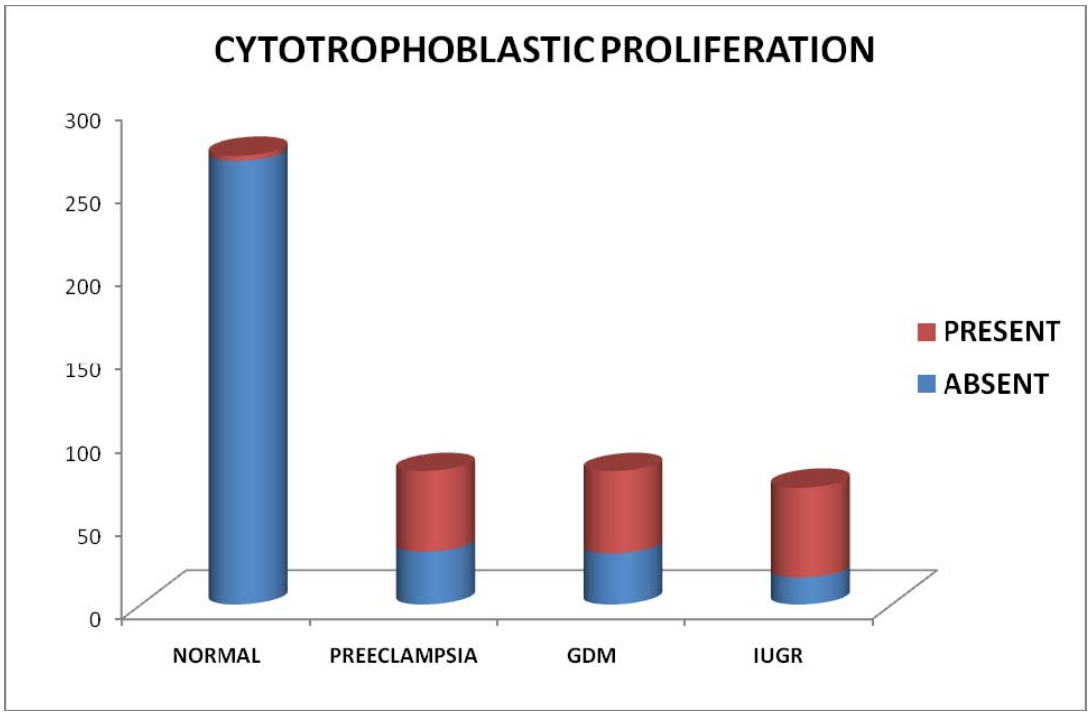
Above table shows the presence and absence of neutrophilic infiltraton in normal and high risk group.

6.6 -CALCIFICATION

CALCIFICATION	NORMAL	PREECLAMPSIA	GDM	IUGR
Absent	242 (89.8%)	6 (7.23%)	56 (70%)	15 (21.44%)
Present	28 (10.2%)	74 (92.76%)	24 (30%)	55 (78.56%)

Above table shows the presence and absence of calcification in normal and high risk group.


STROMAL FIBROSIS
300
250
200
150
100
50
0
NORMAL
PREECLAMPSIA
GDM
IUGR
8-10%
3-7%
<3%


CYTOTROPHOBLASTIC PROLIFERATION
300
250
200
150
100
50
0
NORMAL
PREECLAMPSIA
GDM
IUGR
PRESENT
ABSENT

6.7-STROMAL FIBROSIS

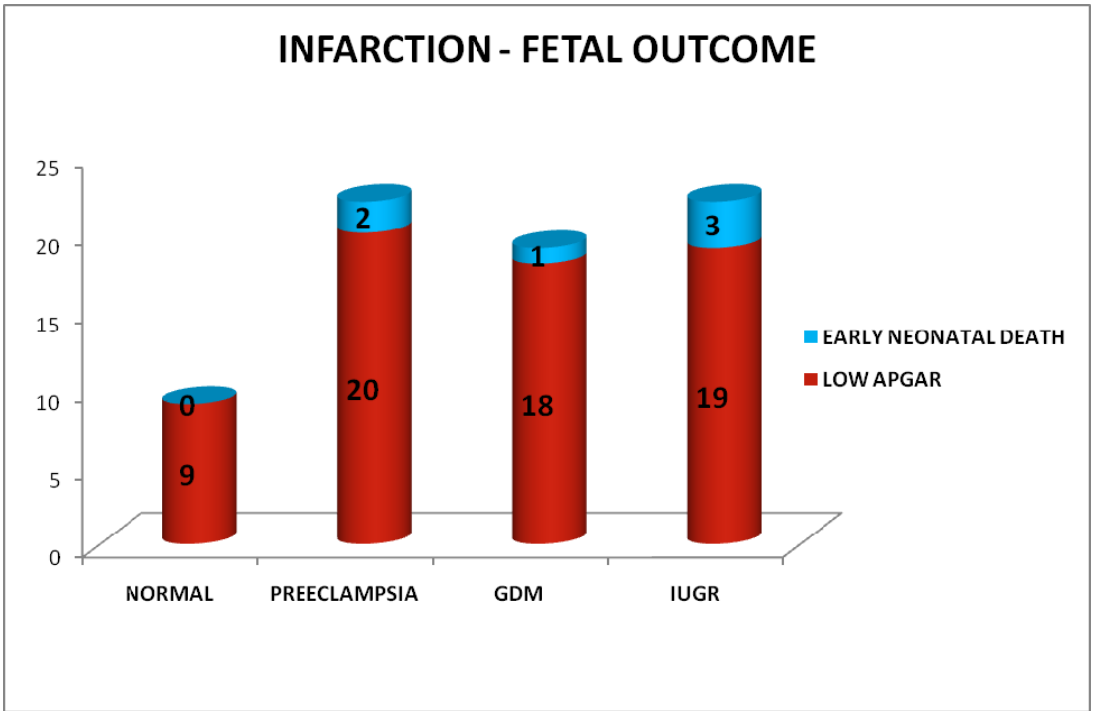
STROMAL FIBROSIS	NORMAL	PREECLAMPSIA	GDM	IUGR
I) <3%	268 (99.25%)	35 (43.75%)	33(41.25%)	31 (43.48%)
(II) 3-7% GR I	2 (0.75%)	43 (53.94%)	47(58.75%)	39 (56.52%)
(III) 8-10 GRII		2 (2.5%)		

Above table shows stromal fibrosis pattern in normal and high risk cases.

6.8-CYTOTROPHOBLASTIC PROLIFERATION

CYTOTROPHOBLASTIC PROLIFERATION	NORMAL	PREECLAMPSIA	GDM	IUGR
Absent	267(98.8%)	32 (40.78%)	31(38.75%)	16(21.73%)
Present	3 (1.2%)	48 (59.22%)	49(61.25%)	54(78.26%)

Above table shows presence and absence of cytotrophoblastic proliferation in normal and high risk groups.


INFARCTION - FETAL OUTCOME
25
20
15
10
5
0
0
9
2
20
1
18
3
19
NORMAL
PREECLAMPSIA
GDM
IUGR
EARLY NEONATAL DEATH
LOW APGAR

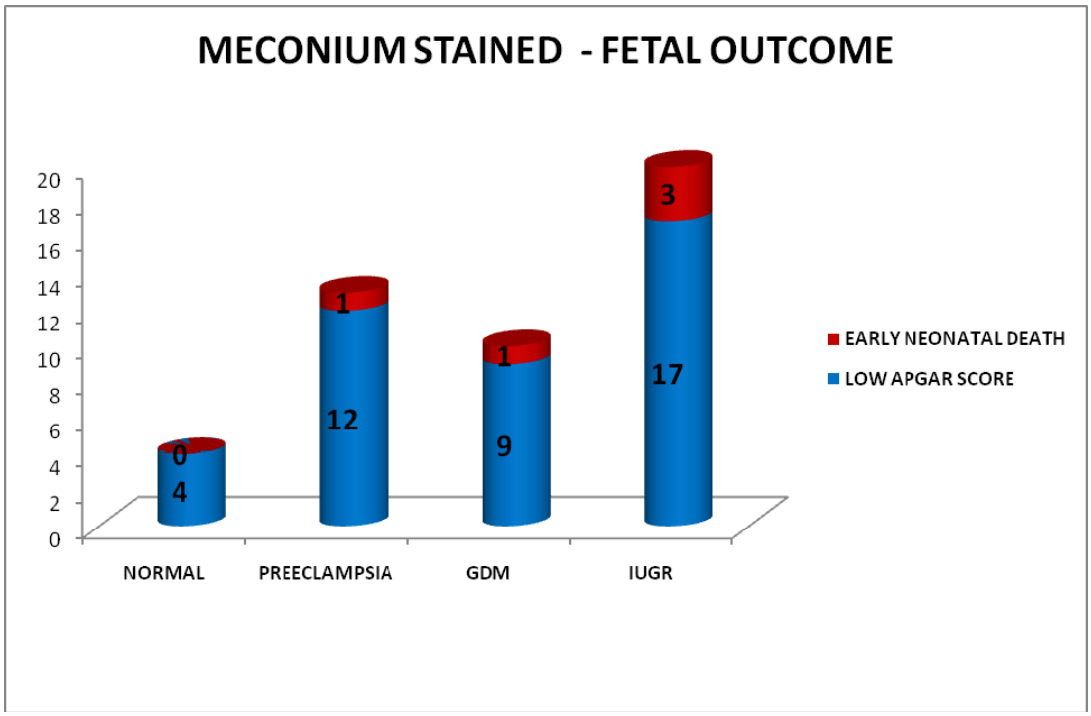

MECONIUM STAINED - FETAL OUTCOME
20
18
16
14
12
10
8
6
4
2
0
0
4
1
12
1
9
3
17
NORMAL
PREECLAMPSIA
GDM
IUGR
EARLY NEONATAL DEATH
LOW APGAR SCORE

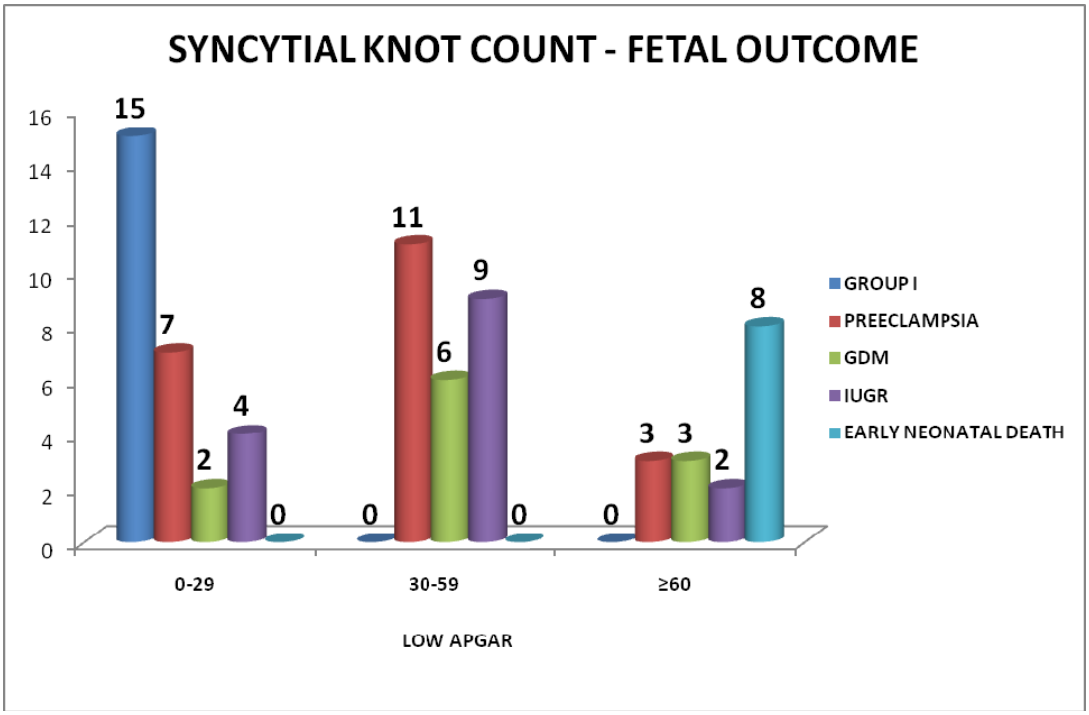
TABLE – 7

CORRELATION OF FETAL OUTCOME WITH MACROSCOPIC PLACENTAL PATHOLOGY

VARIABLES	GROUP I	GROUP II	LOW APGAR		EARLY NEONATAL DEATH		P Value
			I	II	I	II	
INFARCTION	4O	110	9(22.5%)	57(52.2%)	-	6 (5.45%)	>0.01
CALCIFICATION	34	138	5(14.7%)	14(9.9%)	-	-	>0.01
MECONIUM STAINED	22	90	4(18.1%)	38(41.89 %)	-	5 (5.55%)	>0.01
TRUE KNOT	2	2	-	1(50%)	-	-	>0.01
SEPTAL CYST	19	2	-	-	-	-	>0.01
SINGLE UMBILICAL ARTERY	-	2	-	2(100%)	-	1 (50%)	>0.01
PLACENTAL WEIGHT <350 GMS	8	109	1(12.5%)	104(96%)	-	15(14.28%)	<0.01
FP RATIO <3.5	10	107	3(30%)	102(96%)	-	17 (16%)	<0.01

GROUP I – Normal pregnancies
GROUP II – High risk pregnancies

Above table shows correlation of fetal outcomes with macroscopic placental pathology in normal and high risk groups.


SYNCYTIAL KNOT COUNT - FETAL OUTCOME
16
14
12
10
8
6
4
2
0
15
7
2
4
0
0
11
6
9
0
0
3
3
2
8
0-29
30-59
≥60
LOW APGAR
GROUP I
PREECLAMPSIA
GDM
IUGR
EARLY NEONATAL DEATH

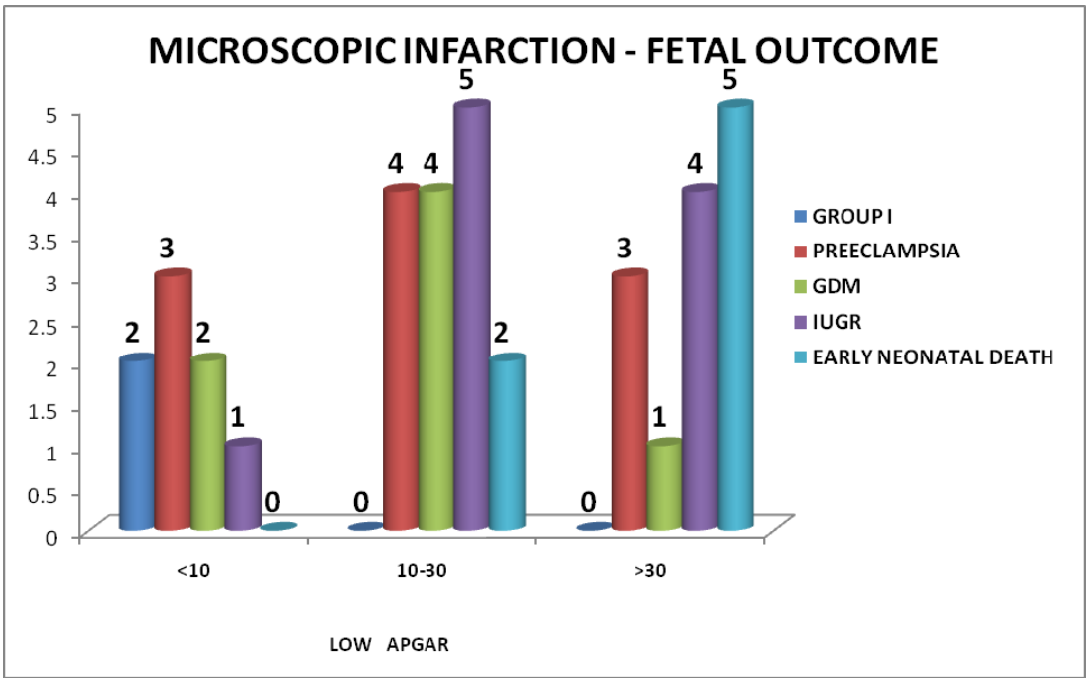

MICROSCOPIC INFARCTION - FETAL OUTCOME
5
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
2
3
2
1
0
0
4
4
5
2
0
3
1
4
5
<10
10-30
>30
LOW APGAR
GROUP I
PREECLAMPSIA
GDM
IUGR
EARLY NEONATAL DEATH

