ANTENATAL ULTRASOUND AND POSTPARTUM HISTOPATHOLOGICAL STUDY OF PLACENTA IN HIGH RISK PREGNANCIES AND ITS CORRELATION WITH FETAL OUTCOME

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CERTIFICATE

This is to certify that the dissertation titled "**ANTENATAL ULTRASOUND AND POSTPARTUM HISTOPATHOLOGICAL STUDY OF PLACENTA IN HIGH RISK PREGNANCIES AND ITS CORRELATION WITH FETAL OUTCOME"** is a bonafide work done by **Dr.R.VIDHYA** in the Department of Obstetrics and Gynaecology (Thanjavur Medical College) Thanjavur, in partial fulfillment of the university rules and regulations for award of M.D degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2015-2018

Prof.DR.R. RAJA RAJESWARI M.D.,DGO.,
Profesor,
Dept of Obstetrics and Gynaecology
Thanjavur Medical College,
Thanjavur.

Prof.DR. S. PRADEEBA, M.D.,OG,
Head of the Dept,
Dept of Obstetrics and Gynaecology,
Thanjavur Medical College,
Thanjavur.

Prof. DR.JEYAKUMAR, M.S.,Mch.,
Dean.
Thanjavur Medical College,
Thanjavur

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA - 613001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

INSTITUTIONAL ETHICAL COMMITTEE

CERTIFICATE

Approval No. : **335**

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submitted by Dr. VIDHYA. R. **of**

Dept. of OBSTETRICS AND GYNAECOLOGY **Thanjavur Medical College, Thanjavur**

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Thanjavur

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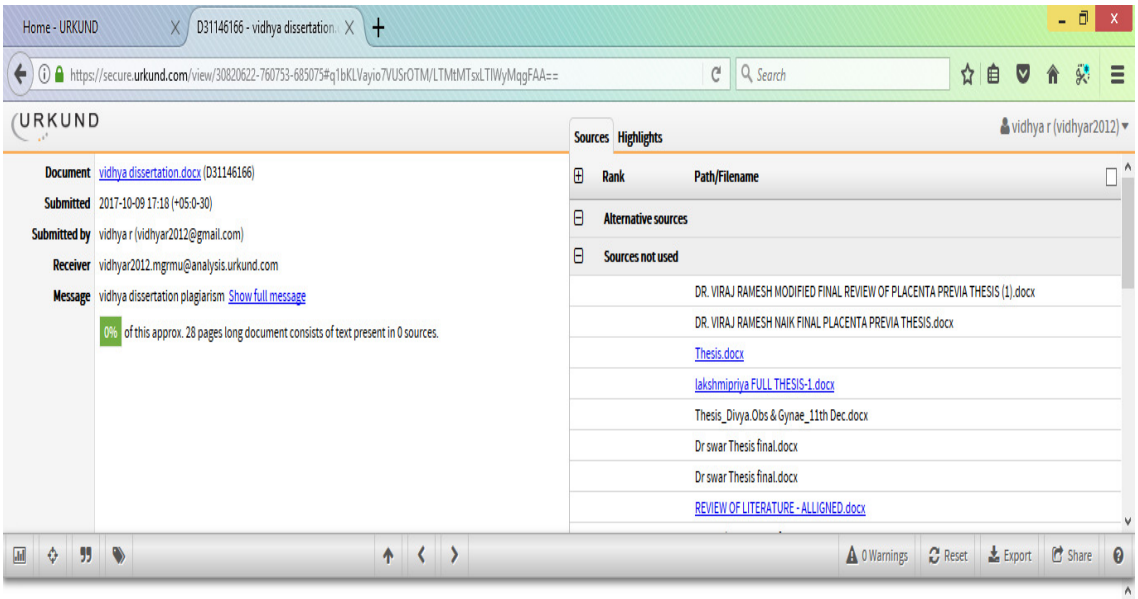

INTRODUCTION

In a high risk pregnancy, the birth of a health child is a time of joy and expectation. The determinant of fetal growth and metabolism is the exchange between mother and fetus across the placenta. A well nourished new born therefore, is the best evidence of adequate placental functioning. The placenta acts as a major functional unit for the well being of the fetus.

Moosman defined placenta as a fusion of the fetal membranes to the uterine mucosa for the transfer of oxygen and metabolites between mother and fetal blood (Willians 24th Edition).

As the placenta grows and ages, the histological changes suggest an increase in the efficiency of transport to meet the metabolic requirements of growing fetus. Hence any insult to placenta during development stage quantification of the placental changes is essential to correlate the outcome in terms of the fetus.

According to Benirshcke (1961), the placenta is the most accurate record to infant's prenatal experience and before the advent of ultrasonography, its evaluation was possible only after delivery.

Ultrasound placentography was first described by Cotterfeld (1968) while Cannum et al. (1970) proposed

CERTIFICATE – II

This is to certify that this dissertation work titled **ANTENATAL ULTRASOUND AND POSTPARTUM HISTOPATHOLOGICAL STUDY OF PLACENTA IN HIGH RISK PREGNANCIES AND ITS CORRELATION WITH FETAL OUTCOME** of the candidate **Dr.R.VIDHYA** with registration Number **221516205** for the award of the degree of in the branch of M.S Obstetrics & Gynaecology. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from Introduction to conclusion pages and result shows **0** percentage of plagiarism in the dissertation.

Prof.DR.R. RAJA RAJESWARI
M.D.,DGO.,
Profesor,
Dept of Obstetrics and Gynaecology
Thanjavur Medical College,
Thanjavur.

DECLARATION

I solemnly declare that this dissertation titled **"ANTENATAL ULTRASOUND AND POSTPARTUM HISTOPATHOLOGICAL STUDY OF PLACENTA IN HIGH RISK PREGNANCIES AND ITS CORRELATION WITH FETAL OUTCOME "** was done by me at department of Obstetrics and Gynaecology, Thanjavur Medical College during the year 2015 - 2018 under the guidance and supervision of Prof. Dr. R.RAJA RAJESWARI, M.D.,DGO., This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree in Obstetrics and Gynaecology (Branch -II)

Dr.R. VIDHYA

Place:

Date:

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TABLE OF CONTENTS

ABBREVIATIONS

GA: Gestational Age

MPT: Mean Placental Thickness

PIH: Pregnancy Induced Hypertension

GDM: Gestational Diabetes Mellitus

NICU: Neonatal Intensive Care Unit

APH: Antepartum Hemorrhage

INTRODUCTION

In a high risk pregnancy, the birth of a healthy child is a time of joy and expectation. The determinant of fetal growth and metabolism is the exchange between mother and fetus across the placenta. A well nourished new born therefore, is the best evidence of adequate placental functioning. The placenta acts as a major functional unit for the well being of the fetus.

Moosman defined placenta as a fusion of the fetal membranes to the uterine mucosa for the transfer of oxygen and metabolites between mother and fetal blood (Williams 24th Edition).

As the placenta grows and ages, the histological changes suggest an increase in the efficiency of transport to meet the metabolic requirements of growing fetus. Hence any insult to placenta during development stage quantification of the placental changes is essential to correlate the outcome in terms of the fetus.

According to Benirshcke (1961)[6] , the placenta is the most accurate record to infant's prenatal experience and before the advent of ultrasonography, its evaluation was possible only after delivery.

Ultrasound placentography was first described by Gottesfeld (1966)[15] while Grannum et al., (1979)[16] proposed grading system for the maturation of placenta occurring with advancing gestational period With the advent of ultrasonography, antenatal evaluation of placenta has become essential in high risk pregnancy, as fetal problems and neonatal outcome depend upon the status, growth and abnormalities of placenta.