TABLE -8

CORRELATION OF FETAL OUTCOME WITH MICROSCOPIC PLACENTAL PATHOLOGY

8.1 SYNCYTIAL KNOT COUNT

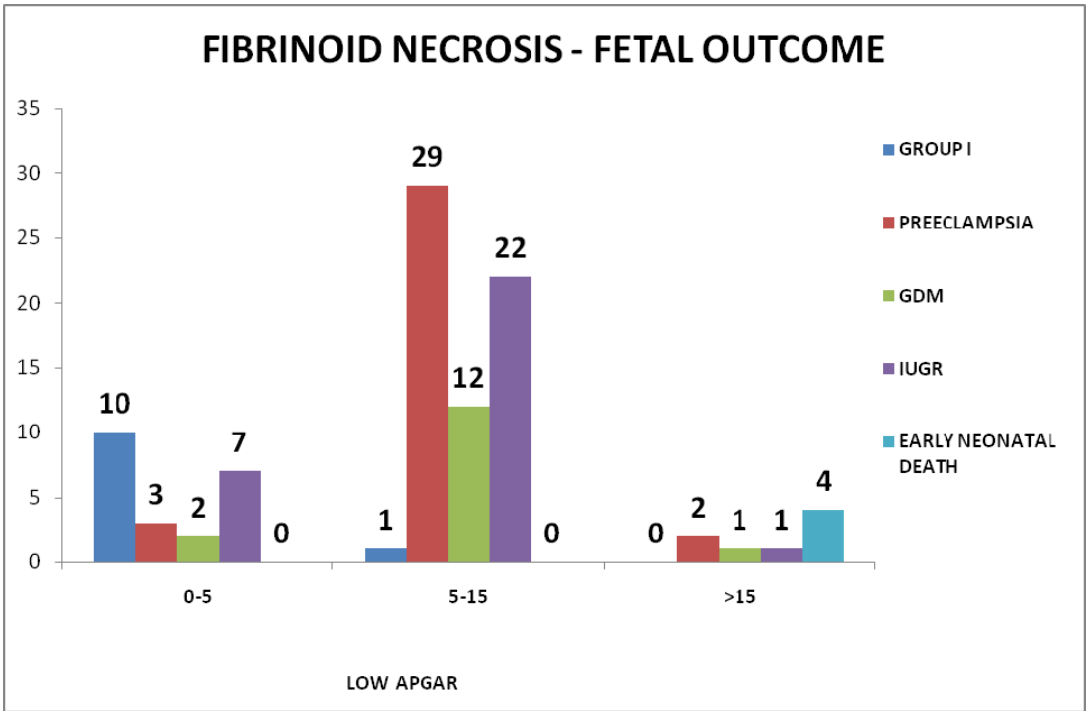
SYNCYTIAL KNOT COUNT	GROUP I	GROUP II	LOW APGAR		EARLY NEONATAL DEATH	
			I	II	I	II
I (0 -29)	268	54	15(5.9%)	13(24.74%)	-	-
II (30-59)	2	162	-	26(16.01%)	-	3(1.57%)
III (>60)		14	-	8(57.14%)	-	8(57.14%)

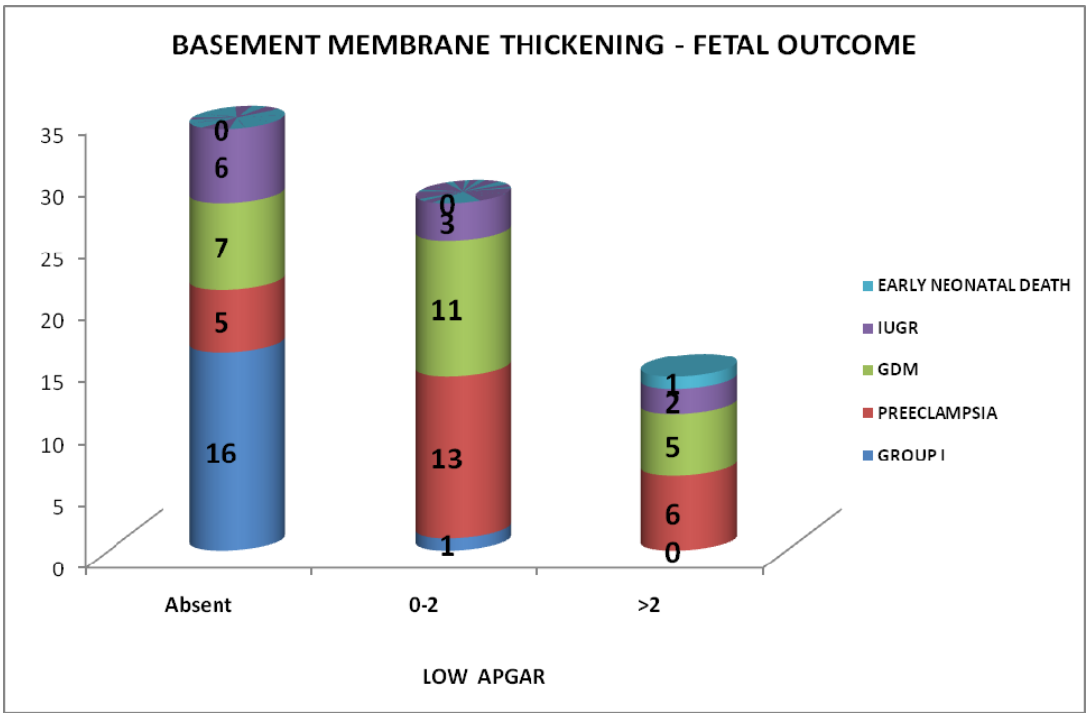
Above table shows correlation of fetal outcome with syncytial knot count in normal and high risk groups.

8.2 INFARCTION

INFARCTION	GROUP I	GROUP II	LOW APGAR		EARLY NEONATAL DEATH	
			I	II	I	II
ABSENT	231	162	-	1(0.62%)	-	-
<10%	39	32	2(5.55%)	6(18.75%)	-	-
10-30%	-	24	-	13(54.16%)	-	1(4.16%)
>30%	-	12	-	8(66.66%)	-	5(41.67%)

Above table shows correlation of fetal outcome with Infarction in normal and high riskgroups.


FIBRINOID NECROSIS - FETAL OUTCOME
35
30
25
20
15
10
5
0
10
3
2
7
0
1
29
12
22
0
0
2
1
1
4
0-5
5-15
>15
LOW APGAR
GROUP I
PREECLAMPSIA
GDM
IUGR
EARLY NEONATAL DEATH


BASEMENT MEMBRANE THICKENING - FETAL OUTCOME
35
30
25
20
15
10
5
0
0
6
7
5
16
0
3
11
13
1
1
2
5
6
0
Absent
0-2
>2
LOW APGAR
EARLY NEONATAL DEATH
IUGR
GDM
PREECLAMPSIA
GROUP I

8.3 FIBRINOID NECROSIS

FIBRINOID NECROSIS	GROUP I	GROUP II	LOW APGAR		EARLY NEONATAL DEATH	
			I	II	I	II
0-5%	267	46	5 (1.9%)	12 (26.86%)	-	-
5-15%	3	180	1(33.33%)	63 (39.97%)	-	-
>15%	-	4	-	4 (100%)	-	4 (100%)

Above table shows correlation of fetal outcome with Fibrinoid necrosis in normal and high risk groups.

8.4 BASEMENT MEMBRANE THICKENING

BASEMENT MEMBRANE THICKENING	GROUP I	GROUP II	LOW APGAR		EARLY NEONATAL DEATH	
			I	II	I	II
ABSENT	261	48	16 (6%)	18 (37.9%)	-	-
0 - 2%	9	155	1(11.11%)	27 (17.56%)	-	-
>2%	-	27	-	13 (48.14%)	-	1 (3.70%)

Above table shows correlation of fetal outcome with Basement membrane thickening in normal and high risk groups.

8.5 STROMAL FIBROSIS

STROMAL FIBROSIS	GROUP I	GROUP II	LOW APGAR		EARLY NEONATAL DEATH	
			I	II	I	II
<3%	268	99	16(6.22%)	20(20.2%)	-	-
GR I 3-7%	2	129	-	29(22.48%)	-	1(0.77%)
GR II >7%	-	2	-	2 (100%)	-	1(50%)

Above table shows correlation of fetal outcome with Stromal fibrosis in normal and high risk groups.

8.6 CYTOTROPHOBLASTIC PROLIFERATION

CYTOTROPHOBLASTIC PROLIFERATION	GROUP I	GROUP II	LOW APGAR		EARLY NEONATAL DEATH	
			I	II	I	II
ABSENT	267	79	17(6.44%)	23(29.11%)	-	-
PRESENT	3	151	-	22(14.56%)	-	2(1.32%)

Above table shows correlation of fetal outcome with Cytotrophoblastic proliferation in normal and high risk groups.

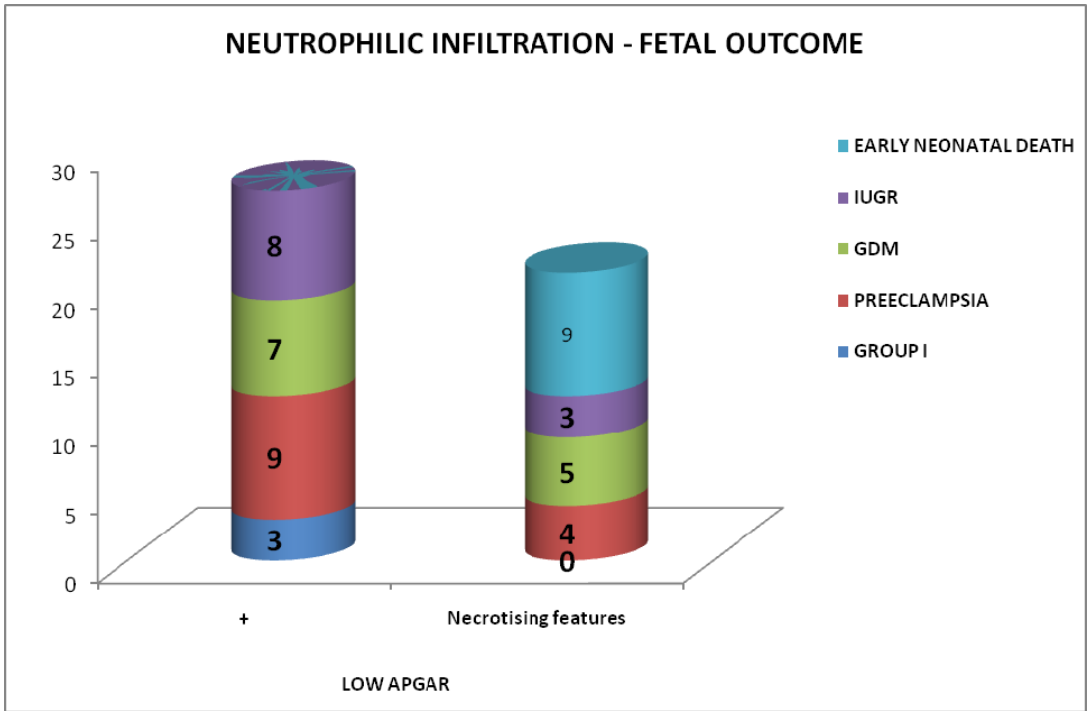

NEUTROPHILIC INFILTRATION - FETAL OUTCOME
30
25
20
15
10
5
0
8
7
9
3
9
3
5
4
0
+
Necrotising features
LOW APGAR
EARLY NEONATAL DEATH
IUGR
GDM
PREECLAMPSIA
GROUP I

TABLE – 8.7

NEUTROPHILIC INFILTRATION OF MEMBRANE / CORD

NEUTROPHILIC INFILTRATION	GROUP I	GROUP II	LOW APGAR		EARLY NEONATAL DEATH	
			I	II	I	II
ABSENT	256	174	1(0.3%)	2(1.15%)	-	-
+	14	44	3(21%)	24(54.55%)	-	-
NECROTIZING FEATURES	-	12	-	12(100%)	-	9(75%)
TOTAL	270	230				

Above table shows correlation of fetal outcome with neutrophilic infiltration of membrane / cord in normal and high risk groups.

TABLE – 9

CROSS TABULATION OF DIFFERENT VALUES BY APGAR

9.1 SYNCYTIAL KNOT COUNT

SYNCYTIAL KNOT COUNT	LOW APGAR				TOTAL	
	YES		NO			
	N	%	N	%	N	%
0-29	28	8.70%	294	91.3%	322	64.4%
30-59	26	15.8%	138	84.2%	164	32.8%
≥60	8	57.14%	6	42.86%	14	2.8%

Chi Square Value – 31.67

P value <0.001 Significant

Above table shows correlation of syncytial knot count with low apgar score.

9.2 INFARCTION

INFARCTION	LOW APGAR				TOTAL	
	YES		NO			
	N	%	N	%	N	%
ABSENT	1	0.25%	392	99.75%	393	78.6%
<10	8	11.26	63	88.74%	71	14.2%
10-30	13	54.16%	11	43.84%	24	4.8%
>30	8	66.66%	4	33.34%	12	2.4%

Chi Square Value – 27.42

P value <0.001 Significant

Above table shows correlation of infarction with low apgar score.

9.3-FIBRINOID NECROSIS

FIBRINOID NECROSIS	LOW APGAR				TOTAL	
	YES		NO			
	N	%	N	%	N	%
0-5%	17	5.43%	296	94.57%	313	62.6%
5-15%	64	35%	119	65%	183	36.6%
>15%	4	100%	0	0%	4	0.8%
						500

Chi Square Value – 91.11

P value <0.001 Significant

Above table shows correlation of low APGAR score with fibrinoid necrosis.

9.4- THICKENED BASEMENT MEMBRANE

THICKENED BASEMENT MEMBRANE	LOW APGAR				TOTAL	
	YES		NO			
	N	%	N	%	N	%
ABSENT	84	11%	275	89%	309	61.8%
0-2%	28	17.07%	136	82.93%	164	32.8%
>2%	13	48.15%	14	51.85%	27	5.4%
						500

Chi Square Value – 27.71

P value <0.001 Significant

Above table shows correlation of low APGAR score with basement membrane thickening.

TABLE – 9.5

NEUTROPHILIC INFILTRATION

NEUTROPHILIC INFILTRATION	LOW APGAR				TOTAL	
	YES		NO			
	N	%	N	%	N	%
ABSENT	3	0.7%	427	99.3%	430	86%
+	27	46.55%	31	53.45%	58	11.6%
NECROTISING FEATURE	12	100%	0	0%	12	2.4%
						500

Chi Square Value – 9.45

P value <0.02 Significant

Above table shows correlation of low APGAR score with neutrophilic infiltration of membrane/ cord.(p value < 0.01 is considered significant)

TABLE – 10

CROSS TABULATION OF DIFFERENT VALUES BY EARLY NEONATAL DEATH

10.1 SYNCYTIAL KNOT COUNT

SYNCYTIAL KNOT COUNT	EARLY NEONATAL DEATH				TOTAL	
	YES		NO			
	N	%	N	%	N	%
0-29%	0	0%	322	100%	322	64.4%
30-59%	3	1.8%	161	98.2%	164	32.8%
≥60	8	57.14%	6	42.86%	14	2.8%
						500

Chi Square Value – 203.77
P value <0.001 Significant

Above table shows correlation of early neonatal death with syncytial knot count.

10.2 INFARCTION

INFARCTION	EARLY NEONATAL DEATH				TOTAL	
	YES		NO			
	N	%	N	%	N	%
ABSENT	0	0%	393	100%	393	78.6%
<10	0	0%	71	100%	71	14.2%
10-30	1	4.17%	23	95.83%	24	4.8%
>30	5	41.67%	7	58.33%	12	2.4%
						500

Chi Square Value – 32.83
P value <0.001 Significant

Above table shows correlation of early neonatal death with infarction.