The present work was undertaken to study the placental ultrasonography in relation to histological changes occurring in high risk pregnancy and its correlation with fetal outcome.

Placental study may help in future pregnancy. Structural derangement may be the only way to study the fetal environment.

HISTORICAL ASPECT

Vesalius (1955) correctly, illustrated the human placenta. Realduscolumbus in 1559 first introduced the latin word ‘ Placenta’, Aristotle 384 – 322 BC first used the word ‘Chorion’,

Harvey in 1651, set forth clearly the concept of fetal arterial and venous circulation in placenta. By the end of 17th century accurate concept regarding structure and functional significance of placenta and placental barrier was established.

In 1771, ‘Decidua’ was accurately described. In 1821, John and Lanter descrinbed ‘ Decidual basalis’, In 1832, Webber established the presence of ‘ Intervillous space’. In 1882, Langhans demonstrated that villi were covered by two layers of cells. In 1889, Habrecht introduced the terms ‘ Trophoblast’.

ULTRASONOGRAPHIC STUDY

Ultrasonographic placental grading was described in 1966. Original, bistable technique with placenta appearing in outline as white speckled areas with boundaries in the uterine wall and amnio – chorionic plate. Early descriptions only provided positional relationship with uterus. With the advent of gray scale / real time technique, variations in density, anatomy and positional relationships can be demonstrated.

Ultrasound evidence of the developing placenta can be seen as early as 6 weeks of gestation. It appears as an area of high – level echoes surrounding a border representing the developing gestational sac. The echoes represent the chorion frondosom, which develops into definitive placenta. From 12 weeks of gestation the structures of the placenta can be more clearly discrened. The 3 Structure are the

a. chorionic plate
b. substance of the placenta
c. basal layer

Changes in these structures represent the basis of placental grading. Grading of placenta forms a par of fetal bio – physical profile. Grannum's classification of placental grading.

Region	Grade			
	0	I	II	III
Basal Layer	No densities	No densities	Linear arrangement of small echogenic areas (basal stippling)	Larger and partially confluent echogenic areas
Placental substance	Finely homogenous	Few scattered echogenic areas	Linear echogenic densities (comma like densities)	Circular densities with central echo poor areas
Chorionic plate	Straight and well defined	Subtle undulations	Early demarcation of cotyledons in the direction of basal layer	Septation of cotyledons extending to the basal layer

The mean gestational age at which different placental grades appear and incidence of grade I placenta at term is shown in the following

Table

Mean Gestational Age in Weeks	Placental Grade	Incidence of grade I placenta at term (%)
31.3	I	40
36	II	45
38	III	5-15

Modified Classification of Placental Grading by Kazzi

The Standard classification of placental grading, assigns placental grades according to the most advanced portion of the placenta. For example if grade II changes are seen in one area, it is classified as grade II, irrespective of whether grade I or II exists. Hence, Kazzi and associates (1955) examined placenta of 230 high risk patients previously graded by Grannum's technique and proposed a modified classification system.

The mean gestational age at which different placental grades appear and incidence of grade I placenta at term is shown in the following

Table

Immature	**Intermediate**	**Mature**
Diagnose only when not grade III areas were seen, i.e., grade 0.1 or II according to Grannum	When only a portion was grade III, the remaining with grade I and II.	Only grade III areas were present.

Clinical Significance of Placental Grading ;

1. In post term pregnancy (>42 weeks) grade 0 or I does not exist. Only grade II or III configuration is found. With grade III Placenta abnormal fetal heart rate patterns have been noted.

2. I cases of Intra uterine growth retardation and Pregnancy induced hypertension, there is accelerated placental maturation. i.e. grade in < 27 weeks, grad II in <32 weeks and grade III in < 34 weeks.

3. Delayed maturation is associated with gestational diabetes. i.e, grade I in > 30 – 32 weeks.

PLACENTAL THICKNESS

The thickness of placenta is measured between cord insertion and lateral edge of the placenta. The following table shows the mean placental thickness associated with different placental grades.

mean thickness of placenta (cm)	grades of placenta
3.8	I
3.6	II
3.4	III

Clinical significance of placental thickness

1. Thickness of < 2.5 cm is associated with poor intrauterine environment (IUGR).
2. Thickness of > 4 cm is an early sign of fetal hydrops (upto 8 cm) diabetes mellitus and maternal syphilis.

In conclusion, acceleration of placental maturation and ultrasonographic grading leads to two to eight fold increase in fetal morbidity.

DEVELOPMENTAL STAGES OF PLACENTA

The human placenta is of haemochorioendothelial type.

Late day 6 to early day 8 post conception : Prelacunar stage of trophoblast : The blastocyst is partially implanted.

Late day 8 to day 9 : Early lacunar of Trabecular stage : The syncitiotrophoblast at implantation pole exhibits vacuoles – forerunners of the lacunar system.

Day 10 to day 12 : Late lacunar or trabecular stage : Implantation is complete. The syncitiotrophoblastic vacuoles fuse to form the lacunar system. First contact of lacunar system with eroded endometrial capillaries is the first sign of decidualization.

Day 13 : Early Primary villous stage (first free primary villi) : The syncitiotrophoblast forms the trophoblastic trabecular forerunners of stem villi. Trophoblastic out growth into the lacunae from the villous stem.

Day 15 to Day 16 : Early secondary villous stage : From implantation to anti implantation pole, the extra embroyonic mesenchyme from the chorionic cavity invades the villi forming the secondary villi.

Day 9 to Day 21 : Early tertiary villous stage : In villus, fetal capillaries develop.

Day 23 to Day 26 : Villous trophoblastic surface composed of outer syncitio and complete inner cytotrophoblast. Chorionic plate consists of fetal mesenchyme, cytotrophoblast and syncitio trophoblast. Intense mixing of decidual and trophoblastic cells from the basal plate. Tissue necrosis in the contact zone form the Nitabuch fibrinoid.

Day 29 to Day 49 : Mesenchymal villi show increased number of macrophages and stromal channels. Medium sized villi show reticular stroma of immature intermediate villi, Peripheral portions of villi in primary stage increase in size and ten undergo fibrinoid degeneration.

Day 43 to 70 (third month) : Around anti – implantation pole, the fibrinoid deposition initiate the formation of chorion laeve.

Fourth month : Continuous degeneration of villi at anti – implantation pole and proliferation of villi at implantation pole initiate the differentiation of chorionic sac into chorion and placenta.

The fetal vessels show concentric adventitial fibrosis to such an extent that about half of the arterial and venous adventitia fuse forming central villous core – first real stem villi.

Fifth Month : Stem villi increase in number.

Sixth Month ; Immature intermediate villi are transformed into stem villi and the large villi are completely fibrosed.

Seventh Month : In locally restricted areas, obviously in the periphery of villous trees, small group of grape – like, highly capillarized terminal villi develop. Cell columns surrounded by fibrinoid deeply invaginate into the basal plate. Syncitiotrophoblast covering the chorionic plate is replaced by a thin layer of fibrinoid which grows in thickness forming Langhan's stria.

Ninth Month : Large parts of syncitiotrophoblast of stem villi are replaced by fibrinoid. Superficial stroma shows increased number of fibroblasts. Central stem villi are highly fibrous. Majority of villi are intermediate and terminal villi.

Tenth Month : Mean placental weight increases from 400 to 470 grams. Increased number of terminal villi (40 % of total villous volume of the placenta). 30 to 40 % villous volume consists of intermediate villi. The amount of previllous fibrinoid is extremely variable.