10.3 FIBRINOID NECROSIS

FIBRINOID NECROSIS	EARLY NEONATAL DEATH				TOTAL	
	YES		NO			
	N	%	N	%	N	%
0-5%	0	0%	313	100%	313	62.6%
5-15%	0	0%	183	100%	183	36.6%
>15%	4	100%	0	0%	4	0.8%
						500

Chi Square Value – 230

P value <0.001 Significant

Above table shows correlation of early neonatal death with fibrinoid necrosis.

10.4 BASEMENT MEMBRANE THICKENING

BASEMENT MEMBRANE THICKENING	EARLY NEONATAL DEATH				TOTAL	
	YES		NO			
	N	%	N	%	N	%
ABSENT	0	0%	309	100%	309	61.8%
0-2%	0	0%	164	100%	164	32.8%
>2%	1	3.7%	26	96.3%	27	5.4%
						500

Chi Square Value – 7.55

P value <0.023 Significant

Above table shows correlation of early neonatal death with basement membrane Thickening.

TABLE -10.5

NEUTROPHILIC INFILTRATION

NEUTROPHILIC INFILTRATION	EARLY NEONATAL DEATH				TOTAL	
	YES		NO			
	N	%	N	%	N	%
ABSENT	0	0%	430	100%	430	86%
+	0	0%	58	100%	58	11.6%
NECROTISING FEATURES	9	75%	3	25%	12	2.4%
						500

Chi Square Value – 33.96

P value <0.001 Significant

Above table shows correlation of early neonatal death with neutrophilic infiltration.

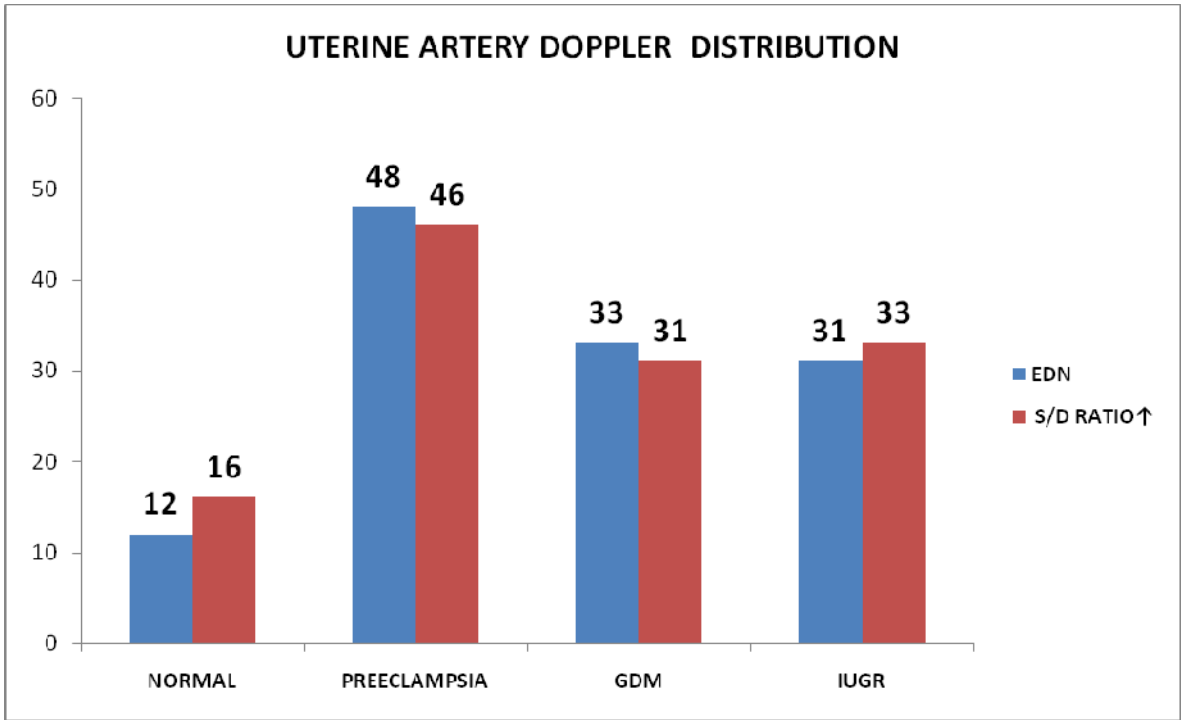

UTERINE ARTERY DOPPLER DISTRIBUTION
60
50
40
30
20
10
0
12
16
48
46
33
31
31
33
NORMAL
PREECLAMPSIA
GDM
IUGR
EDN
S/D RATIO↑

TABLE – 11

GROUPWISE DISTRIBUTION OF UTERINE ARTERY DOPPLER

UTERINE ARTERY DOPPLER		GROUP							
		NORMAL		PREECLAMPSIA		GDM		IUGR	
		N	%	N	%	N	%	N	%
EARLY DIASTOLIC NOTCH	NO	258	95.56%	32	40%	47	58.75	39	55.71%
	YES	12	4.44%	48	60%	33	41.25%	31	44.29%
SYSTOLIC DIASTOLIC RATIO ↑	NO	254	94.07%	34	42.5%	49	61.25%	37	52.85%
	YES	16	5.93%	43	57.5%	31	38.75%	33	47.15%
TOTAL		270		80		80		70	

CHI SQUARE VALUE

EARLY DIASTOLIC NOTCH - 139.0

↑ SYSTOLIC DIASTOLIC RATIO – 118.61

P VALUE

EARLY DIASTOLIC NOTCH - <0.001 Significant

↑ SYSTOLIC DIASTOLIC RATIO - <0.001 Significant

Above table shows the groupwise distribution of uterine artery Doppler findings.

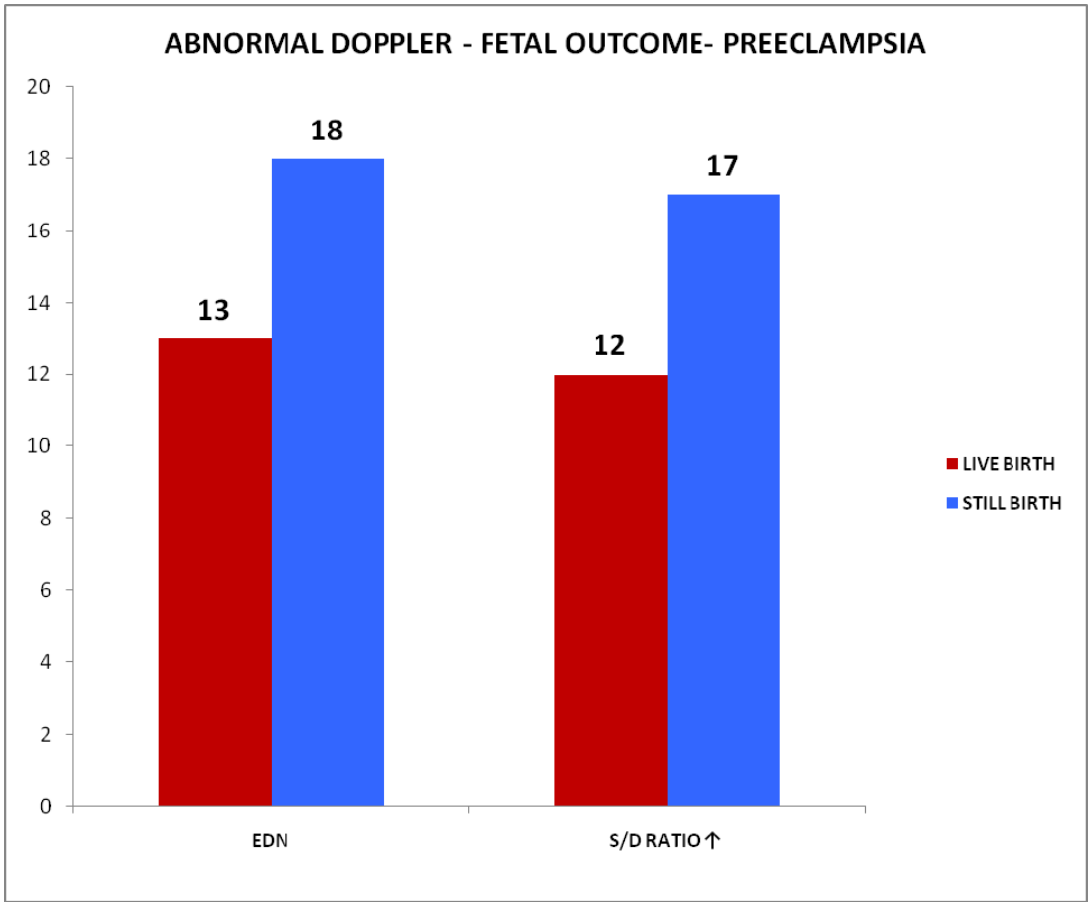

ABNORMAL DOPPLER - FETAL OUTCOME- PREECLAMPSIA
20
18
16
14
12
10
8
6
4
2
0
18
17
13
12
EDN
S/D RATIO↑
LIVE BIRTH
STILL BIRTH

TABLE – 12

ASSOCIATION BETWEEN FETAL OUTCOME AND UTERINE ARTERY DOPPLER IN PREECLAMPSIA GROUP

UTERINE ARTERY DOPPLER		FETAL OUTCOME				TOTAL		CHI SQUARE VALUE	P VALUE
		LIVE BIRTH		STILL BIRTH					
		N	%	N	%	N	%		
EARLY DIASTOLIC NOTCH	NO	48	97.9%	1	2.1%	49	61.25%	29.89	<0.001
	YES	13	41.9%	18	58.1%	31	38.75%		
SYSTOLIC DIASTOLIC RATIO↑	NO	49	96.07%	2	3.93%	51	63.75%	27.60	<0.001
	YES	12	41.4%	17	58.6%	29	36.25%		
TOTAL		61		19		80			

P VALUE <0.001 Significant

Above table shows the association between uterine artery Doppler and fetal outcome in Preeclampsia group.

TABLE – 13

ASSOCIATION BETWEEN APGAR AND UTERINE ARTERY DOPPLER IN PREECLAMPSIA GROUP

UTERINE ARTERY DOPPLER		APGAR SCORE				TOTAL		CHI SQUARE VALUE	P VALUE
		NORMAL		LOW					
		N	%	N	%	N	%		
EARLY DIASTOLIC NOTCH	NO	32	53.33%	4	20%	36	44.5%	6.73	<0.09
	YES	28	46.67%	16	80%	44	55.5%		
SYSTOLIC DIASTOLIC RATIO ↑	NO	31	51.67%	3	15%	34	42.5%	8.65	0.04
	YES	29	48.33%	17	85%	46	57.5%		
TOTAL		60		20		80	100%		

P VALUE

EARLY DIASTOLIC NOTCH - <0.09 Significant

↑ SYSTOLIC DIASTOLIC RATIO – 0.04 Borderline significant

Above table shows the association between uterine artery Doppler and APGAR score in Preeclampsia group.

DISCUSSION

DISCUSSION

Obstetric endorsement of the utility of placental histologic examination is commonly lukewarm, especially from obstetricians who do not have a placental pathologist as part of their own local clinical care team.Placental pathologic examinations are pointless if they donot provide clinically useful data.

One important limitation of placental examination is the inability to make a one to one link between any one placental or uteroplacental vascular lesion and a particular maternal or fetal, neonatal problem. This would be an unrealistic goal, because in no other organs are such relationship the rule. Neither hepatocellular necrosis nor glomerulosclerosis for example, is diagnostic of one and only one hepatic or renal disease; in those diseases individual lesions are considered as part of a greater histopathologic pattern. Finally the pattern is correlated with a variety of clinical data, laboratory data, and additional pathologic studies to produce the final clinical pathologic diagnosis.

Obstetric technologies and therapies are also directed towards general pathophysiologic process (such as uterine and uteroplacental Doppler velocimetry, anticoagulant therapy and maternal immunization and intravenous gamma globulin) rather than specific lesions or tissue diagnoses.

Table-1 shows distribution of cases in our study. Out of 500 cases, 270 cases were term uncomplicated pregnancies. Remaining 230 cases were pregnancies complicated by various disorders.**(80-preeclampsia,80-GDM,70-IUGR).**

Table-2 shows the distribution for age in both groups. Age ranged between 21-35 yrs in both group of patients. Most of the patients fall between 26-29yrs. 62.6% in normal group and 54.78% in high risk group.

Table-3 shows gross features of placenta in both groups. The placenta of group II was associated with low placental weight and low fetal placental ratios. In the clinicopathological study by **NAEYE (1987)** low placental weight was associated with maternal uteroplacental vascular insufficiency.

In our study, five cases of GDM had placental weight of 600gms, and one case of GDM had placental weight of 700gms, which showed extensive villous edema on histopathological examination. In our study, abnormalities of maternal and fetal surface of placenta, and cord insertion is given below:

Battledore placenta	-	nine cases
Velamentous insertion of cord	-	11 cases
Circumvallate placenta	-	four cases
Bilobed placenta	-	three cases

Cord Length

The average cord length was within normal limits in both groups.

Cord attachment

In normal group, cord attachment was central in 48.14%, paracentral in 46.29% and marginal in 5.5%. In high risk group the same being 52.17%,, 43.47%, 4.3% respectively.

In our study, there was no adverse perinatal outcome in fetuses with marginal cord insertion. **Rashmi and Rangekhar (1993)** quotes 72% incidence of paracentral cord attachment in normal singleton fetuses.

Infarction

Table-4, shows the macroscopic abnormalities in both normal and high risk groups. In our study, placental infarcts are seen in 14.8% of normal placenta, 57.5% of preeclampsia, 43.75% of GDM, 41.42% of IUGR.

In normal pregnancy,infarct size was <2cm. In high risk cases, the size was 2.5-3.5cm. The size was >5cm in 13 cases. (five cases of preeclampsia, three cases of GDM, five cases of IUGR) and were placed centrally.

25% of term uncomplicated pregnancies were associated with infarcts in placenta and usually situated in periphery and involve <5% of placental area. **Fox 1979** states that extensive infarction involving 10% or more of parenchyma is accompanied by a high incidence of fetal hypoxia, growth retardation and intrauterine death.

Calcification

Seen in 12.6% of normal term pregnancies, 63.75% of preeclampsia cases, 55% of GDM, 61.42% of IUGR.

There is no association between fetal hypoxia, low birth weight or IUD and calcification of placenta according to **TINDAL SCOTTIS (1966)** in their study of placenta of 3026 singleton pregnancies.

Meconium Stained Membranes

Occurred in 47.14% of IUGR, 43.75% of preeclampsia, 22% of GDM, and only in 8% of normal pregnancies**. Altshuler, G.Scott** reported meconium staining in 27% consecutively related placenta from at risk singleton newborn.

True knot

Noted only in two cases of normal, one case of preeclampsia, one case of IUGR. True knots incidence varies 0.4-1% of all deliveries. Knots in the cord are associated with perinatal mortality of 8-11% according to **Fox (1979).**

Single umbilical artery

Was noted in two cases of GDM. One baby had cardiac anomaly – Transposition of great vessels. Another baby had duodenal atresia.

Septal cyst

In our study septal cyst was noted in 19 cases of normal placenta and one case of GDM, one case of IUGR. **Fox (1978**) observed that septal cysts are seen in 7-11% of term uncomplicated pregnancies.

Table -5 shows the histopathological features of basal plate or decidua.

Neutrophilic infiltration

Neutrophilic infiltration is seen in 9.62% of normal placenta, 21% of preeclampsia, 5% of GDM, 7% of IUGR placenta. Microabscess is present in one case of normal placenta,,two cases of GDM,three cases of preeclampsia, three cases of IUGR.

Infarction in basal plate

Infarction is seen in 4.4% of normal placenta, whereas it is seen in 27% of preeclampsia, 25% of GDM, 43% of IUGR.

Of these, in four cases of preeclampsia,,five cases of IUGR, two cases of GDM, infarction was present covering >30% of placental surface, indicating compromised uteroplacental perfusion.