GENERAL PATHOLOGY OF PLACENTA

MACROSCOPIC ABNORMALITIES OF PLACENTA

1. Development Abnormalities

a. Placental extrachorialis: Commonest development abnormality 30 % - H. Fox 2007)[10]. The Chorionic plate from which the villi arise is smaller than the basal plate and hence the transition from villous to membranous chorion takes place not at the edge but at same distance within the circumference of te fetal surface, thus leaving a ridge of villous tissue projecting beyoudn the chorionic plate.

b. Placenta Membranacea : All or most of the membranes are covered on their outer aspect by placental villi. Incidence - I in 20000 to I in 40000.

c. Accessory lobe ; Adjacent to the main placenta there may be found one or more accessory lobes of variable size. Incidence – 30 %.

d. Bilobate placenta : This consists of two approximately equal lobes which may be connected by a bridge of chorionic tissue or may be quite discrete from each other. Incidence – I in 350 (Earn 1951).

e. Fenestrate placenta : This a condition in which the central portion of a discoidal placenta is missing.

f. Ring shaped placenta : Also known as ' gride or annular placenta' The placenta is annular in shape and resembles a segment of a hollow cylinder.

2. Lesions Reducing Mass of Functioning villi :

a. Previllous fibrin deposition : Some degree of fibrin deposition around villi occurs in almost in all placentae, but in a proportion is sufficiently extensive to be macroscopically visible either as a firm white plaque or as area of irregular, whitish mottling. There is a low incidence of these plaques in placentae from eclamptic women and very low incidence of fetal hypoxic complication associated with the presence of this lesion (Fox 2007).

b. Infarction : A fresh placental infarct is well demarcated, dark red and moderately firm. Older lesions are hard, yellow white plaques. Small areas of infarction involving < 5% of villous parenchyma, are common

occurring in 25% of placenta from uncomplicated pregnancy and are of no clinical significance. > 10 % is associated with a high incidence of fetal hypoxia, growth retardation and Intra uterine Death (Kloosterman and Heridekoper 1954; Little 1960 ; Fox 1976 ; Naeye (1977)[30]. Infarction occurs against a background of a markedly abnormal maternal vasculature and restricted blood flow to placenta as a result of inadequate transformation of spiral arterioles into utero – placental vessels (Robertson et al., 1975 ; Robertson 1976, 1981).

c. Fetal artery thrombosis ; Seen macroscopically as a rough triangular area of pallor within the placental substance. Histologically, te stroma of these villi becomes fibrotic and fetal vessels undergo an obliterative sclerosis. Found in 4% of placentae from live births. Seen in fetus of mothers with hyper coagulable status in association with anticardiolipin antibodies and protein S deficiency (Kraus 1993 and Rayne and Kraus 1993).

d. Primary defect in placental growth : Physiological capacity of the placenta is related to its weight and the functional deficiencies of a small placenta well restrict fetal growth. Placental : fetal weight ratio has been considered to be of significance (Little 1961, Thomson et al 1969 ; Lemtis and Hadrich 1974, Motteir et al 1978). Placenta has a potential for further incremental growth, indicated by unduly large placenta as

found in pregnancies in high altitude, severe maternal anaemia and maternal heart failure (Clavero – Nunez 1963; Beischer et al 1970, Kruger and Arias Stella 1970 ; Agboola 1975; Godfrey et al 1991).

e. Haematomas and Thrombi

- Retroplacental haematoma : This haematoma lies between, and seperates the basal plate of the placenta and the uterine wall. They are found in 5% of all placentas though it is increased three fold in those from pre – eclamptic women (Fox 1978).
- Subamniotic haematoma : This occurs on the fetal surface of the placenta as a plum coloured tumefaction which lifts the amnion from the chorion, results from tearing of surface chorionic veins by excessive cord traction. Old haematomas tend to be associated with a low birth weight (Deans 1998).
- Marginal Heamatoma : This is crescentric lesion at one edge of the placenta. This type occurs in placenta which is implanted in the lower uterine segment, i.e., a lateral placenta praevia (Wilkin 1965).
- Intervillous thrombi : Lies approximately mid way between fetal and maternal surfaces. Measures 1 to 2 cm and are known as ‘ Kline’s haemorrhages. Found in 40 % of all placentas (Fox 1978). They can cause elevated maternal alpha fetoprotein ;levels (saffix 1988).

f. Calcification : Calcification is considered as an evidence of placental senescence or degeneration. The cause of calcification is unknown. Occurs most commonly in first pregnancies, low maternal age, high maternal socio – economic status and delivery during summer months increasing material serum calcium levels (Fox 2007).

HISTOLOGICAL ABNORMALITIES OF PLACENTA

a . Abnormalities of villous maturation and differentiation (Villous maturity) : A deficiency of terminal villi towards the end of gestation (Villous immaturity) is associated with high incidence of fetal growth retardation (Becker 1975, 1981).

b. Changes secondary to reduced maternal blood flow (Cytotrophoblastic proliferation) : There is an undue prominence and number of villous cytotrophoblastic cells together with irregular thickening of the trophoblastic basement membrane, which is probably an incidental by – product of cytotrophoblastic cell hyperplasia. Hence, the response of placenta to ischaemia is a reparative one.

c. Changes secondary to reduced fetal blood flow (Syncitial knots and Fibrosis) : These are seen in a group of villi, which while fully `oxygenated from maternal blood, have been deprived of their fetal

circulation by thrombosis of a fetal stem artery. such villi show stromal fibrosis and excess syncitial knots as seen in cases of prolonged pregnancies. their appearance in placenta as gestation progresses is a time related phenomenon, but is not a true aging change.

d. Abnormalities of unknown pathogenesis :

- Fibrinoid Necrosis : Appears as a small nodule of homogeneous, acidophilic, PAS – Positive material at one point in the villous trophoblast. Enlarges as fresh fibrinoid material to form a mass, attributed to immunological reaction within villous tissues.

- Villous edema : Its presence is correlate with increased water content of placenta (Barker et al 1994). It has been suggested that the increased size of placental villi decreases the capacity of the intervillous space and hence restricts maternal blood flow to the placenta. (Alvarez et al. 1972 ; kovalouszki et al. 1990). Naeye et al. (1983) also considered villous edema to be indicative of fetal hypoxia.

ANATOMY OF THE UMBILICAL CORD

Consists of 2 arteries and one vein, surrounded by Wharton's jelly which, in turn, is coated by one or more layers of amniotic epithelium. Wharton's jelly is made of mucous or myxoid tissue, composed of scanty, large, stellate cells, giving rise to collagenous fibres, which increase in number with gestational age and are concentrically arranged around the blood vessels.

The 'valves' of Hoboken are a peculiar feature of the umbilitcal cord arteries – Constructions of the lumen, occurring at varying intervals. Schare (1986) considers these as a transient phenomenon.

ABNORMALITIES OF UMBILICAL CORD

Length of the cord

The minimal length of the cord allowing normal vertex delivery at term has been calculated to be 32 cm. A too – short cord may lead to obstetric problems like mal presentation, abruptio placenta, intrafunicular haemorrhage and inversion of the uterus. Too long a cord, is likely to facilitate entanglement, prolapsed of the cord and thrombosis.

Placental Insertion of the Cord

The relation of the attachment of the Umbilical cord to the surface of the placenta is determined by the position of the inner cell mass in relation to the uterine wall. Marginal insertion gives rise to so called Battledore placenta. When the cord is implanted on the membrane away from the margin of the placenta it is called velamentous insertion.

Knots of the Cord

According to Spellacy and co – workers, the incidence of true – knotting is 1.1 % and is associated with 6.1 % of perinatal mortality.

Absent Umbilical artery

It is noted in about 1 % According to Benirschke, 20 to 40 % of all infants with one umbilical artery have associated Congential anomalies. The birth weight is likely to be lower than the normal.