Fibrinoid change in vessels

Present in 2.4% of normal cases, 69% of preeclampsia, 10% of GDM, 87% of IUGR. According to **Fox (1968)** it represents degenerative change.

Table -6 shows histopathological features of villi in normal and high risk groups.

1. Basement Membrane Thickening

As shown in Table – 6.1 In complicated groups, basement membrane thickening of 0-2% was observed in 65% of cases, whereas in normal group, thickening is absent in 96.7% of cases. This is a result of ischaemia. Thickening of basement membrane normally occurs in 3% of villi at term according to **Fox (1978).** It is best shown by PAS staining.

2. Syncytical knot count:

According to **Benerische (1961**) and **Fox (1978)** syncytial knots are primary responses to hypoxia, being homeostatic response to hypoxemic stress. Syncytial knots are present in 10-30% of villi at term in uncomplicated pregnancies.As shown in Table – 6.2 In our study, in normal cases – the count was found to range between 0-29% constituting about 99.25% of normal placenta. Nearly 60-70% of cases of high risk group showed syncytial knot count in the range of 30-59% (preeclampsia – 73.36%, IUGR – 65.21%, GDM 70.69%). Four cases of preeclampsia, seven cases of GDM, three cases of IUGR showed syncytial knot count $\geq$60%.

3. Fibrinoid Necrosis:

As shown in Table 6.3 the count was between 0-5% in 99.2% of normal cases. The range was 5-15% in 70-80% of high risk cases indicating degenerative change (preeclampsia-79.6% GDM-71% IUGR, 85.71%).

4. Infarction

As shows in Table 6.4 it is present in 30% of preeclampsia, 23.75% of GDM, 35.7% of IUGR. Five cases of preeclampsia, one case of GDM, six cases of IUGR showed involvement of >30% area.

5. Neutrophilic Infiltration

As shows in Table 6.5, Neutrophilic infiltration is present in 26.75% of preeclampsia, 23.75% of GDM, 5% of normal cases. Necrotising features are noted in four cases of preeclampsia, five cases of GDM, three cases of IUGR.

6. Calcification

As shows in Table – 6.6., calcification is present in 10% of normal placenta. Calcification is a highly obscure association with no serious abnormality of placenta according to **Fox.**

7. Stromal fibrosis

As shows in Table -6.7, In our study, grade I lesions were present in complicated (50%) groups. In normal group, stromal fibrosis was present only in <3 of villi. Ageing process and reduced uteroplacental blood flow being the prime factors.

8. Cytotrophoblastic Proliferation

As shows in Table 6.8 it is absent in 98.8% of normal placenta, and present in high risk pregnancies, in various degrees. 59.22% of preeclampsia, 61% of GDM, 78.26% of IUGR. Degree of cytotrophoblastic proliferation is related to the extent of syncytial damage.

Blanc scoring system

To grade inflammation severity of extra placental membrane surface.

Grade 1: Polymorphs confined to deep connective tissue above chorionic plate

Grade 2: Amniotic and chorionic surface

Grade 3: Necrotising choriomnionitis

Umbilical cord funisitis

Grade 1: Focally present

Grade 2: Diffusely present

Grade 3: Necrotizing

As seen in Table-7 there is a significant rise in the low APGAR percentage in most of the morphological changes of placenta in the high risk group.

In infarction, low APGAR percentage is 52.2% in high risk group compared to 22.5% in normal pregnancies. In meconium staining of placenta, low APGAR percentage in group II is 41.89% compared to 18.1% in group I. Nearly 96% with placental weight <350gms and fetoplacental ratio <3.5 in group II had low APGAR.

In calcification, low APGAR percentage is nearly equal in both groups, with no early neonatal death. Early neonatal death was observed in six cases with infarction, noted in preeclampsia. Early neonatal death was observed in five cases of meconium stained membranes. Early neonatal death was observed in nearly 14.28% (15cases) with placental weight <350gm, fetoplacental ratio <3.5-17 cases of early neonatal death, all due to preeclampsia.

As seen in Table -8 there is a significant rise in the low APGAR percentage in the group of cases with higher grade histological changes of placenta. This correlation is seen more with high risk pregnancies (Group II).

Syncytial knot count

As seen in Table-8.1, higher grade syncytial knot count (II,III) are seen mainly in group II pregnancies. 57.14% of fetuses with knot count of ≥60, gr.III, had low APGAR and early neonatal death. Among eight cases of early neonatal death, three cases were due to preeclampsia, two cases due to IUGR, three cases were due to GDM.

Infarction

As seen in Table 8.2 higher grades of infarction are noted only in group-II pregnancies. In infarction covering >30% area, low APGAR and early neonatal death were observed in 66.66% and 41.67% (five cases) respectively.

Low APGAR was observed in 54% of cases with infarction of 10-30% and 4% had early neonatal death. All the six cases of death were due to preeclampsia.

Fibrinoid Necrosis

As seen in Table -8.3, fibrinoid necrosis of >15% involvement, seen in group II nearly 100% (four cases) had low APGAR and 100% (four cases) had early neonatal death. Nearly 39% of group II pregnancies had low APGAR with involvement of 5-15% area. Of the four cases of early neonatal death seen in >15% involvement, three cases were due to preeclampsia, one case of death was due to IUGR.

Thickened Basement Membrance

As seen in Table -8.4, >2% involvement of basement membrane is noted only in group II pregnancies which revealed low APGAR in nearly 48% of pregnancies.

Stromal Fibrosis

As shown in Table -8.5, grade II stromal fibrosis noted in group II was associated with low APGAR in two cases (100%) and early neonatal death (50%) in one case.Death is due to preeclampsia.

Cytotrophoblastic proliferation

As shown in Table -8.6, cytotrophoblastic proliferation observed mainly in group II pregnancies, is associated with low APGAR in 15% of cases and early neonatal death in two cases (1.32%) due to preeclampsia.

Neutrophilic Infiltration

As shown in Table-8.7, presence of neutrophilic infiltration is associated with low APGAR in 54.55% of cases. Necrotizing features observed in 12 cases of group II (four cases of preeclampsia, five cases of GDM, three cases of IUGR) had low APGAR in 100% and early neonatal death in nearly 75% (nine cases).

As shown in Table -9.1, there is significant correlation with syncytial knot count and low APGAR by chisquare test $P<0.001$.

As shown in Table -9.2, there is significant correlation between infarction and low APGAR by chisquare test $P<0.001$.

As shown in Table 9.3, there is significant correlation between fibrinoid necrosis and low APGAR ($P<0.001$)

As shown in Table -9.4, there is significant correlation between thickened basement membrane with low APGAR. ($P<0.001$)

As shown in Table -9.5, there is significant correlation between necrotizing features with low APGAR. In this chisquare test with Yates correction is used. ($P<0.002$).

As shown in Table - 10.1, there is significant correlation with syncytial knot count with early neonatal death, more in higher grade. ($P<0.001$).

As shown in Table - 10.2, there is significant correlation with infarction with early neonatal death. P value (<0.001) more in higher grades.

As shown in Table - 10.3, there is significant correlation with fibrinoid necrosis with early neonatal death, more in higher grades. ($P<0.001$).

As shown in Table - 10.4, there is significant correlation between thickened basement membrane with early neonatal death, P value <0.001 more in higher grades.

As shown in Table - 10.5, there is significant correlation between necrotizing features and early neonatal death. $P<0.001$.

According to **Daskalakis G et al**,2008,there is significant correlation between fibrinoid necrosis,(also other vascular lesions) and poor fetal outcome,in diabetes group.

Aordema M W et al 2001,found “Pathological Changes” in 58% of complicated pregnancies (Placenta 22: 405,2001)

As shown in **Table-11** Abnormal Uterine artery Doppler findings are seen in 4% of normal group,60% Hypertensive group,40% of GDM grup,46% IUGR group. There is significant association between abnormal uterine artery Doppler waveforms and high risk pregnancies.($P<0.001$ for early diastolic notch and increased systolic diastolic ratio).

According to **Ambreen Qureshi et al,**,**Swarnalata Samal et al,2009,**Abnormal uterine artery Doppler findings were reported in 55%.Hypertensive,and 4% normotensive group. Our study also shows similar results.

According to **Oztelein K,Ozdemir N et al,1999**,The mean systolic diastolic ratio,resistance index,and pulsatility index of uterine artery in women with Preeclampsia and IUGR was significantly higher than women with normal pregnancies($P<0.01$)

Sagol S,Ozkinay E et al 1999 High Uterine artery flow resistance is related to reduced trophoblast migration in to the myometrium and inadequate physiological changes in the spiral arteries in women with Preeclampsia and IUGR.

As shown in Table – 12, stillbirth rate in abnormal uterine artery Doppler group in Preeclampsia is **58%.** (58.1% in early diastolic notch, 58.6% in increased systolic diastolic ratio group). There is significant association between early diastolic notch and stillbirth in preeclampsia ($P < 0.001$). There is significant association between increased systolic diastolic ratio and stillbirth in preeclampsia ($P < 0.001$).

As shown in Table – 13, in Preeclampsia group, low APGAR is seen in 85% of those with early diastolic notch and 85% of those with increased systolic diastolic ratio. There is significant association between early diastolic notch and low APGAR in Preeclampsia $P<0.09$ (<0.05 is considered significant). There is borderline significant association between low APGAR and increased systolic diastolic ratio P - 0.04.

According to **KOFINAS et al 1992,** there was significant association between low APGAR status and abnormal uterine artery Doppler waveforms.($P<0.001$)

According to **SL COSTA et al,2008** there was significant correlation between low APGAR status and abnormal uterine artery waveforms.($P<0.025$)

Out of the 20 deaths observed, one was due to congenital anomalies which died within 48hours. It has single umbilical artery.

The remaining 19 were observed between 5^{th} and 8^{th} day.

six on 5^{th} day

seven on 6^{th} day

two on 7^{th} day

four on 8^{th} day

nine died due to respiratory distress, in preeclampsia

five died due to meconium aspiration, seen in preeclampsia

six died due to septicemia seen in GDM

All these early neonatal deaths were associated with significant morphological and histopathological changes in placenta. Hence routine histomorphological study of placenta will be of immense use in reducing neonatal morbidity or mortality.

SUMMARY

SUMMARY

This study is important by 3 factors

1. Volume-500 cases of normal and high risk pregnancies
2. Armamentarium – a tertiary hospital with hitech gadgets meeting all needs
3. Variety – both morphological and histopathological study.

Both positive and negative correlations are brought out.

The positive correlations are for syncytial knot count, neutrophilic, infiltration, infarction, thickening of basement membrane, fibrinoid necrosis with fetal outcome.

There is significant correlation between the presence of syncytial knot count with low APGAR and early neonatal death respectively, when the syncytial knot count is present in >60% of villi.

There is significant correlation with infarction and low APGAR and early neonatal death.There is very significant correlation between fibrinoid necrosis and thickened basement membrane with low APGAR and early neonatal death

Finally there is significant correlation with neutrophilic infiltration with necrotizing features with low APGAR. There is significant correlation between fetal placental ratio and low APGAR and early neonatal death. $p<0.01$.

These morphological and histological changes of placenta and its relation to fetal morbidity and mortality is more pronounced in high risk pregnancies.

There is significant association between abnormal uterine artery waveforms and high risk pregnancies .There is significant correlation between Uterine artery Doppler early diastolic notch and low APGAR in preeclampsia. There is borderline association between Uterine artery Doppler, elevated systolic diastolic ratio and low APGAR in preeclampsia.

There is significant association between abnormal uterine artery Doppler waveforms and stillbirth in preeclampsia.

Gross features – there was no significant correlation with calcification, other morphological features such as both the type of placenta, septal cyst, true knot, , with fetal outcome.($P > 0.01$) In our study only two cases had single umbilical artery, among which one died due to cardiac anomaly.

Neonatal mortality was observed on the later part of 1st week of delivery in cases where we see such histomorphological changes of placenta. The morbidity and mortality can be reduced by initiating judicial better neonatal care and better antibiotic coverage once we see those histomorphological changes, not waiting for the baby to exhibit symptoms or signs. Hence the present study is important as on application, it can reduce neonatal morbidity and mortality.

CONCLUSION

CONCLUSION

This study is an endeavour to assess the possible probable link of morphological and histopathological changes in placenta and the fetal outcome. Many such studies pile up, days may not be for off when a definite link can be proved in a very positive way. This will help to save many babies.

Through examination of the delivered placenta can provide a better picture of the intrauterine environment of the fetoplacental unit than a placental bed biopsy. The ability to routinely identify failure of uteroplacental vascular adaptation, fibrinoid necrosis, atherosis, persistence of endovascular trophoblasts, thrombosis and chronic vasculitis in the basal plate can clarify the nature and mechanisms involved in pregnancy compromise.

Many studies are upcoming for placenta screening for high risk pregnancies by using a combination of ultrasound and blood tests to screen high risk pregnant mothers for placental damage. By completing these non invasive tests, most high risk mothers can be reassured that their placenta is formed and functioning properly, so they can expect a healthy pregnancy. This is an important first step in identifying placental abnormalities in early pregnancy, at a time when a number of interventions can be used to improve outcomes for those with the highest risk. This study will lead the way for future research in placenta screening and help us provide quality care for all mothers.

BIBLIOGRAPHY

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Aardema mw, Oosternof H, Timmer Aet al; Uterine artery Doppler flow and uteroplacental vascular pathology in normal pregnancies and pregnancies complicated by pre-eclampsia and small for gestational age fetuses. Placenta 22:405,2001.

Aladjern, S.Perri and Fanaooff, A. Placental score and neonatal outcome. Obstet Gynecol. 39 : 591 ; 1982.

Altshuler, G. The role of placenta in perinatal paediatric pathology, 6207 -233 ; 1996.

Altshuler, G. The epidemiology of placental features, association with gestational age and neonatal outcome. Obstet and Gynaecol. 87 : 771 - 778 ; 1991.

Altshuler, G. Choriangiosis an important placental sign of neonatal morbidity and mortality, Arch.Pathol.Lab medicine, 108 : 71 - 74 ; 1984.

Autio - Harmainen, H. A morphometric study of placenta in fetal congenital nephrotic syndrome.

Bain, AD. Newborn after prolonged leakage of liquor amnii. Br. Med. J. 2 : 598 ; 1968.

Bartman, J. and Driscoll, SG. Amnion nodosum and hypoplastic cystic kidneys. Obstet. Gynecol, 32 : 700 ; 1968.

Benirschke, K. and Kaufmann, The pathology of maternal floor infarction. In pathology of human placenta. New York NY - Springier verlage Inc - 406 : 411 ; 1990.

Benirschke, K. and Kaufmann, P. Pathology of human placenta 3rd edition. Springier Verlag New York 1995.

Chrolyn and Chandler. Placental pathology in perinatal diagnosis in Gynecology and Obstetrics. J.B. Lippin cott. company. Philadelphia. E/dy^r John J. MD.Phd., Vol. 31 Ch : 106 1 - 39 ; 1992.

Caspi Soloman, F. Amnionitis and T strainmycoplasma. Am.J.Obstet Gynecol. Ill : 1102 ; 1971.

Cynthia, G., Kaplan. Carolyn Salafia - College of American Pathologists. conference XIX on examination of placenta. Report of working group on the def of structural changes associated with abnormal functions in maternal / fetal placental unit in 2nd and 3rd trimester. Arch.Pathol.Lab. med. 115 : 709 - 716 ; 1991.

Driscoll, SG. Pathology of placenta in intrauterine growth retardation. Ann. Chir, Gynaecol., 70 : 316 ; 1987.

Daskalakis G, marinapoulas, Krielesiv, PapapanagiotouA, placental pathology in women with gestational diabetes. Acta Gynecol Scand 2008; 87(4) 403-7.

Ethches, PC. Stewart. The relationship between disorders of umbilical cord and intrauterine growth retardation. Acta. Obstot, Gynecol scand. 72 ; 15 ; 1982.

Fox, H. Pathology of placenta, London. WB Souder Ltd., 1978.

Fox, H. Thrombosis of fetal arteries in human placenta, BJOG. 73 : 961 -965 ; 1966.

Fox, H. General pathology of placenta in Fox, H. ed : Obstetrical and Gynecological pathology New York NY: Churchill Linngstone, Inc Z: 972 : 1000 ; 1987.

Geoffrey Altshuler. Scott - R - clinicopathological implications of placental pathology clinical obstet and Gynaecol, 29 : 549 - 570 ; 1996.

Gruenwal, P (Ed). The placenta and its maternal supply Line. Lancaster, medical and technical publishing, 1975.

Hans.G.Kohler Pathology of Umbilical cord and fetal membranes in Hainee M.Obstetrical and gynecological. Path Vol2.Pages 1079-1116. 1987.

Haust, MD. Maternal Diabetes mellitus - effects on the Fetus and placenta, Ch.8. In Naeye Kissane, JM (eds). Prenatal disease, Williams or Wilkins (1981).

Jone, KL. et al. The amniotic band disruption complex. J.Pediatrics. 95 : 554 ; 1981.

Kaufmann, P., Sen, DK. and Sch welkhart, G. Classification of human placental villi cell tissue. Res : 200 : 409 - 423 ; 1979

Kaplan, C. Placental pathology Pathol Annual. 28 : 15 - 72 ; 1993.

Kliman, HJ. Perotta, PL. The efficacy of placental biopsy. AmJ.OG. 1995, Oct 173 (4) ; 1084 - 8.

Las Heras, J. and Harding, PG. *et al.* Morphometric studies of fetal vasculature in hypertensive disease of pregnancy. Lab invest, 38 : 353 ; 1980.

Macpherson, T. The principles of surgical pathology 2nd ed. New York, NY. Churchill, Linngstore, Inc. 1825 - 1856 ; 1990.

Many A, Hubel CA, Fisher SJ et al: Invasive cytotrophoblasts manifest evidence of oxidative stress in preeclampsia. AM J. pathol 156: 321, 2000

Mirchandani, JJ., Mallik and Chitra, S. Histopathology of placenta. J.Obstetrics, Gyne - India. 29 : 40 ; 1989.

Moe, N. Deposits of fibrin and plasma protein in the normal human placenta : an immunofluorescence study. Acta. Pathol. microbial. Scand. 76 : 74 ; 1969.

Naeye, RL. Placental infarction leading to fetal or neonatal death a prospective study. Obstect Gynecol 50 : 583 - 388 ; 1997.

Naeye, RL. Functionally important disorders of placenta, umbilical cord and fetal membranes. Hum. Patho. 18 : 680 - 691 : 1987

Naeye, RL. Do placental weight have clinical significance? Human. Pathol. 18 : 387 - 391 ; 1987.

Ounsted, M. Histopathology of placenta in Intrauterine growth restriction -Nature, 212 : 995 ; 1966,

Rayburn, WF and Brinknan, DL. Umbilical cord length and intraposition complications. Obst. Gynecol. 57 : 450 - 452 ; 1981.

Redman CW, Sargent IL: Placental debris, oxidative stress and preeclampsia. Placenta 21: 597, 2000.

Russel, P. Infections of placental villi. In Fox, H. ed. Obstetrical and Gynecological pathology, Newyork, NY. Churchill Living stone, Inc. 2 : 1979 - 1116 ; 1987.

Sabharwal, B.D. Malhotra, HPE of placenta in anaemia. Journal of obstetrics Gynecology, India. 37 : 773.; 1987.

Sagol S, Ozkinay E,: The comparison of Uterine artery Doppler velocimetry with the histopathology of placental bed. Aust NZJ Obstet Gynecol. 39:324,1999.

Salafia cin - vintzileos. Gill berman and Banthom, KF. Placental pathology in idiopathic IUGR at term. Amh. Perinatal, 8 - 179 - 184 ; 1992.

Sheppard BL, Bonnar J: Uteroplacental hemostasis in intrauterine fetal growth retardation. Semin thromb Hemost 25: 443, 1999.

Shirley, G. and Driscoll, MD. Placental examination in a clinical setting. Arch. Patho. Lab. Med. Vol. 116, 668 - 671 ; 1991.

S.L.Costa, L Proctor, JM Dodd : Placenta Vol 29, I 12 P 1034-1040, Dec 2008.

Teasdale, F. Functional significance of the zonal morphologic, differences in the normal human placenta. Am J. Obstet Gynecol. 130 : 773 ; 1978.

Trera Macpherson. Fact Fancy. What can we really tell from placenta. Arch.Patho. Lab.Med. 115 : 672 - 678 : 1991.

Wiggleswoth, JS. The placenta in perinatal pathology Philadelphia Pa; WB Saunder, Co. 48 - 3 ; 1984.

William ob • 21 edition. Pathology of placental membranes. (86.106).

Wilkoning, RB. Anderson, S. etal., Placental transfer as a function of uterine blood flow. Am. J.Physioll ; 242 - 249 ; 1982.

Zeek, PM. and Assali, NS. Vascular changes in the decidua associated with eclamptogenic toxaemia, of pregnancy. Am.J.Clin. Pathol. 20 : 1099 : 1986.

PROFORMA

PROFORMA

Name :

Age :

IP.No :

Unit :

DOA :

GPLA :

Booked :

Study Group :

Menstrual history :

Marital history :

OBSTETRIC HISTORY

I Trimester :

II Trimester :

III Trimester :

DETAILS OF MEDICAL DISORDER :

Past history :

Family history :

Personal history :

EXAMINATION

General Examination :

Vital Signs :

Per Abdomen :

Mode of delivery :

Duration of II Stage :

Duration of III Stage :

Mode of delivery of placenta :

BABY DETAILS:

Fetal distress / Birth weight /

APGAR / NICU admission,

Cause of death :

Postnatal follow up :

INVESTIGATION

Basal :

Specific to disease

UTERINE ARTERY DOPPLER

Systolic diastolic ratio

Early diastolic notch

Pulsatility index

Resistance index

EXAMINATION OF PLACENTA

Gross Weight :

Placental completeness :

Size :

Shape :

Fetal Placental ratio :

PLACENTAL MATERNAL SURFACE

Infarct :

Fibrosis :

Placental Bleeding :

Other details :

FETAL SURFACE:

Anemia :

Circumvallate :

Circummarginate :

Other details :

CORD

Length :

Edema :

Cord insertion :

Knot : **No of Vessels :**

Other details :

MEMBRANE

Colour : **Foul Smelling :**

Other Details :

MICROSCOPIC EXAMINATION

BASAL PLATE OR DECIDUA

Inflammation :

Abscess :

Infarction :

Fibrinoid change in vessels :

Thrombosis :

VILLOUS PATHOLOGY

1).Synctytial Knot count

	GR I	GR II	GR III
	0-29%	30-59%	> 60%
2)Fibrinoid necrosis	0-5%	5-10%	>10%
3)Basement Membrane	Absent	Present	
Thickening		0-2%	>2%
4)Stromal Fibrosis	<3	3 – 8	>8
5)Cytotrophoblastic Proliferation	Absent	Present	
6)Infarction	<10	10-30	>30
7)Calcification	Absent	Present	

CORD AND MEMBRANES

Neutrophilic infiltration :

Necrotizing features :

Other details :

MASTER CHART

MASTER CHART

SL.NO	Name	Age	Group	UAD		FPR	Macroscopic						MICROSCOPY																																Fetal Outcome		
							I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I			FN			TBM			NI			SF			CP		CAL			LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
1	Anitha	22	I			5.2						+					+			+				+			+			+			+			+		+		LB		
2	Aasha	23	II a			4.5										+		+			+				+			+		+				+			+	+		LB	LAS	
3	Aarthi	22	I			4.9											+			+				+			+			+			+			+		+	LB		
4	Anindita	24	II b	+	↑	3.5	+		+				+	+	+	+	+			+					+		+			+			+			+		+	LB		
5	Archana	25	I			5						+					+			+			+			+			+			+			+		+	LB		
6	Ashikha	24	I			5.2											+			+			+			+			+			+			+		+	LB		
7	Anusha	25	II C	+	↑	3.2	+		+						+	+		+			+				+			+			+		+		+	+		SB		
8	Abinaya	26	I			5.1				+							+			+			+			+			+			+			+		+	LB		
9	Abirami	27	I			4.9											+			+			+			+			+			+			+		+	LB		
10	Aalaya	30	II a	+	↑	3.5	+								+			+		+					+			+		+	+		+		+	+		SB		
11	Anushiya	26	I			5											+			+			+			+			+			+			+		+	LB		
12	Akshara	27	II c			4			+				+		+	+	+					+			+		+			+			+		+	+		LB	LAS	
13	Akshaya	22	II b	+		5.5					+			+				+		+					+		+			+			+			+		+	LB		END
14	Bharathi	23	I			4.8											+			+			+			+			+			+			+		+	LB		
15	Bharani	24	II B			6	+		+									+		+			+				+		+			+			+		+	LB	LAS	
16	Begum	23	I			3.9										+	+			+			+			+			+			+			+		+	LB		
17	Badri	22	II a	+	↑	3.5	+	+	+						+			+					+		+			+		+			+		+			+	SB		
18	Bhargavi	26	I			4.7											+			+			+			+			+			+			+		+	LB		
19	Bhakya	27	I		↑	4.5											+			+			+			+			+			+			+		+	LB		
20	Chitra	31	II B			5.8		+					+				+			+					+			+			+			+		+	LB	LAS	
21	chaitu	26	I			4.8			+								+			+			+			+			+			+			+		+	LB		
22	chithu	24	II C			3.6	+	+							+	+		+			+				+			+		+			+		+	+		LB	LAS	
23	Devi	23	I			4.9		+					+					+		+			+			+			+			+			+		+	LB	LAS	
24	Dhanya	24	II A	+	↑	3.7	+	+	+							+	+			+			+			+			+			+			+		+	LB		
25	Dhana	26	I			4.5											+			+			+			+			+			+			+		+	LB		
26	Diva	23	II b			5.4	+	+										+							+			+			+			+		+	LB	LAS	
27	Divya	25	I			5											+			+			+			+			+			+			+		+	LB		
28	Deethi	26	II c	+	↑	3.8		+		+			+			+		+			+				+			+			+			+		+	+		LB	LAS	
29	Deepthi	25	I			4.9								+			+			+			+			+			+			+			+		+	LB		
30	Durga	26	II B			5		+							+			+			+				+			+			+			+		+	LB	LAS	
31	Eeashwari	27	I			4.8											+			+			+			+			+			+			+		+	LB		
32	Elavarasi	28	I	+		4.6			+								+			+			+			+			+			+			+		+	LB		
33	Elamathi	25	II C			3.4	+	+					+	+	+	+			+		+				+			+		+	+		+		+	+		SB		
34	Fathima	26	I			4.5											+			+			+			+			+			+			+		+	LB		
35	Fousal	25	II a	+	↑	3.8		+	+					+		+			+		+				+			+			+			+		+	+		LB	LAS	
36	Gowri	30	I			4.2											+			+			+			+			+			+			+		+	LB		
37	Geetha	27	II B			5	+	+	+									+		+					+			+			+			+			+		+	LB		
38	Gayathri	28	II a			4		+		+						+	+			+			+			+			+			+			+		+	LB		
39	Ganga	29	I			4.6											+			+			+				+			+			+			+		+	LB		
40	Gayu	26	II c	+	↑	3.9	+	+	+					+		+		+			+				+			+			+			+		+	+		LB	LAS	
41	Haffeesa	27	I			4.6											+			+			+			+			+			+			+		+	LB		
42	Hepsiba	28	II a	+	↑	3.5	+	+						+		+		+			+				+			+			+			+		+	+		LB		END
43	Ilakkiya	27	I			4.2											+			+			+			+			+			+			+		+	LB		
44	Ilanjiyam	32	II c		↑	3.2	+	+						+				+			+				+			+		+			+			+		+	LB		END
45	Janaki	28	I			4.8											+			+			+			+			+			+			+		+	LB		
46	Janavi	27	II B			5.2		+			+						+			+			+			+			+			+			+		+	LB	LAS	
47	Jayanthi	32	I			4.1	+										+			+			+			+			+			+			+		+	LB		
48	Kamala	24	I			4.2											+			+			+					+		+		+			+		+	LB		
49	Komala	23	II a	+	↑	3.7	+	+						+				+		+			+			+			+			+			+		+	LB		END

SL.NO	Name	Age	Group			FPR	Macroscopic						MICROSCOPY																												Fetal Outcome	
				UAD			I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I				FN			TBM			NI			SF			CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
50	Kalaiselvi	24	I			4.8											+			+				+				+			+		+			+		+		LB		
51	Kannaki	22	I			5.2						+					+										+				+		+			+		+		LB		
52	Kavitha	23	II a			4.5										+		+		+	+			+	+			+		+		+		+				+		LB		
53	Kannanmma	22	I			4.9											+										+	+			+		+			+	+		+	LB	LAS	
54	Kannaki	24	II b		↑	3.5	+								+	+	+			+				+	+		+				+		+			+		+		LB		
55	Kamatchi	25	I	+		5			+			+	+				+			+							+				+		+			+		+		LB		
56	Kanmani	24	I			5.2											+			+				+			+	+			+		+			+			+	LB		
57	Kalaiarasi	25	II C		↑	3.2	+								+	+		+		+	+			+					+		+							+		SB		
58	Kalaimagal	26	I			5.1			+								+										+				+		+			+	+	+		LB		
59	Lavanya	27	I			4.9											+			+				+			+				+		+			+			+	LB		
60	Subha	30	II a		↑	3.5	+								+			+		+				+	+					+				+			+	+		SB		
61	Angalo	26	I			5											+			+							+	+			+		+			+			+	LB		
62	Vijaya	27	II c			4									+	+	+			+		+		+	+		+				+			+			+	+		LB		
+	Revathi	22	II b			5.5			+				+					+							+		+				+		+			+			+	LB	LAS	END
64	Meena	23	I	+		4.8											+			+							+				+		+			+		+		LB		
65	Subhashini	24	II B			6	+											+		+				+						+		+		+		+			+	LB		
66	Sumathi	23	I			3.9			+							+	+			+				+			+	+			+		+			+		+		LB	LAS	
67	Suseela	22	II a		↑	3.5	+	+							+			+		+			+	+	+				+		+			+			+		+	SB		
68	Suganya	26	I			4.7			+								+									+	+				+		+			+		+		LB		
69	Uma	27	I		↑	4.5											+			+				+			+				+		+			+		+		LB		
70	Usha	31	II B	+		5.8		+									+			+				+	+						+			+			+	+		LB		
71	Kalpana	26	I			4.8							+				+			+						+	+	+			+		+			+			+	LB	LAS	
72	Sharvani	24	II C			3.6	+	+	+						+	+		+		+		+		+	+					+		+		+	+		+	+		LB		
73	Poornima	23	I			4.9		+										+								+		+			+		+			+			+	LB	LAS	
74	Subhashree	24	II A		↑	3.7	+	+								+	+			+				+			+	+			+		+			+		+		LB	LAS	
75	Jayashree	26	I	+		4.5			+								+			+				+			+				+		+			+			+	LB		
76	Ayisha	23	II b			5.4	+	+										+		+				+	+						+			+			+	+		LB		
77	Vinula	25	I			5											+										+	+			+		+			+			+	LB	LAS	
78	Bharani	26	II c		↑	3.8		+								+		+		+	+			+	+						+		+				+	+		LB		
79	Bargavi	25	I			4.9							+				+										+	+			+		+			+		+		LB	LAS	
80	Sumithra	26	II B			5		+							+			+		+				+	+						+			+		+		+		LB		
81	Sujitha	27	I			4.8											+			+							+	+			+		+			+			+	LB	LAS	
82	Sujitha	28	I			4.6											+			+				+			+				+		+			+		+		LB		
83	Janavi	25	II C	+		3.4	+	+	+						+	+			+	+	+			+	+					+		+		+			+	+		SB		
84	Jayanthi	26	I			4.5							+				+										+	+			+		+			+			+	LB		
85	Kamala	25	II a		↑	3.8		+								+			+	+	+			+	+						+		+				+	+		LB		
86	Komala	30	I	+		4.2			+								+											+			+		+			+		+		LB	LAS	
87	Kalaiselvi	27	II B			5	+	+										+		+				+	+		+				+		+			+			+	LB		
88	Akshara	28	II a			4		+	+							+	+			+							+				+		+			+		+		LB		
89	Akshaya	29	I			4.6											+			+				+							+		+			+		+		LB		
90	Bharathi	26	II c		↑	3.9	+	+								+		+		+	+			+	+		+	+			+		+				+	+		LB		
91	Bharani	27	I	+		4.6			+								+														+		+			+			+	LB	LAS	
92	Begum	28	II a		↑	3.5	+	+								+		+		+	+			+	+		+	+			+			+			+	+		LB		END
93	Badri	27	I			4.2											+										+				+		+			+		+		LB		
94	Bhargavi	32	II c		↑	3.2	+	+										+		+				+	+					+		+	+				+		+	LB		END
95	Bhakya	28	I	+		4.8											+			+								+			+		+			+		+		LB		
96	Chitra	27	II B			5.2		+									+			+				+			+	+			+			+		+		+		LB		
97	chaitu	32	I	+		4.1	+										+			+				+			+				+		+			+			+	LB	LAS	
98	Ganga	24	I			4.2											+			+				+					+		+		+			+		+		LB		