Supernummary Umbilical Vessels

The possibility that accessory vessels found in the umbilical cord may be partial remnants of an incompletely regressed umbilical vein (Meyer et al ; 1969). Gupta et al (1993) found a positive correlation with inflammatory cell infiltration of the cord and a highly significant association with maternal smoking.

PATHOLOGY OF MEMBRANES

Amnion

Reflects the condition of infant's skin as it is continuous with the surface of the umbilical cord Therefore, reflects the epithelial disorders the infant and can itself respond to injury with inflammation.

Colour of the membrane

Opaque membrane is associated with acute or chronic infection. Meconium passage is uncommon prior to 38 weeks and increase after 40 weeks and is associated with increased perinatal morbidity and mortality. Meconium staining of the umbilical cord and membranes has recently been claimed to induce necrosis of umbilical vessels and cord (Altscyler et al, 1992 ; Bernischke, 1994)[2,6].

AIMS AND OBJECTIVES

1. To find out placental maturational changes (Grading and Thickness) ultrasonographically in high risk pregnancy.

2. To find out the histopathological changes of placenta in high risk pregnancies including pregnancy induced hypertension, post term pregnancy, Diabetes mellitus complicating pregnancy, preterm pregnancy and pregnancy complicated by antepartum haemorrhage.

3. To correlate above findings with fetal outcome (fetal distress, mode of delivery, birth weight, Apgar at 5 minutes, still births and perinatal deaths).

4. To compare the results of normal and high risk pregnancy groups.

REVIEW OF LITERATURE

All placenta should be examined grossly. This can be performed by the healthcare providers present at the time of birth and can be done with a basic knowledge of placental anatomy, and an understanding of the abnormalities and variations that affect the placenta.

Pathological findings in the placenta may be useful in understanding adverse outcome in one of two ways. Firstly, the placenta per se can be abnormal and thus contribute to the adverse outcome. Secondly, the placenta may harbor abnormalities that may point towards the presence of an adverse intrauterine environment.

Examination of the placenta performed in the labour room provides information that may be important to the care of both mother and infant.

During the examination, the size, shape, consistency and completeness of the placenta, the presence of accessory lobes, placental infarcts, hemorrhagic areas, tumours and nodules should be noted. The umbilical cord should also be assessed for length, insertion, number of vessels, knots and the presence of Wharton's jelly.

The color, luster and odor of the fetal membranes should be noted. Tissue may be retained because of accessory lobe of the placenta or because of placenta accrete, placenta increta or placenta percreta.

Numerous common and uncommon findings of the placenta, umbilical cord and membranes are related with abnormal fetal development and perinatal morbidity. The placenta should be sent for pathologic evaluation if an abnormality is detected.

Examination of the placenta can yield information that can help in the immediate and later management of mother and infant. This information may also be essential for protecting the attending physician in the event of an adverse maternal or fetal outcome.

Although some experts say that all placenta should be examined by a pathologist, most hospitals do not mandate this examination. Instead, the delivering doctor is usually responsible for determining when pathologic analysis is necessary.The examination of normal placenta and most abnormal placentas can be done within one minute. Universal assessment of the placenta in the labour room, with documentation of findings and submission for pathologic evaluation based on abnormal findings or certain clinical indications, is standard medical practice.

Boyd and Hamilton (1970) William 24 edition. 97 : The placenta at term is. on average, 185 mm in diameter and 23 mm in thickness, with an average, volume of 397 ml and weight of 508 g ; but these measurement s vary widely. There are multiple shapes and forms fo the human placenta and a variety of types of umbilical cord insertions.

Wiswell and associates (1990) Williams 24 edition, ; Incidence of meconium staining in 12 % of 1,75,000 live born infants.

Benirschke and Kaufmann (2000) Williams 24 edition, : Identified visible meconium staining in 18 % of 13, 000 consecutive placentas.

Morrisan JE, ' Fetal & Neonatal Pathology' , 2nd Edn. Butterworths : :London, 1963. At term the placental – fetal ratio lies between 1 : 6 to 1 : 8[29].

Hansen A, Collins MH, Genset D, et al. Pediatr Dev Pathol 2000 Sep – Oct ; 3 (5) 419 -30 (ISSN : 1093 – 5266) : Examination of the VLW Infant's placenta provides insight into the etiology and management of VLBW and preterm deliveries[20].

Naeye RL Human Pathology 18 : 387 - 392, - 1987. This study attempted to determine if placental size has implications of fetal and

neonatal health and subsequent childhood growth and development. 38,351 placentas were trimmed and weighed in a standardized way. Over weight placentas, largely a result of villous edema, were associated with following neonatal evidences of acute neonatal hypoxia. Low Apgar scores, respiratory distress syndrome, neurologic abnormalities and neonatal death. Neurologic abnormalities were 33 % more frequent when placentas had been overweight than they had been normal weight[30].

Anjali RM, Leela RK and Prabhatkumar SP FOGSI J. 2 : 294 – 300, April 1985. Subject – 100 high risk cases. The counts of syncitial knots were found to range between 0-39 % in normal control group while between 30 – 59 % in all other groups. Cases of fetal distress (46. 8 %) and still births (62. 5%) in were showing syncitial knot count of grade II and grade III respectively. Leucocytic infiltration of umbilical cord was found in 2.3 % of toxaemia and 4 % of normal group. No correlation with fetal hypoxia was noted. Villi showing fibrinoid necrosis of more than 5 % were found in cases of toxaemia (59.5%) and diabetes (60 %) . The count was between 0-5 % in all uncomplicated cases and in most cost with IUGR (70 %). Incidence of fetal distress was found to be more in toxaemia (40. 4%) Low Apgar

score was observed more in toxaemia (38. 09%) and intrauterine growth retardation (30 %) cases. Percentage of still birth in toxaemia was 11.9 % and in diabetes 20 % with placentas weighing less than 300 gms, fetal distress (43.3%) and low APGAR scores (43.3 %) were observed[3].

Marianne S, Chakrawarthi RN, and Devi PK : Journal of Obstetric Gynaecology. India, 26 : 216 – 21, 1976, 53 high risk subjects. Counts of syncitial knots were found to range between 30 – 50 % in majority of normal cases. Malkani, Bhasin PK and Bhasia : J. Obstetrics. Gynaecology. India, 18 : 666, 1968. Suggested and excess of over 30 % count to be indicative of excessive aging due either to post – maturity or a disease stage causing placental insufficiency. About 93 % of the normal cases showed < 7% stromal fibrosis (> 10%). The Rh incompatibility group showed moderate fibrosis – (6 – 10%) in 28% of the placentas, although almost 71 % had mild fibrosis (6%) . In the post mature group, stromal fibrosis of > 10 % was observed in 25 % of the cases. In the normal group only 16.6 % showed fibrinoid necrosis of < 3 % 42 % of Rh incompatible group and 34 % of toxaemic group showed > 6 % fibrinoid material[28].

Neerja, Bal Manjit S and Chandra P : Journal of Obs. Gynae. India, 5 : 603 - 607, October 1999, Normal Subjects – 50 and high risk subjects 50. Placental weight in study group was found to be less than 300 gm in 22 % of cases, 300 to 400 gm in 50 % and > 400 gm was found in 28 % of cases. None of the patient in control group had placental weight less than 300 gms. instead all cases had more than 400 gms. Syncitial knot formation in > 30 % of villi was found in 66 % of patients in study group, while only 6 % of control group. 12% had 0.3 to 3 % stromal fibrosis and 32 % in study group had > 3% stromal fibrosis of villi. 1.5 % of fibrinoid necrosis was observed in 36 % of of study group, 5 to 10 % in 40 % and > 10 % in 24% of cases. Leucocytic infiltration was observed in 30 % of cases of study group compared to none in normal group. Fetal distress was observed in 34 % of cases of study group as compared to 12 % in control group[32].

Karla VB, Aggarwal A and Sareen PM : FOGSI J. Obstet. Gynec. India, 35 : 7 to 11, 1985. Histopathological changes in placenta in toxaemia in pregnancy. Syncitial knot counts per 100 vili were found to be significantly increased in cases of severe pre – eclampsia. There was increased incidence if stromal fibrosis and fibrinoid necrosis in toxaemia cases[23].

Godbole PV, Mehendale SS and Vasanti Le Le : J. Obset. Gynee. India, 138 : 406 to 409, 1988. 100 High Risk subjects. On Gross examination of the placenta from cases of IUGR 35 % showed calcification. 41 % showed infarction and 27 % showed fibrin deposition. 70.2% showed presence of leucocytic infiltration and syncitial knots were present in 88 % of placentas[14].

Bandana, D. Dutta D, Chakraborty S, and Nath P : FOGSI Journal of Obstet. Gynaec. Ind. 46 : 40 to 46, Feb. 1996. 20 normal subjects and 80 hypertensive subjects. Weight of 25 placentas were below 300 gms at term, whereas none of the placenta was below 300 gms in normotensive control group. The shape of placenta was normal in 85 % of normotensive and 65 % of hypertensive cases. The insertion of the cord was central in 80 % cases of control group and 56.25 % in hypertensive group. The incidence of placental infarction involving > 5% of parenchyma in hyoertensive disorders (50 %) and its was absent in 75 % of normal cases and when present, it involved < 5 % of the total surface area[5].

Avasthi K, Midha U, Sabharwal, B.D. and Kirna Devi : J. Obset . Gynaec. India, 41 : 317 to 23, 1991. 125 placentas from abnormal and 100 from normal subjects. 48 % showing syncitial knots in 30. 59 % of

villi and 80 % cases syncitial knot formation in 60 . 89 % of villi had fetal distress. Incidence of still birth was 22.44 % in group showing syncitial knots 30. 59 % of villi and 40 % in group with syncitial knot in 60. 89 % of Villi. The incidence of fetal distress was highest in post – maturity (60%) and Rh incompatibility (60 %) group followed by toxaemia (35 %) Low APGAR scores were more frequently observed in cases with toxaemia (25 %) percentage of still births were highest in Rh incompatibility group (20 %) followed by toxaemia (12.5 %)[4].

Wolf H, Dosting H and Jreffers PE : Am J. Obstet. Gynaecol. 16 : 121- 129, 1989. has given evidence that fetal growth retardation is preceded by reduced placental volume growth[52].

Gerisson RT, Oystow SA and Patel NG : Br J. Obst. Gynaec. 92 : 46 – 53, 1985. Found the growth rate of placental volume reduces after 30wks and even falls towards term[13].

Yin L, Liu Y, Ma H Tianjin Medical University, Second Hospital : Chung Hua Fu Chan Ko Tsa Chin 1998 July ; 33 (7) : 415 – 8. After term pregnancy the placental function is gradually lowered especially in prolonged pregnancy. The situation of the placenta, amniotic fluid and fetus should be monitored after 40 gestational weeks[53].

Jauniaux E, Moscoco G, Champbell S, Gibb O, Driver M and Nicoladides KH : Correlation of ultrasound and pathologic findings of placenta. Euro Journal of Obset. Gynec. Reprod. Biol 1990. Concluded that placental thickness is not diagnostic of any particular condition but can contribute to the management of fetus at risk. Thick placenta of > 4 cm can be an early sign of developing fetal hydrops of various causes as in uncontrolled maternal diabetes[22].

Petrucha RA and Lawrence D : Am J. Obset. Gynec. 144 : 733 to 735, 1982. The study suggest that placental grade is a function of gestational age and no conclusion can be reached concerning the relation of these grade III placentas to pulmonary maturity[35].

Kazzi GM, Thomas LG, Mortimer GR et al : Am. J. Obset. Gyneac. 148 : 54 – 58, 1984. In their prospective study of 230 term and preterm complicated subjects studied the relationship between placental grading, fetal lung maturity and neonatal outcome in normal and complicated pregnancies, suggested that t presence of grade III placenta is affected by both gestational age and pregnancy complications and that a grade III placenta in a reliable indicator of fetal lung maturity.

Kazzi GM, Thomas LG, Robbert JS and Nadya Jk : Am J. Obset & Gyne. 1145 : 733 – 737, 1986. 224 subject examined sonographically

within 7 days of delivery. The presence of a grade III Placenta followed by the delivery of a SGA infant in 59 % of cases and that 62 % of SGA infants can be correctly identified.

Harman CR, Manning FA, Stearns E and Marisson : Am J. Obset. Gynae. 143 : 941 – 43, 1982. The correlation of ultrasonic placental grading and fetal pulmonary maturation in 573 pregnancies found that grade III plancenta was associated with immature L / S ratio in 8 to 42 % of cases[21].

Roy B : J. Obset. Gynaec. 44 : 343 to 48, 1994. Clinical Significance of placental grading by sonor. 50 normal and 115 high risk subjects. In normal cases placental grading had a definite correlation with gestational age, being higher with advancing pregnancy. In high risk group, placental maturity was accelerated, being higher than normal pregnancy of similar gestational age. Cases of diabetes and Rh incompatibility (16 %) however. showed delayed (100%) placental maturity and lower grades were found even in cases approaching term. Placental maturity showed a direct correlation with pulmonary maturity. RDS never developed when placenta had shown grade II or III maturity[41].

Quinlan RW, Amelia CC : Am J. Obset. Gynae. 142 ; 110 – 111 1982, The association of preterm appearance of grade III changes in the placenta in complicated pregnancies suggest that the changes are associated with premature senescence of placenta. A high incidence (78 %) of prenatal problems were found in association with grade III placenta[38].

Quinlan RW, Amelia CC, William C, Bahi and Magdeline MR : AM J. Obset. Gynae. 144 : 471 – 473, 1981. In the 1 Year period of time between January 1, 1981 and December 31, 1981, 1936 obstetric ultrasound, the grade III placental changes occur with increased frequency in post mature pregnancies. 78 % of pregnancies with preterm grade III changes were associated with significant perinatal problems like : hypertensive complications of pregnancy, IUGR, fetal distress and intrapartum bleeding.

Gasst MJ and William Ott : AM J. Obstet. Gynaec. 146 : 464 to 465, 1983. Reports suggested that there is a 100% correlation between neonatal lung maturity, mature amniotic fluid, concentration of surfactant and the appearance of a grade III placenta[12].

Grannum P, Berkowitz RL and John HC : Am J. Obset. Gynaec. 133 : 915 to 922, 1973. 129 subjects Mature L / S ratio (2.0) were found in 68 % of Grade. 1, 88 % of Grade II and 100 % of Grade III

Placentas. These results suggest correlation between maturational changes of the placenta as seen by ultrasound and fetal pulmonic maturity as indicated by L/s ratio.

Veena A and Sapna J : Placental grading by ultrasonography : J. Obstet. Gynaec. India , 50 : 59 – 62, 2000. 125 normal and 125 high risk pregnancy cases after 28 wks. Placental grading advanced with the gestational age in both low risk and high risk groups, but found to be accelerated in cases of IUGR and hypertension, with a delay in maturation in cases of Rh incompatibility and diabetes mellitus. There is no definite relationship between low birth weight and low APGAR scores, but there was a direct correlation with pulmonary maturity. There was no development of RDS when placenta was grade III[45].