SL.NO	Name	Age	Group	UAD		FPR	Macroscopic					MICROSCOPY																																Fetal Outcome	
							I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I			FN			TBM			NI			SF			CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
99	Gayu	23	II a		↑	3.7	+	+										+		+				+	+		+			+	+	+	+		+		+		LB		END
100	Haffeesa	24	I	+		4.8											+			+				+			+				+		+		+		+		LB		
101	Sasi	22	I			5.2						+					+							+			+				+		+		+		+		LB		
102	Devi	23	II a			4.5										+		+		+	+				+					+				+		+	+	LB		
103	Shobana	22	I			4.9											+							+			+	+			+		+		+			+	LB	LAS	
104	Sangavi	24	II b	+	↑	3.5	+								+	+	+			+					+		+				+		+		+		+		LB		
105	Sangeetha	25	I			5			+			+	+				+			+				+			+				+		+		+		+		LB		
106	Saranya	24	I			5.2											+			+				+			+				+		+		+			+	LB		
107	Pragathi	25	II C	+	↑	3.2	+								+	+		+		+	+			+				+	+		+					+	+		SB		
108	Praveena	26	I			5.1			+								+							+			+				+		+		+		+		LB		
109	Rupina	27	I			4.9											+			+				+			+				+		+		+			+	LB		
110	Akshaya	30	II a	+	↑	3.5	+								+			+		+				+	+					+		+			+	+			SB		
111	Arulmary	26	I			5											+			+				+			+	+			+		+		+			+	LB		
112	Imaiya	27	II c			4									+	+	+			+		+			+		+				+			+		+	+	LB		
113	Ilakkiya	22	II b	+		5.5			+				+					+						+	+		+				+		+		+			+	LB	LAS	END
114	Dhanusha	23	I			4.8											+			+				+			+				+		+		+		+		LB		
115	Dharani	24	II B			6	+											+		+										+			+		+			+	LB		
116	Tamilarasi	23	I			3.9			+							+	+			+				+			+	+			+		+		+		+		LB	LAS	
117	Shivani	22	II a	+	↑	3.5	+	+							+			+		+			+	+	+			+		+			+			+		+	SB		
118	Ilanjiyam	26	I			4.7			+								+							+			+				+		+		+		+		LB		
119	Murugayi	27	I		↑	4.5											+			+				+			+				+		+		+		+		LB		
120	Vasantha	31	II B			5.8		+									+			+				+	+						+			+		+	+		LB		
121	Kumutha	26	I			4.8							+				+			+				+			+	+			+		+		+			+	LB	LAS	
122	Priya	24	II C			3.6	+	+	+						+	+		+		+		+			+					+			+			+	+	LB		
123	Prasanna	23	I			4.9		+										+						+				+				+		+		+			+	LB	LAS	
124	Sneha	24	II A	+	↑	3.7	+	+								+	+			+				+				+	+			+		+		+		+		LB	LAS	
125	Gopika	26	I			4.5			+								+			+				+			+				+		+		+			+	LB		
126	Elamathi	23	II b			5.4	+	+										+		+				+	+						+			+		+	+		LB		
127	Fathima	25	I			5											+							+				+			+		+		+			+	LB	LAS	
128	Fousal	26	II c	+	↑	3.8		+								+		+		+	+				+						+			+		+	+		LB		
129	Gowri	25	I			4.9							+				+							+				+			+		+		+			+	LB	LAS	
130	Geetha	26	II B			5		+							+			+		+					+						+			+		+			LB		
131	Gayathri	27	I			4.8											+			+				+				+	+		+			+		+			+	LB	LAS	
132	Ganga	28	I	+		4.6											+			+								+			+		+		+		+		LB		
133	Gayu	25	II C			3.4	+	+	+						+	+			+	+	+			+	+					+			+			+	+		SB		
134	Thara	26	I			4.5							+				+							+				+	+					+			+	LB		
135	Aruna	25	II a	+	↑	3.8		+								+			+	+	+					+					+		+				+	+	LB		
136	Renu	30	I			4.2			+	+							+							+				+			+		+		+		+		LB	LAS	
137	Banu	27	II B			5	+	+										+		+					+		+				+		+		+			+	LB		
138	Bharathi	28	II a			4		+	+							+	+			+				+			+				+		+		+		+		LB		
139	Bhavani	29	I			4.6											+			+											+		+		+		+		LB		
140	Bhavithra	26	II c		↑	3.9	+	+								+		+		+	+			+	+		+	+			+		+				+	+	LB		
141	Alamelu	27	I			4.6			+								+							+							+		+		+			+	LB	LAS	
142	Valarmathi	28	II a	+	↑	3.5	+	+								+		+		+	+				+			+	+			+			+		+	+	LB		END
143	Vasuki	27	I			4.2											+							+				+			+		+		+		+		LB		
144	Shanthi	32	II c	+	↑	3.2	+	+										+		+				+							+		+		+			+	LB		END
145	Rama	28	I			4.8											+			+				+				+			+		+		+		+		LB		
146	Ramalakshmi	27	II B	+		5.2		+									+			+							+	+			+			+		+			LB		
147	Ruppa	32	I			4.1	+										+			+				+				+			+		+		+			+	LB	LAS	

SL.NO	Name	Age	Group	UAD		FPR	Macroscopic						MICROSCOPY																																Fetal Outcome		
							I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I				FN			TBM			NI			SF			CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
148	Ruppvathi	24	I			4.2											+			+				+					+	+					+		+		LB		
149	Selvi	23	II a	+	↑	3.7	+	+										+		+				+	+		+			+	+		+			+		+	LB		END
150	Kanitha	24	I			4.8											+			+				+						+		+			+		+		LB		
151	Kala	22	I			5.2						+					+							+			+			+		+			+		+		LB		
152	Rajalakshmi	23	II a			4.5										+		+		+	+				+					+				+			+	+		LB		
153	Meenakshi	22	I			4.9											+							+			+	+			+		+			+			+	LB	LAS	
154	Mangalam	24	II b		↑	3.5	+								+	+	+			+					+			+			+		+			+		+		LB		
155	Chandra	25	I	+		5			+			+	+				+			+				+			+			+		+			+		+		LB		
156	Janavi	24	I			5.2											+			+				+			+			+		+			+			+	LB		
157	Janaki	25	II C		↑	3.2	+								+	+		+		+	+			+					+	+						+	+		SB		
158	Yokeshwari	26	I	+		5.1			+								+							+			+			+		+			+		+		LB		
159	Murugalakshmi	27	I			4.9											+			+				+			+			+		+			+			+	LB		
160	Semmalar	30	II a		↑	3.5	+								+			+		+				+	+				+				+			+	+		SB		
161	Srividhya	26	I	+		5											+			+				+			+	+		+		+			+			+	LB		
162	Sridevi	27	II c			4									+	+	+			+	+			+			+			+		+			+		+	+	LB		
163	Badmapriya	22	II b			5.5			+				+					+						+	+		+			+		+			+			+	LB	LAS	END
164	Sabari	23	I	+		4.8											+			+							+			+		+			+		+		LB		
165	Malarkodi	24	II B			6	+											+		+										+			+			+		+	LB		
166	Lakshmi	23	I			3.9			+							+	+			+				+			+	+		+		+			+		+		LB	LAS	
167	Nalini	22	II a		↑	3.5	+	+							+			+		+			+	+				+		+		+			+		+	+	SB		
168	Usharani	26	I			4.7			+								+							+			+			+		+			+		+		LB		
169	Valli	27	I		↑	4.5											+			+							+			+		+			+		+		LB		
170	Vedhavalli	31	II B	+		5.8		+									+			+				+	+					+					+		+		LB		
171	Sankari	26	I			4.8							+				+			+				+			+	+		+		+			+			+	LB	LAS	
172	Sarala	24	II C			3.6	+	+	+						+	+		+		+		+			+					+			+			+	+		LB		
173	Charu	23	I			4.9		+										+						+				+		+		+			+			+	LB	LAS	
174	Chandra	24	II A		↑	3.7	+	+								+	+			+				+	+		+			+		+			+		+		LB	LAS	
175	Renu	26	I	+		4.5			+								+			+				+			+			+		+			+			+	LB		
176	Banu	23	II b			5.4	+	+										+		+				+	+					+			+			+	+		LB		
177	Bharathi	25	I			5											+							+			+	+		+		+			+			+	LB	LAS	
178	Bhavani	26	II c		↑	3.8		+								+		+		+	+				+					+			+			+	+		LB		
179	Bhavithra	25	I	+		4.9							+				+							+			+	+		+		+			+			+	LB	LAS	
180	Alamelu	26	II B			5		+							+			+		+					+					+			+			+		+	LB		
181	Valarmathi	27	I			4.8											+			+				+				+		+		+			+			+	LB	LAS	
182	Vasuki	28	I			4.6											+			+				+			+			+		+			+		+		LB		
183	Shanthi	25	II C	+		3.4	+	+	+						+	+			+		+	+			+	+				+			+			+	+		SB		
184	Rama	26	I			4.5							+				+							+			+	+		+		+			+			+	LB		
185	Ramalakshmi	25	II a		↑	3.8		+								+			+		+	+				+					+			+			+	+		LB		
186	Ruppa	30	I	+		4.2			+								+							+				+		+		+			+			+	LB	LAS	
187	Gayathri	27	II B			5	+	+										+		+				+			+			+		+			+			+	LB		
188	Ganga	28	II a			4		+	+							+	+			+				+			+			+		+			+			+	LB		
189	Gayu	29	I			4.6											+			+										+		+			+			+	LB		
190	Thara	26	II c		↑	3.9	+	+								+		+		+	+			+	+		+	+		+		+			+		+	+	LB		
191	Aruna	27	I	+		4.6			+								+							+						+		+			+			+	LB	LAS	
192	Renu	28	II a		↑	3.5	+	+								+		+		+	+				+		+	+		+			+			+	+		LB		END
193	Banu	27	I	+		4.2											+							+						+		+			+			+	LB		
194	Mithra	32	II c		↑	3.2	+	+										+		+					+				+		+	+	+			+			+	LB		END
195	Madhu	28	I			4.8											+			+				+				+		+		+			+			+	LB		
196	Madhumathi	27	II B			5.2		+									+			+							+	+		+			+			+		+	LB		

SL.NO	Name	Age	Group	UAD		FPR	Macroscopic						MICROSCOPY																											Fetal Outcome		
							I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I				FN			TBM			NI			SF			CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
246	Vasantha	27	II B	+		5.2		+									+			+							+	+			+			+		+		+		LB		
247	Pooniamma	32	I			4.1	+										+			+				+			+				+		+			+			+	LB	LAS	
248	Poongothai	24	I			4.2											+			+				+					+		+		+			+		+		LB		
249	Poovarasi	23	II a	+	↑	3.7	+	+										+		+				+	+		+			+				+			+		+	LB		END
250	Reena	24	I			4.8											+			+				+							+		+			+		+		LB		
251	Neena	22	I			5.2						+					+							+			+				+		+			+		+		LB		
252	Fathima	23	II a			4.5										+		+		+	+				+					+				+				+		LB		
253	Arivazhaki	22	I			4.9											+							+			+	+			+		+			+	+		+	LB	LAS	
254	Andal	24	II b		↑	3.5	+								+	+	+			+					+		+				+		+			+		+		LB		
255	Anbarasi	25	I			5						+	+				+			+				+			+				+		+			+		+		LB		
256	Selvi	24	I			5.2			+								+			+							+				+		+			+			+	LB		
257	Sangeetha	25	II C		↑	3.2	+								+	+		+		+	+			+					+		+						+	+		SB		
258	Geetha	26	I	+		5.1											+							+			+				+		+			+		+		LB		
259	Jayashree	27	I			4.9			+								+			+							+				+		+			+			+	LB		
260	Deepika	30	II a		↑	3.5	+								+			+		+				+	+					+				+			+	+		SB		
261	Manjula	26	I			5											+			+				+			+	+			+		+			+			+	LB		
262	Devipriya	27	II c			4									+	+	+			+		+			+		+				+			+			+	+		LB		
263	Durgadevi	22	II b			5.5							+					+						+	+		+				+		+			+			+	LB	LAS	END
264	Vijaya	23	I	+		4.8			+								+			+							+				+		+			+		+		LB		
265	Vijayalakshmi	24	II B			6	+											+		+										+				+		+			+	LB		
266	Vimala	23	I			3.9										+	+			+				+			+	+			+		+			+		+		LB	LAS	
267	Vimalarani	22	II a		↑	3.5	+	+	+						+			+		+			+	+	+				+		+			+			+		+	SB		
268	Vimsi	26	I	+		4.7											+							+			+				+		+			+		+		LB		
269	Roopa	27	I		↑	4.5			+								+			+							+				+		+			+		+		LB		
270	Prasanna	31	II B	+		5.8		+									+			+				+	+						+			+			+	+		LB		
271	Sneha	26	I			4.8							+				+			+				+			+	+			+		+			+			+	LB	LAS	
272	Gopika	24	II C			3.6	+	+							+	+		+		+		+			+					+				+			+	+		LB		
273	Elamathi	23	I			4.9		+	+									+						+				+			+		+			+			+	LB	LAS	
274	Fathima	24	II A		↑	3.7	+	+								+	+			+							+	+			+		+			+		+		LB	LAS	
275	Bagya	26	I	+		4.5											+			+				+			+				+		+			+			+	LB		
276	Bagyalakshmi	23	II b			5.4	+	+	+									+		+				+	+						+			+			+	+		LB		
277	V elammal	25	I			5											+							+			+	+			+		+			+			+	LB	LAS	
278	Palaniammal	26	II c		↑	3.8		+								+		+		+	+				+						+		+				+	+		LB		
279	Kala	25	I			4.9							+				+							+			+	+			+		+			+		+		LB	LAS	
280	Durga	26	II B			5		+							+			+		+					+						+			+		+		+		LB		
281	Jothi	27	I			4.8											+			+				+			+	+			+		+			+			+	LB	LAS	
282	Sarashwathi	28	I			4.6											+		+	+							+				+		+			+		+		LB		
283	Sarala	25	II C	+		3.4	+	+							+	+				+	+			+	+					+			+	+			+	+		SB		
284	Hema	26	I			4.5			+				+				+		+					+			+	+			+		+			+			+	LB		
285	Lavanya	25	II a		↑	3.8		+								+				+	+				+						+		+				+	+		LB		
286	Sara	30	I	+		4.2											+							+				+			+		+			+		+		LB	LAS	
287	Anu	27	II B			5	+	+	+									+		+					+		+				+		+			+			+	LB		
288	Anjalaiammal	28	II a			4		+								+	+			+				+			+				+		+			+		+		LB		
289	Keerthana	29	I			4.6			+								+			+											+		+			+		+		LB		
290	Keerthi	26	II c		↑	3.9	+	+								+		+		+	+			+	+		+	+			+		+				+	+		LB		
291	Sahayarani	27	I			4.6											+							+							+		+			+			+	LB	LAS	
292	Safina	28	II a		↑	3.5	+	+	+							+		+		+	+				+		+	+			+			+			+	+		LB		END
293	Sarashwathi	27	I			4.2											+							+			+				+		+			+		+		LB		
294	Komalavalli	32	II c		↑	3.2	+	+										+		+					+					+			+				+		+	LB		END