Tewari K, Tyagi SP, Saxena K, Usmanai F and Begum R : ultrasonographic and histological study of placenta in abnormal pregnancy cases J. Obstet Gynaec. India, 47 ; 199 – 26, 1977. 49 high risk subjects. The mean gestational age for appearance of placental grade I, II and III was significantly earlier in cases of IUGR, while it was only significantly earlier in grade I and II in toxaemia. 70% of cases with preeclampsia had grade III changes prior to delivery as compared to 40 % of normal case. In cases of prolonged pregnancy, gestational age for appearance of different placental grades was similar

to that of normal pregnancy, while 100 % of cases with diabetes showed delay in appearance of grade I changes only and none had grade III changes even at 38 weeks. In Rh incompatibility, appearance of different grade changes (50 %) were almost similar to normal pregnancy. There is an increase in syncitial knot count , fibrinoid necrosis and fibrosis in grade II Placenta of pre – eclampsia and prolonged pregnancy as compared to grade II and grade III normal placenta[43].

Tewari K, Tyagi SP, Saxena K, Usmani F and Begum R : J. Obstet. Gynaec. 45 : 440 – 44, August , 1995. 20 normal subjects. The mean placental thickness was found to be maximum between 28 to 32 wks of gestation and a progressive decrease was noted till term.

Mean gestational age	**Grade**
31.2 + 1.92 wks	I
35.6 + 1.6 wks	II
37.5 + 1.2 wks	III

Grade	Mean placental thickness (Single + serial scan)
I	49. 7 + 5 .4mm
II	40 + 3.4 mm
III	34.2 + 3. 6 mm

Fibrinoid necrosis, vasculo – syncitial membrane changes and stromal fibrosis were increased significantly in grade III as compared to grade II placenta, while syncitial knot count increased insignificantly.

MATERIALS AND METHODS

1. Stratified random selection of 50 cases of high risk pregnancy [Hypertension – 10 , preterm labour – 10 cases, diabetes mellitus – 10 , antepartum haemorrhage – 10 and post term pregnancy – 10 and 50 cases of normal pregnancy of gestational age more than 28 weeks, from RMH, THANJAVUR MEDICAL COLLEGE, THANJAVUR. (from june 2016 to july 2017).
2. Ultrasonographic placental study was done [grading according to Grannum's grading system (1979) and thickness measured from site of cord insertion to the margin of the placenta], with in one week prior to delivery.
3. Placentas from high risk and normal pregnancies were collected and fixed with 1 % formalin. Placental cross sectioning was done through the entire thickness beginning from one to the other margin at 3 to 4 cms distance. 3 bits, one from normal and two from abnormal areas were taken for histopathological examination.

4. Macroscopic study of placenta included the following :

- shape of the placenta
- weight in grams
- size, diameter in centimetres

Examination of the umbilical cord :

- cord length in centimetres
- number of cord vessels
- anomaly of the cord

5. Macroscopic study of placenta included the following :

Syncitial knots (graded according to the percentage of area involved)

Fibrosis (graded according to the percentage of area involved)

Fibrinoid necrosis (graded according to the percentage of area involved)

Leucocytic infiltration

Infarction (in percentage of area involved)

Calcification (in percentage of area involved)

6. For Fetal outcome the following parameters were studied :

- Fetal distress
- Mode of delivery
- Birth weight (gms)

- Apgar score at 5 minutes
- Still births
- Perinatal deaths

INCLUSION AND EXCLUSION CRITERIAS:

- **EXCLUSION CRITERIA:**
  - G.A < 28 weeks.
- **INCLUSION CRITERIA:**
  - Hypertension.
  - Preterm labour.
  - Diabetes mellitus.
  - Antepartum Hemorrhage.
  - Post term pregnancy.
  - Normal pregnancy of G.A > 28 weeks.

Table 1: Distribution of complications in the high risk group of the study.

S. No	High risk factor	Primi gravida (n=25)	Percentage (%)	Multi gravida (n=25)	Percentage (%)	Total (n=50)	Percentage (%)
1	Antepartum Hemorrhage	5	20	5	20	10	20
2	Gestational Diabetes mellitus	4	16	6	24	10	20
3	Gestational Hypertension	6	24	4	16	10	20
4	Post dated	4	16	6	24	10	20
5	Preterm	6	24	4	16	10	20

Data are expressed as absolute number with percentage

Figure 1: Distribution of the complications amongst primi gravida in the high risk group of the study.

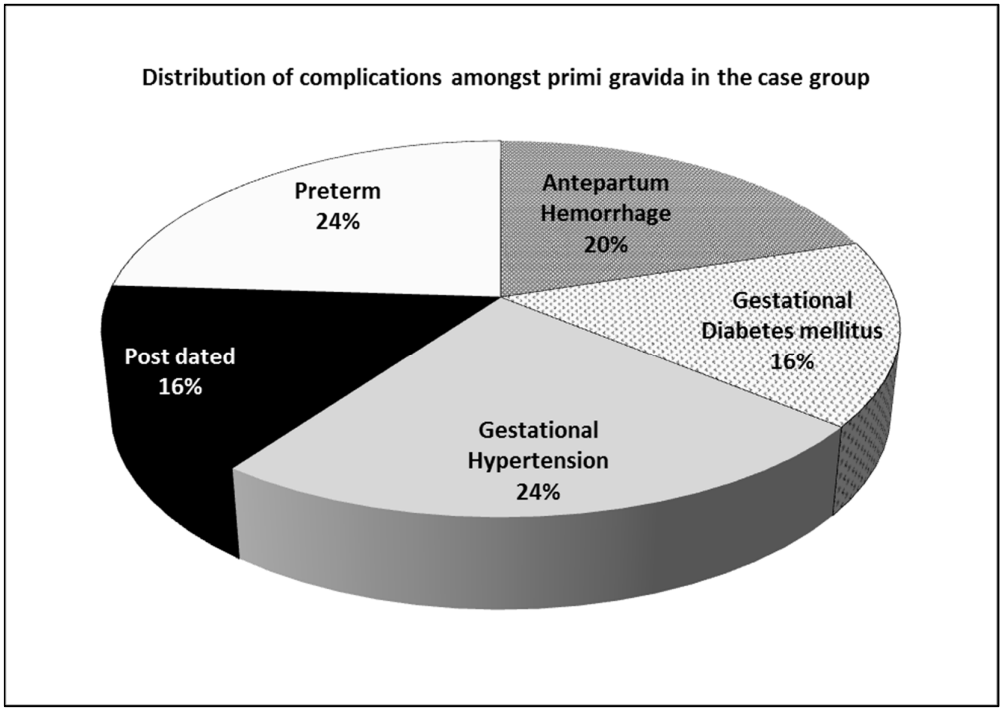


Figure 2: Distribution of the complications amongst multi gravida in the high risk group of the study.

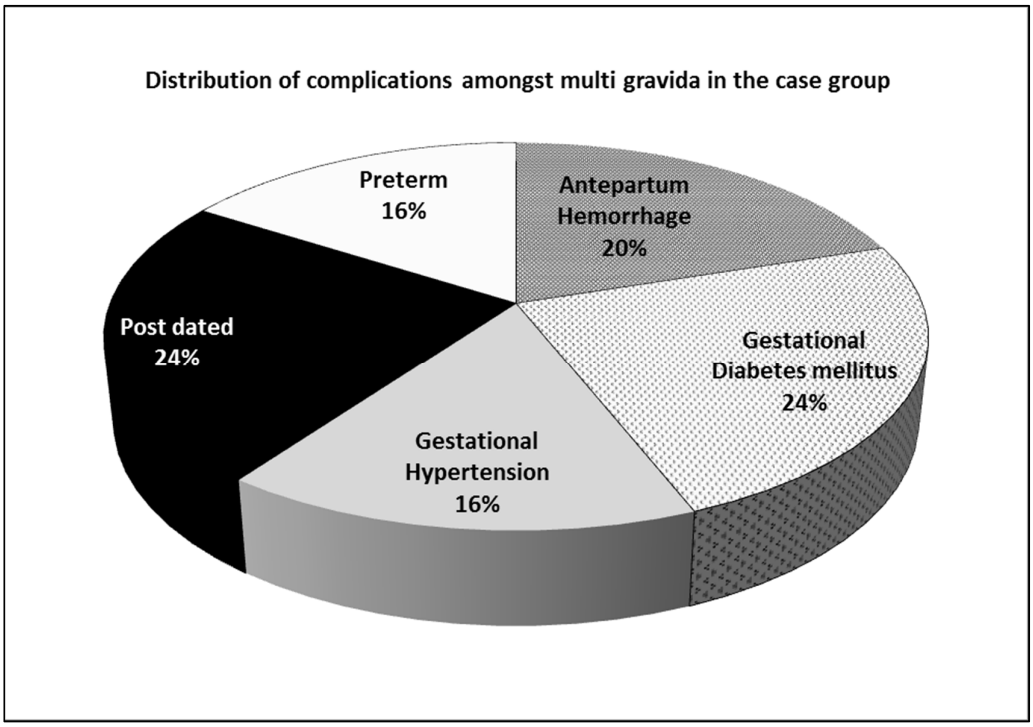


Table 2: Comparison of maternal age between the high risk group and normal group in the study population.

S. No	Parameter	Normal group (n=50)	High risk group (n=50)	P value	Statistical test
1	Age in years	25.2 ± 3.4	25.6 ± 3.7	0.539 (NS)	Unpaired 't' test

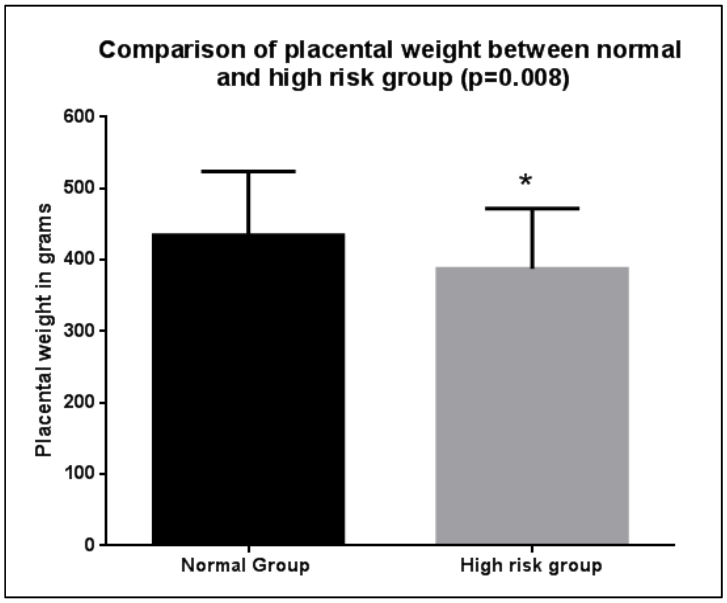
Data are expressed in mean ± SD. n= 50 in each group. P<0.05 is considered statistically significant and unpaired 't' test was used to find the statistical difference. Both the groups are comparable with respect to the maternal age in years.

Table 3: Comparison of maternal placental weight between the normal and high risk groups.

S. No	Parameter	Normal group (n=50)	High risk group (n=50)	P value	Statistical test
1	Placental weight in grams	434.6 ± 89.1	387.8 ± 84.3	0.008*	Unpaired 't' test

Data are expressed in mean ± SD. n= 50 in each group. * indicates $P<0.05$ and considered statistically significant. Hence high risk group has lower placental weight than the normal group patients.

Figure 3: Comparison of maternal placental weight between the normal and high risk groups.



Data are expressed as mean with standard deviation. The length of the bar in the vertical bar diagram represents mean and error bars represent standard deviation. * indicates p value <0.05. Unpaired 't' test was used to find the statistical significance.

Table 4: Comparison of maternal placental weight between the normal and high risk groups based on the parity.

S. No	Parameter	Normal group (n=50)			High risk group (n=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	P value	Primi Gravida (n=25)	Multi Gravida (n=25)	P value
1	Placental weight in grams	432.6 ± 85.8	436.3 ± 93.5	0.885 (NS)	392.8 ± 77	382.8 ± 92.4	0.679 (NS)

Data are expressed in mean ± SD. $P<0.05$ is considered statistically significant and unpaired 't' test was used to find the statistical difference. When based on parity there is no significant difference in the placental weight.

The mean placental weight amongst primis, 23 cases was 432 and multis was 436.9 in the normal group.

The mean placental weight amongst primis , 25 cases was 392.8 and multis was 382.8 in the high risk group.

However placental weight of less than 300 gms is mainly noted in the high risk group.

Table 5: Comparison of size of the placenta in cm between the normal and high risk groups.

S. No	Parameter	Normal group (n=50)	High risk group (n=50)	P value	Statistical test
1	Placental size in cm	16.2 ± 3.4	16.5 ± 4.6	0.712 (NS)	Unpaired 't' test

Data are expressed in mean ± SD. n= 50 in each group. Both the groups are comparable in respect to the placental cord length.

The mean placental size in the normal group is 16.2 cm and the mean placental size in the high risk group is 16.5 cm where there is no significant difference.

Table 6: Comparison of placental size in cm between the normal and high risk groups based on the parity.

S.No	Parameter	Normal group (n=50)			High risk group (n=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	P value	Primi Gravida (n=25)	Multi Gravida (n=25)	P value
1	Placental size in cm	15.7 ± 3.8	16.5 ± 3.05	0.38 (NS)	16.3 ± 4.8	16.9 ± 4.3	0.785 (NS)

Data are expressed in mean ± SD. $P<0.05$ is considered statistically significant and unpaired 't' test was used to find the statistical difference.

There is no significant difference in the placental size when compared with the parity

Table 7: Distribution of shape of placenta in normal and high risk group with respect to the parity.

S. No	Shape of the placenta	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	Discoid	21(92%)	27 (100%)	48 (96%)	24 (96%)	23 (92%)	47 (94%)
2	Bilobed	1 (4%)	0 (0%)	1 (2%)	0 (0%)	0 (0%)	0 (0%)
3	Club shaped	1 (4%)	0 (0%)	1 (2%)	1 (4%)	2 (8%)	3 (6%)

Data are expressed as absolute number with percentage.

Number of cases with abnormal placental shape(club shaped and bilobed) was 4.

There was only one case with abnormal placental shape in the normal group and whereas there are 3 cases in the high risk group with the abnormal shape..

Table 8: Frequency distribution of grading of syncytial knots in the umbilical cord between the normal and high risk group in respect to the parity.

S. No	Grading of the syncytial knots	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	Grade I	7 (30.4%)	8 (29.6%)	15 (30%)	3 (12%)	3 (12%)	6 (12%)
2	Grade II	13 (56.5%)	17 (63%)	30 (60%)	11 (44%)	13 (52%)	24 (48%)
3	Grade III	3 (13%)	2 (7.4%)	5 (10%)	11 (44%)	9 (36%)	20 (40%)

Syncytial knots in high risk group:

No of cases with syncytial knots grade I(0- 29%) were 3 cases in PIH, 1 case in post dated, 1 case in preterm,1 in APH and nil in diabetes complicating pregnancy.(total- 6 cases)

Syncytial knots gradeII(30-59%) were seen in 5 cases of PIH, 5 cases of GDM, 6 CASES of preterm, 4 cases of postdated, and 4 cases of APH.(total- 24 cases)

Syncitial knots grade III(60- 90%) were seen in 1 case of PIH, 6 cases of GDM, 3 cases of PRETERM, 5 case of postdated and 5 cases of APH. (total- 20 cases).