SL.NO	Name	Age	Group			FPR	Macroscopic						MICROSCOPY																											Fetal Outcome	
				UAD			I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I				FN			TBM			NI			SF			CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
246	Vasantha	27	II B	+		5.2		+									+			+							+	+			+			+		+		+		LB		
247	Pooniamma	32	I			4.1	+										+			+				+			+				+		+			+			+	LB	LAS	
248	Poongothai	24	I			4.2											+			+				+					+		+		+			+		+		LB		
249	Poovarasi	23	II a	+	↑	3.7	+	+										+		+				+	+		+			+				+			+		+	LB		END
250	Reena	24	I			4.8											+			+				+							+		+			+		+		LB		
251	Neena	22	I			5.2						+					+							+			+				+		+			+		+		LB		
252	Fathima	23	II a			4.5										+		+		+	+				+					+				+				+		LB		
253	Arivazhaki	22	I			4.9											+							+			+	+			+		+			+	+		+	LB	LAS	
254	Andal	24	II b		↑	3.5	+								+	+	+			+					+		+				+		+			+		+		LB		
255	Anbarasi	25	I			5						+	+				+			+				+			+				+		+			+		+		LB		
256	Selvi	24	I			5.2			+								+			+							+				+		+			+			+	LB		
257	Sangeetha	25	II C		↑	3.2	+								+	+		+		+	+			+					+		+						+	+		SB		
258	Geetha	26	I	+		5.1											+							+			+				+		+			+		+		LB		
259	Jayashree	27	I			4.9			+								+			+							+				+		+			+			+	LB		
260	Deepika	30	II a		↑	3.5	+								+			+		+				+	+					+				+			+	+		SB		
261	Manjula	26	I			5											+			+				+			+	+			+		+			+			+	LB		
262	Devipriya	27	II c			4									+	+	+			+		+			+		+				+			+			+	+		LB		
263	Durgadevi	22	II b			5.5							+					+						+	+		+				+		+			+			+	LB	LAS	END
264	Vijaya	23	I	+		4.8			+								+			+							+				+		+			+		+		LB		
265	Vijayalakshmi	24	II B			6	+											+		+										+				+		+			+	LB		
266	Vimala	23	I			3.9										+	+			+				+			+	+			+		+			+		+		LB	LAS	
267	Vimalarani	22	II a		↑	3.5	+	+	+						+			+		+			+	+	+				+		+			+			+		+	SB		
268	Vimsi	26	I	+		4.7											+							+			+				+		+			+		+		LB		
269	Roopa	27	I		↑	4.5			+								+			+							+				+		+			+		+		LB		
270	Prasanna	31	II B	+		5.8		+									+			+				+	+						+			+			+	+		LB		
271	Sneha	26	I			4.8							+				+			+				+			+	+			+		+			+			+	LB	LAS	
272	Gopika	24	II C			3.6	+	+							+	+		+		+		+			+					+				+			+	+		LB		
273	Elamathi	23	I			4.9		+	+									+						+				+			+		+			+			+	LB	LAS	
274	Fathima	24	II A		↑	3.7	+	+								+	+			+							+	+			+		+			+		+		LB	LAS	
275	Bagya	26	I	+		4.5											+			+				+			+				+		+			+			+	LB		
276	Bagyalakshmi	23	II b			5.4	+	+	+									+		+				+	+						+			+			+	+		LB		
277	V elammal	25	I			5											+							+			+	+			+		+			+			+	LB	LAS	
278	Palaniammal	26	II c		↑	3.8		+								+		+		+	+				+						+		+				+	+		LB		
279	Kala	25	I			4.9							+				+							+			+	+			+		+			+		+		LB	LAS	
280	Durga	26	II B			5		+							+			+		+					+						+			+		+		+		LB		
281	Jothi	27	I			4.8											+			+				+			+	+			+		+			+			+	LB	LAS	
282	Sarashwathi	28	I			4.6											+		+	+							+				+		+			+		+		LB		
283	Sarala	25	II C	+		3.4	+	+							+	+				+	+			+	+					+			+	+			+	+		SB		
284	Hema	26	I			4.5			+				+				+		+					+			+	+			+		+			+			+	LB		
285	Lavanya	25	II a		↑	3.8		+								+				+	+				+						+		+				+	+		LB		
286	Sara	30	I	+		4.2											+							+				+			+		+			+		+		LB	LAS	
287	Anu	27	II B			5	+	+	+									+		+					+		+				+		+			+			+	LB		
288	Anjalaiammal	28	II a			4		+								+	+			+				+			+				+		+			+		+		LB		
289	Keerthana	29	I			4.6			+								+			+											+		+			+		+		LB		
290	Keerthi	26	II c		↑	3.9	+	+								+		+		+	+			+	+		+	+			+		+				+	+		LB		
291	Sahayarani	27	I			4.6											+							+							+		+			+			+	LB	LAS	
292	Safina	28	II a		↑	3.5	+	+	+							+		+		+	+				+		+	+			+			+			+	+		LB		END
293	Sarashwathi	27	I			4.2											+							+			+				+		+			+		+		LB		
294	Komalavalli	32	II c		↑	3.2	+	+										+		+					+					+			+				+		+	LB		END

SL.NO	Name	Age	Group	UAD EDN	UAD S/DR	FPR	Macroscopic I	Macroscopic C	MEC	TK	SU	SC	BASAL PLATE NI	BASAL PLATE A	BASAL PLATE I	BASAL PLATE FN	SK 0-29	SK 30 - 59	SK >60	I A	I <10	I IO-3O	I >30	FN o - 5	FN 5-15	FN > 15	TBM A	TBM I - 2	TBM > 2	NI P	NI A	SF ABS	SF <3	SF 3-8	SF >8	CP A	CP P	CAL A	CAL P	LB/SB	Fetal Outcome APGAR	Fetal Outcome END
295	Dharani	28	I			4.8											+			+				+				+			+		+			+		+		LB		
296	Neelambari	27	II B			5.2		+									+			+							+	+			+			+		+		+		LB		
297	Prasanna	32	I	+		4.1	+										+			+				+			+				+		+			+			+	LB	LAS	
298	Sneha	24	I			4.2											+			+				+					+		+		+			+		+		LB		
299	Gopika	23	II a		↑	3.7	+	+										+		+				+	+		+			+				+			+		+	LB		END
300	Elamathi	24	I	+		4.8											+			+				+							+		+			+		+		LB		
301	Fathima	22	I			5.2						+					+							+			+				+		+			+		+		LB		
302	Fousal	23	II a			4.5										+		+		+	+				+					+				+			+	+		LB		
303	Gowri	22	I			4.9											+							+			+	+			+		+			+			+	LB	LAS	
304	Sarathiammal	24	II b	+	↑	3.5	+								+	+	+			+					+		+				+		+			+		+		LB		
305	Sathyapriya	25	I			5			+			+	+				+			+				+			+				+		+			+		+		LB		
306	Santhosi	24	I			5.2											+			+							+				+		+			+			+	LB		
307	Chitra	25	II C	+	↑	3.2	+								+	+		+		+	+			+					+		+						+	+		SB		
308	chaitu	26	I			5.1			+								+							+			+				+		+			+		+		LB		
309	chithu	27	I			4.9											+			+							+				+		+			+			+	LB		
310	Devi	30	II a	+	↑	3.5	+								+			+		+				+	+					+				+			+	+		SB		
311	Dhanya	26	I			5											+			+				+			+	+			+		+			+			+	LB		
312	Dhana	27	II c			4									+	+	+			+		+			+		+				+			+			+	+		LB		
313	Diva	22	II b	+		5.5			+				+					+						+	+		+				+		+			+			+	LB	LAS	END
314	Divya	23	I			4.8											+			+							+				+		+			+		+		LB		
315	Deethi	24	II B			6	+											+		+										+				+		+			+	LB		
316	Deepthi	23	I			3.9			+							+	+			+				+			+	+			+		+			+		+		LB	LAS	
317	Anjalaiammal	22	II a	+	↑	3.5	+	+							+			+		+			+	+	+				+		+		+	+			+		+	SB		
318	Anjalidevi	26	I			4.7			+								+							+			+				+		+			+		+		LB		
319	Chiristina	27	I	+	↑	4.5											+			+							+				+		+			+		+		LB		
320	Sasikala	31	II B			5.8		+									+			+				+	+						+			+			+	+		LB		
321	Pramila	26	I			4.8							+				+			+				+			+	+			+		+			+			+	LB	LAS	
322	Sathya	24	II C			3.6	+	+	+						+	+		+		+		+			+					+				+			+	+		LB		
323	Ayisha	23	I			4.9		+										+						+				+			+		+			+			+	LB	LAS	
324	Akila	24	II A	+	↑	3.7	+	+								+	+			+							+	+			+		+			+		+		LB	LAS	
325	Abinaya	26	I			4.5			+								+			+				+			+				+		+			+			+	LB		
326	Oviya	23	II b			5.4	+	+										+		+				+	+						+			+			+	+		LB		
327	Pattammal	25	I			5											+							+			+	+			+		+			+			+	LB	LAS	
328	Saroja	26	II c	+	↑	3.8		+								+		+		+	+				+						+		+				+	+		LB		
329	Kunjammal	25	I			4.9							+				+							+			+	+			+		+			+		+		LB	LAS	
330	Marimuthu	26	II B			5		+							+			+		+					+						+		+	+		+		+		LB		
331	Marimuthu	27	I			4.8											+			+				+			+	+			+		+			+			+	LB	LAS	
332	Logeshwari	28	I			4.6											+			+							+				+		+			+		+		LB		
333	Anjeshwari	25	II C			3.4	+	+	+						+	+	+			+	+			+	+					+				+			+	+		SB		
334	Arunadevi	26	I			4.5							+				+							+			+	+			+		+			+			+	LB		
335	Akila	25	II a	+	↑	3.8		+								+	+			+	+				+						+		+				+	+		LB		
336	Anbarasi	30	I			4.2			+						+		+							+				+			+		+			+		+		LB	LAS	
337	Thangam	27	II B			5	+	+										+		+					+		+				+		+			+			+	LB		
338	Sellammal	28	II a			4		+	+							+	+			+				+			+				+		+			+		+		LB		
339	Pavithra	29	I			4.6											+			+											+		+			+		+		LB		
340	Praveena	26	II c	+	↑	3.9	+	+								+		+		+	+			+	+		+	+			+		+				+	+		LB		
341	Poornima	27	I			4.6			+								+							+							+		+			+			+	LB	LAS	
342	Hema	28	II a	+	↑	3.5	+	+								+		+		+	+				+		+	+			+			+			+	+		LB		END
343	Kala	27	I			4.2											+							+			+				+		+			+		+		LB		

SL.NO	Name	Age	Group	UAD		FPR	Macroscopic							MICROSCOPY																																Fetal Outcome		
							I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I			FN			TBM			NI			SF			CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
344	Kousalya	32	II c	+	↑	3.2	+	+										+		+					+					+			+				+		+		LB		END
345	Velankanni	28	I			4.8											+			+				+			+			+			+			+		+		LB		
346	Vellaimmal	27	II B	+		5.2		+									+			+						+	+			+				+		+		+		LB		
347	Banumathi	32	I			4.1	+										+			+				+		+				+			+			+		+		LB	LAS	
348	Yesvandhini	24	I			4.2											+			+				+				+	+	+			+			+		+		LB		
349	Yazhini	23	II a	+	↑	3.7	+	+										+		+				+	+	+			+				+				+		+		LB		END
350	Solaiamma	24	I			4.8											+			+				+						+			+			+		+		LB		
351	Selvi	22	I			5.2						+					+			+				+		+				+			+			+		+		LB		
352	Seethalakshmi	23	II a			4.5										+		+			+				+					+				+			+	+		LB		
353	Lakshmi	22	I			4.9											+			+				+		+	+			+			+			+			+	LB	LAS	
354	Lavanya	24	II b		↑	3.5	+								+	+	+								+		+				+			+			+		+		LB		
355	Jayanthi	25	I	+		5			+			+	+				+			+				+		+				+			+			+		+		LB		
356	Jaya	24	I			5.2											+			+				+		+				+			+			+			+	LB		
357	Sarala	25	II C		↑	3.2	+								+	+		+		+	+			+			+	+		+						+	+		SB		
358	Hema	26	I	+		5.1			+								+			+				+		+				+			+			+		+		LB		
359	Lavanya	27	I			4.9											+							+		+				+			+			+			+	LB		
360	Sara	30	II a		↑	3.5	+								+			+		+				+	+					+				+			+	+		SB		
361	Anu	26	I	+		5											+			+				+		+	+			+			+			+			+	LB		
362	Anjalaiammal	27	II c			4									+	+	+			+		+			+					+				+			+	+		LB		
363	Kavitha	22	II b			5.5			+				+					+		+				+	+		+	+		+			+			+			+	LB	LAS	END
364	Bhanu	23	I	+		4.8											+										+			+			+			+		+		LB		
365	Geetha	24	II B			6	+											+		+										+				+			+			+	LB		
366	Selvi	23	I			3.9			+							+	+			+				+			+	+		+			+			+			+	LB	LAS	
367	Uma	22	II a		↑	3.5	+	+							+			+		+			+	+	+			+			+			+			+		+		SB		
368	Ezhili	26	I			4.7			+						+		+			+				+			+			+			+			+		+		LB		
369	Rani	27	I		↑	4.5											+						+				+			+			+			+		+		LB		
370	Gowri	31	II B	+		5.8		+									+			+				+	+					+				+			+	+		LB		
371	Sathyapriya	26	I			4.8							+				+			+				+			+	+		+			+			+			+	LB	LAS	
372	Santhosi	24	II C			3.6	+	+	+						+	+		+		+		+			+				+				+			+	+		LB		
373	Chitra	23	I			4.9		+										+		+				+			+			+			+			+			+	LB	LAS	
374	chaitu	24	II A		↑	3.7	+	+								+	+						+				+	+		+			+			+		+		LB	LAS	
375	chithu	26	I	+		4.5			+								+			+				+			+			+			+			+			+	LB		
376	Devi	23	II b			5.4	+	+										+		+				+	+					+				+			+	+		LB		
377	Dhanya	25	I			5											+			+				+			+	+		+			+			+			+	LB	LAS	
378	Dhana	26	II c		↑	3.8		+								+		+			+				+					+				+			+	+		LB		
379	Diva	25	I	+		4.9							+				+			+				+			+	+		+			+			+			+	LB	LAS	
380	Divya	26	II B			5		+							+			+		+					+					+				+	+		+			LB	LAS	
381	Deethi	27	I			4.8											+			+				+			+	+		+			+			+			+	LB	LAS	
382	Deepthi	28	I			4.6											+			+							+			+			+			+		+		LB		
383	Anjalaiammal	25	II C	+		3.4	+	+	+						+	+					+	+			+	+				+				+			+	+		SB		
384	Bharathi	26	I			4.5							+				+			+				+			+	+		+			+			+			+	LB		
385	Ezhili	25	II a		↑	3.8		+								+			+		+				+					+				+			+	+		LB		
386	Elamathi	30	I			4.2			+								+			+				+				+		+			+			+			+	LB	LAS	
387	Elavarasi	27	II B			5	+	+										+		+				+			+			+			+			+			+	LB		
388	Radhika	28	II a			4		+	+							+	+			+				+			+			+			+			+			+	LB		
389	Devikala	29	I			4.6											+			+										+			+			+			+	LB		
390	Devi	26	II c		↑	3.9	+	+								+		+			+	+			+	+		+	+								+	+		LB		
391	Geetha	27	I	+		4.6			+								+			+				+			+			+			+			+		+		LB	LAS	
392	Dharani	28	II a		↑	3.5	+	+								+		+			+				+			+	+		+			+			+	+		LB		END

SL.NO	Name	Age	Group	UAD		FPR	Macroscopic			TK	SU	SC	MICROSCOPY																												Fetal Outcome	
							I	C	MEC				BASAL PLATE				SK			I				FN			TBM			NI			SF			CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
393	Kumutha	27	I			4.2									+		+			+				+			+				+		+			+		+		LB		
394	Logeshwari	32	II c		↑	3.2	+	+										+		+					+					+			+				+		+	LB		END
395	Asha	28	I			4.8											+			+				+				+			+		+			+		+		LB		
396	Mangai	27	II B			5.2		+									+			+							+	+			+			+		+		+		LB		
397	Madharasi	32	I	+		4.1	+										+			+				+			+				+		+			+			+	LB	LAS	
398	Mathanki	24	I			4.2											+			+				+					+		+		+			+		+		LB		
399	Sridevi	23	II a		↑	3.7	+	+										+		+				+	+		+	+		+				+			+		+	LB		END
400	Aalaya	24	I			4.8											+			+				+							+		+			+		+		LB		
401	Anushiya	22	I			5.2						+					+			+				+			+				+		+			+		+		LB		
402	Akshara	23	II a			4.5										+		+			+				+					+				+			+	+		LB		
403	Akshaya	22	I			4.9											+			+				+			+	+			+		+			+			+	LB	LAS	
404	Bharathi	24	II b	+	↑	3.5	+								+	+	+			+					+		+				+		+			+		+		LB		
405	Bharani	25	I			5			+			+	+				+			+				+			+				+		+			+		+		LB		
406	Begum	24	I			5.2											+			+							+				+		+			+			+	LB		
407	Badri	25	II C	+	↑	3.2	+								+	+		+		+	+			+				+	+		+						+	+		SB		
408	Bhargavi	26	I			5.1			+								+			+				+			+				+		+			+		+		LB		
409	Bhakya	27	I			4.9											+			+							+				+		+			+			+	LB		
410	Chitra	30	II a	+	↑	3.5	+								+			+		+				+	+					+				+			+	+		SB		
411	chaitu	26	I			5											+			+				+			+	+			+		+			+			+	LB		
412	chithu	27	II c			4									+	+	+			+		+			+		+	+			+			+			+	+		LB		
413	Suseela	22	II b	+		5.5			+				+					+		+				+	+		+				+		+			+			+	LB	LAS	END
414	Santhanalakshmi	23	I			4.8											+			+							+				+		+			+		+		LB		
415	Lakshmibala	24	II B			6	+											+		+										+			+	+			+		+	LB		
416	Saranya	23	I			3.9			+							+	+			+				+			+	+			+		+			+		+		LB	LAS	
417	Bargavi	22	II a	+	↑	3.5	+	+							+			+		+			+	+	+						+			+			+		+	SB		
418	Vinu	26	I			4.7			+								+			+				+			+				+		+			+		+		LB		
419	Seetha	27	I		↑	4.5											+			+							+				+		+			+		+		LB		
420	Seethai	31	II B			5.8		+									+			+				+	+						+			+			+	+		LB		
421	Thangam	26	I			4.8							+				+			+				+			+	+			+		+			+			+	LB	LAS	
422	Hema	24	II C			3.6	+	+	+						+	+		+		+		+			+					+				+			+	+		LB		
423	Velankanni	23	I			4.9		+										+		+				+				+			+		+			+			+	LB	LAS	
424	Vellaimmal	24	II A	+	↑	3.7	+	+								+	+			+							+	+			+		+			+		+		LB	LAS	
425	Banumathi	26	I			4.5			+								+			+				+			+				+		+			+			+	LB		
426	Yesvandhini	23	II b			5.4	+	+										+		+				+	+						+			+			+	+		LB		
427	Yazhini	25	I			5											+			+				+			+	+			+		+			+			+	LB	LAS	
428	Solaiamma	26	II c	+	↑	3.8		+								+		+			+				+						+		+				+	+		LB		
429	Selvi	25	I			4.9							+				+			+				+			+	+			+		+				+	+		LB	LAS	
430	Seethalakshmi	26	II B			5		+							+			+		+					+						+			+		+		+		LB		
431	Lakshmi	27	I			4.8											+			+				+			+	+			+		+			+			+	LB	LAS	
432	Sangeetha	28	I	+		4.6											+	+		+							+				+		+			+		+		LB		
433	Anjali	25	II C			3.4	+	+	+						+	+				+	+			+	+					+				+			+	+		SB		
434	Usha	26	I			4.5							+				+	+		+				+			+	+			+		+			+			+	LB		
435	Vijaya	25	II a	+	↑	3.8		+								+			+		+				+						+		+				+	+		LB		
436	Jayashree	30	I			4.2			+								+			+				+				+			+		+			+		+		LB	LAS	
437	Solaiamma	27	II B			5	+	+										+		+					+		+				+		+			+			+	LB		
438	Valarmathi	28	II a			4		+	+							+	+			+				+			+				+		+			+		+		LB		
439	Thangam	29	I			4.6											+			+											+		+			+		+		LB		
440	Mazharvizhi	26	II c	+	↑	3.9	+	+								+		+		+	+			+	+		+	+			+		+				+	+		LB		
441	Swarnalatha	27	I			4.6			+								+			+				+							+		+			+			+	LB	LAS	

SL.NO	Name	Age	Group			FPR	Macroscopic						MICROSCOPY																													Fetal Outcome	
				UAD			I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I				FN			TBM			NI		SF				CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
442	Neelambari	28	II a	+	↑	3.5	+	+								+		+			+				+		+	+			+			+			+	+		LB		END
443	Prasanna	27	I			4.2											+			+				+			+				+		+			+		+		LB		
444	Sneha	32	II c	+	↑	3.2	+	+										+							+					+			+	+			+		+	LB		END
445	Gopika	28	I			4.8											+			+				+				+			+		+			+		+		LB		
446	Elamathi	27	II B	+		5.2		+									+			+							+	+			+			+			+	+		LB		
447	Fathima	32	I			4.1	+										+			+				+			+				+		+			+			+	LB	LAS	
448	Fousal	24	I			4.2											+			+				+							+		+			+		+		LB		
449	Gowri	23	II a	+	↑	3.7	+	+										+		+				+	+		+			+				+			+		+	LB		END
450	Sarathiammal	24	I			4.8											+			+				+				+			+		+			+		+		LB		
451	Sathyapriya	22	I			5.2						+					+			+				+			+				+		+			+		+		LB		
452	Santhosi	23	II a			4.5		+								+		+			+				+					+				+			+	+		LB		
453	Chitra	22	I			4.9											+			+				+			+	+			+		+			+			+	LB	LAS	
454	Santhosi	24	II b		↑	3.5	+								+	+	+								+		+				+		+			+		+		LB		
455	Chitra	25	I	+		5			+				+				+			+				+			+				+		+			+		+		LB		
456	chaitu	24	I			5.2											+			+							+				+		+			+			+	LB		
457	chithu	25	II C		↑	3.2	+					+			+	+		+		+	+			+					+		+		+				+	+		SB		
458	Devi	26	I	+		5.1			+								+			+				+			+	+			+		+			+		+		LB		
459	Dhanya	27	I			4.9											+					+					+				+		+			+			+	LB		
460	Dhana	30	II a		↑	3.5	+	+	+						+			+		+				+	+					+				+			+	+		SB		
461	Diva	26	I	+		5											+			+				+			+	+			+		+			+			+	LB		
462	Divya	27	II c			4									+	+	+			+		+			+		+				+			+			+	+		LB		
463	Deethi	22	II b			5.5			+				+					+		+				+	+		+	+			+		+				+		+	LB	LAS	END
464	Deepthi	23	I	+		4.8											+										+				+		+			+		+		LB		
465	Anjalaiammal	24	II B			6	+											+		+										+				+		+			+	LB		
466	Bharathi	23	I			3.9			+							+	+			+				+			+	+			+		+			+		+		LB	LAS	
467	Asha	22	II a		↑	3.5	+	+							+			+		+			+	+	+				+		+			+			+		+	SB		
468	Mangai	26	I	+		4.7			+								+			+				+			+				+		+			+		+		LB		
469	Madharasi	27	I		↑	4.5											+					+			+		+				+		+			+		+		LB		
470	Mathanki	31	II B	+		5.8		+									+			+				+	+						+			+			+	+		LB		
471	Thulasi	26	I			4.8							+				+			+				+			+	+			+		+			+			+	LB	LAS	
472	Jeeva	24	II C	+		3.6	+	+	+						+	+		+		+		+			+					+				+			+	+		LB		
473	Thilaka	23	I			4.9		+										+		+				+				+			+		+			+			+	LB	LAS	
474	Arunadevi	24	II A		↑	3.7	+	+								+	+			+		+		+			+	+			+		+			+		+		LB	LAS	
475	Anjalai	26	I	+		4.5			+								+			+				+			+				+		+			+			+	LB		
476	Ruina	23	II b			5.4	+	+										+		+				+	+		+				+		+	+			+	+		LB		
477	Bharani	25	I			5											+			+				+			+	+			+		+			+			+	LB	LAS	
478	Saroja	26	II c	+	↑	3.8		+								+		+			+				+		+				+		+				+	+		LB		
479	Kunjammal	25	I			4.9							+				+			+				+			+	+			+		+			+		+		LB	LAS	
480	Marimuthu	26	II B	+		5		+							+			+				+		+	+		+				+			+		+		+		LB		
481	Marimuthu	27	I			4.8											+			+				+			+	+			+		+			+			+	LB	LAS	
482	Logeshwari	28	I			4.6											+	+		+				+			+				+		+			+		+		LB		
483	Baby	25	II C	+		3.4	+	+	+						+	+				+	+			+	+		+			+				+			+	+		SB		
484	Prathheba	26	I			4.5							+				+	+		+				+			+	+			+		+			+			+	LB		
485	Poornima	25	II a		↑	3.8		+								+					+				+		+				+		+				+	+		LB		
486	Devi	30	I	+		4.2			+								+			+				+				+			+		+			+		+		LB	LAS	
487	Sasikala	27	II B			5	+	+				+						+		+					+		+				+		+			+			+	LB		
488	Sasi	28	II a			4		+	+							+	+			+				+			+	+			+		+			+		+		LB		
489	Fathima	29	I			4.6											+			+					+		+				+		+			+		+		LB		
490	Sengamalam	26	II c		↑	3.9	+	+								+		+		+	+			+	+		+	+			+		+				+	+		LB		

SL.NO	Name	Age	Group	UAD		FPR	Macroscopic						MICROSCOPY																												Fetal Outcome	
							I	C	MEC	TK	SU	SC	BASAL PLATE				SK			I				FN			TBM			NI			SF			CP		CAL		LB/SB	APGAR	END
				EDN	S/DR								NI	A	I	FN	0-29	30 - 59	>60	A	<10	IO-3O	>30	o - 5	5-15	> 15	A	I - 2	> 2	P	A	ABS	<3	3-8	>8	A	P	A	P			
491	Keerthana	27	I	+		4.6			+								+			+				+			+				+		+			+			+	LB	LAS	
492	Janaki	28	II a		↑	3.5	+	+								+		+			+				+		+	+			+			+			+	+		LB		END
493	Mangai	27	I	+		4.2											+			+				+			+				+		+			+		+		LB		
494	Tamil Selvi	32	II c		↑	3.2	+	+										+		+					+		+			+			+				+		+	LB		END
495	Sumi	28	I	+		4.8										+	+			+				+				+			+		+			+		+		LB		
496	Keerthi	27	II B			5.2		+									+			+	+				+		+	+			+			+		+		+		LB		
497	Geetha	32	I	+		4.1	+										+			+				+			+				+		+			+			+	LB	LAS	
498	Elavarasi	24	I			4.2											+			+				+			+				+		+			+		+		LB		
499	Bharani	23	II a		↑	3.7	+	+	+									+		+				+	+		+			+				+			+		+	LB		END
500	Kalaivani	24	I	+		4.8											+			+				+							+		+			+		+		LB		

CODING

Group I - **Normal**

Group II - **High Risk**

a) **Preeclampsia**
b) **GDM**
c) **IUGR**

FPR - **Fetal placental ratio**

UAD - **Uterine artery doppler**

EDN - **Early diastolic notch**

S/D R - **Systolic diastolic ratio**

MACROSCOPIC FEATURES

I - **Infarction**

C - **Calcification**

MEC - **Meconium stained membrane**

TK - **True knot**

SU - **Single umbilical artery**

SC - **Septal cyst**

MICROSCOPIC FEATURES

BP - Basal plate

NI - Neutrophilic Infiltration

A - Abscess

I - Infarction

FN - Fibrinoid Necrosis

SK - Syncytial Knot

CAL - Calcification

SF - Stromal fibrosis

CP - Cytotrophoblastic proliferation

TBM - Thickening of Basement membrane

A - Absent

P - Present

ABS - Abscess

FO	**-**	**FETAL OUTCOME**
LAS	**-**	**Low APGAR score**
END	**-**	**Early neonatal Death**
LB	**-**	**Live Birth**
SB	**-**	**Still Birth**

ETHICAL COMMITTEE CERTIFICATE

I, **DR.P.VASANTHI M.D(O.G) P.G** apply for the ethical committee certificate for the project

"COMPARATIVE STUDY OF PLACENTAL ABNORMALITIES IN NORMAL AND SPECIFIC HIGH RISK PREGNANCIES BY DOPPLER, HISTOLOGY AND THEIR FETAL OUTCOME" under the guidance of **PROF.DR.KALAVATHY**,M.D.,D.G.O , Institute of Social Obstetrics and Govt KGH Chennai.

I understand the implications of doing research with human subjects and will fully comply with the regulations and keep the dignity and protect the health of subjects at all costs.

Vasanthi.

Signature of Postgraduate student

I have no objection to guide this postgraduate student in the project mentioned above. I shall supervise that all the human rights are protected and research is carried out with the utmost humanitarian principles.

V Kalavathy

Signature of the guide

Sen'or Civil Surgeon
Institute of Social Obstetrics and
Govt. Kasturba Gandhi Hospital for
Women and Children, Chepauk,
Triplicane, Chennai-600 005

Seal of guide

I certify that this project has been presented in front of the Ethical Committee, duly formatted in this institution and that all the members of the Ethical Committee have given permission to conduct this research.

CHAIRMAN
ETHICAL COMMITTEE
CHENNAI
Date:-

Chairman of Ethical Committee

Date: 21.12.2009

Seal of Chairman

PATIENT CONSENT FORM

STUDY TITLE:

COMPARATIVE STUDY OF PLACENTAL ABNORMALITIES IN NORMAL AND SPECIFIC HIGH RISK PREGNANCIES BY DOPPLER, HISTOLOGY AND THEIR FETAL OUTCOME

STUDY CENTRE:
Institute of Social Obstetrics and Govt KGH Chennai.

PARTICIPANT NAME: AGE: SEX: I.D. NO.

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties of published, unless as required under the law. I agree not to restrict the use of any or results that arise from the study.

I hereby consent to participate in this study of "**COMPARATIVE STUDY OF PLACENTAL ABNORMALITIES IN NORMAL AND SPECIFIC HIGH RISK PREGNANCIES BY DOPPLER, HISTOLOGY AND THEIR FETAL OUTCOME**"

Signature of Investigator

Place:
Date:

Study Investigators Name

Institution:

Signature / Thumb impression of patient

Thanking you,

Yours faithfully,

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

சாதாரண மற்றும் சிக்கலான கர்ப்பிணிகளில் டாப்ளர் ஸ்கேன் மற்றும் நஞ்சுக்கொடியின் திசு பரிசோதனை மேற்கொண்டு குழந்தையின் வளர்ச்சியை அதனோடு ஒப்பிட்டு கண்டறிதல்

ஆராய்ச்சி நிலையம்	:	சமூக மகப்பேறியல் மற்றும் அரசு கஸ்தூரிபாய்காந்தி தாய் சேய் நல மருத்துவமனை சென்னை மருத்துவக் கல்லூரி மற்றும் மருத்துவமனை சென்னை–600 005
பங்கு பெறுபவரின் பெயர்	:	
பங்கு பெறுபவரின் எண்	:	

மேலே குறிப்பிட்டுள்ள மருத்துவமனை ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும் , அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்தமேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன்.நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும், மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனத்துடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுகளையின் படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலே அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்..... இடம்..... தேதி

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம்.....தேதி

ஆய்வாளரின் பெயர் .....