There was no case in high riskgroup with grade IV(90%) Syncitial knots.

Syncytial knots in normal group:

no of cases with grade I are 15 cases, grade II are 30 cases, grade III are 5 cases and nil in grade IV.

SYNCITIAL KNOTS OF GRADE III are more in the high risk group[40%] when compared to the normal group[10%].

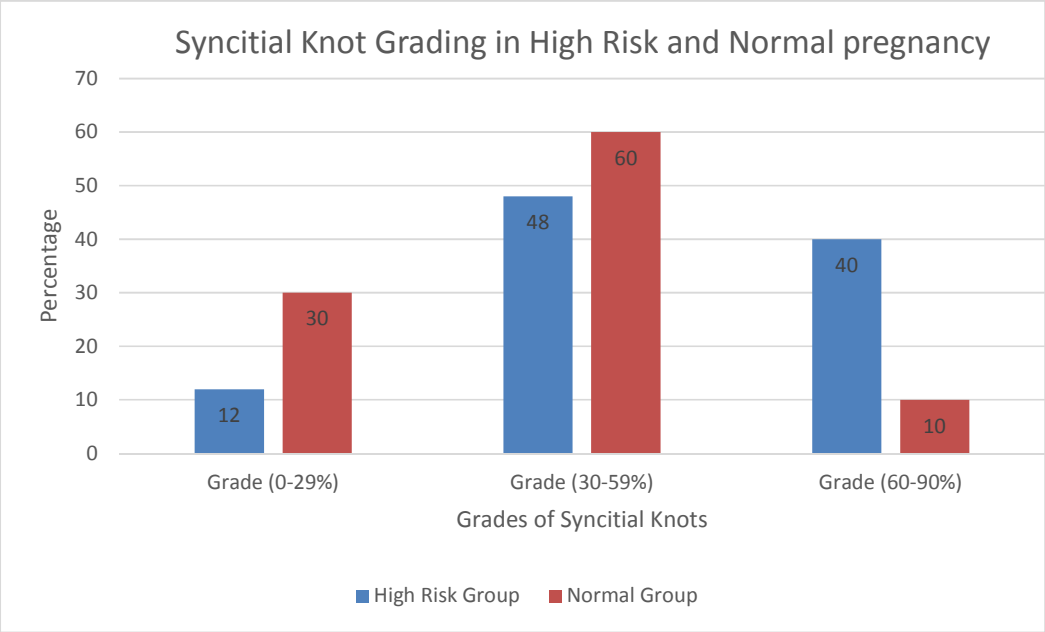


Table 9: Frequency distribution of grading of fibrosis in placenta between the normal and high risk group in respect to the parity.

S. No	Grading of placental fibrosis	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	Grade O	16 (69.6%)	20 (74.1%)	36 (72%)	18 (72%)	13 (52%)	31 (62%)
2	Grade I	4 (17.4%)	3 (11.1%)	7 (14%)	2 (8%)	4 (16%)	6 (12%)
3	Grade II	3 (13%)	4 (14.8%)	7 (14%)	5 (20%)	8 (32%)	13 (26%)

Fibrosis in high risk group:

No of cases with no fibrosis are- 6 cases of PIH, 4 cases of GDM, 8 cases of preterm, 6 cases of postdated, 7 cases of APH totally 31 cases(62%).

Grade I fibrosis(0.3-3%) were seen in 1 case in PIH, 3 cases in GDM, 1 case in preterm, 1 case in post dated and nil in APH. (Total- 6 cases[12%])

Grade II fibrosis (>3%)were seen in 2 cases of PIH, 4 cases of GDM, 1 case of preterm, 3 cases of postdated, and 3 cases of APH(total 13 cases[26%]).

Fibrosis in the normal group:

Grade 0 fibrosis in 36 cases[72%], grade I in 7 cases[14%] and grade III in 7 cases[14%].

Hence fibrosis of grade II is more in the high risk group[26%] when compared to the normal group[14%].

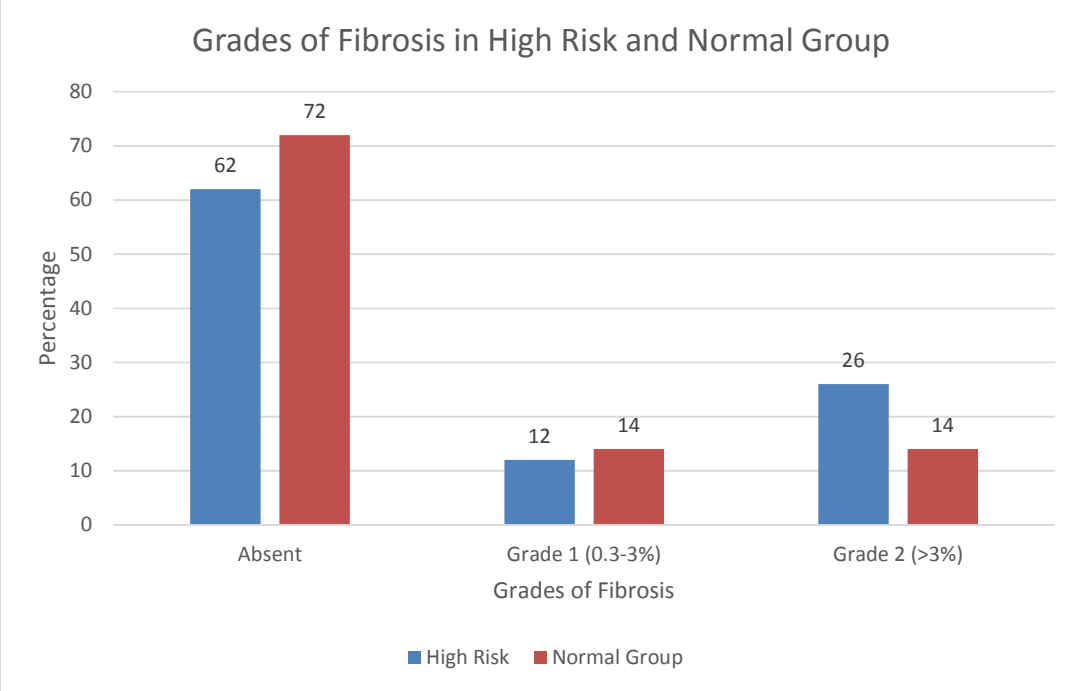


Table 10: Frequency distribution of grading of fibrinoid necrosis in placenta between the normal and high risk group in respect to the parity.

S. No	Grading of placental fibrinoid necrosis	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	Grade O	2 (8.7%)	3 (11.1%)	5 (10%)	0(0%)	0(0%)	0(0%)
2	Grade I	17 (73.9%)	18 (66.6%)	35 (70%)	11 (44%)	12 (48%)	23 (46%)
3	Grade II	4 (17.4%)	6 (22.3%)	10 (20%)	11 (44%)	9 (36%)	20 (40%)
4	Grade III	0(0%)	0(0%)	0(0%)	3 (12%)	4 (16%)	7 (14%)

Fibrinoid necrosis in the high risk group:

Grade I(0-5%) were seen in 2 cases of PIH, 4 cases of GDM, 7 cases of preterm, 5 cases of postdated, and 5 cases of APH. (total- 23 cases[46%]).

Grade II(5-10%) were seen in 6 cases of PIH, 3 cases of GDM, 3 cases of preterm, 4 cases of postdated, and 4 cases of APH. (Total – 20 cases[40%]).

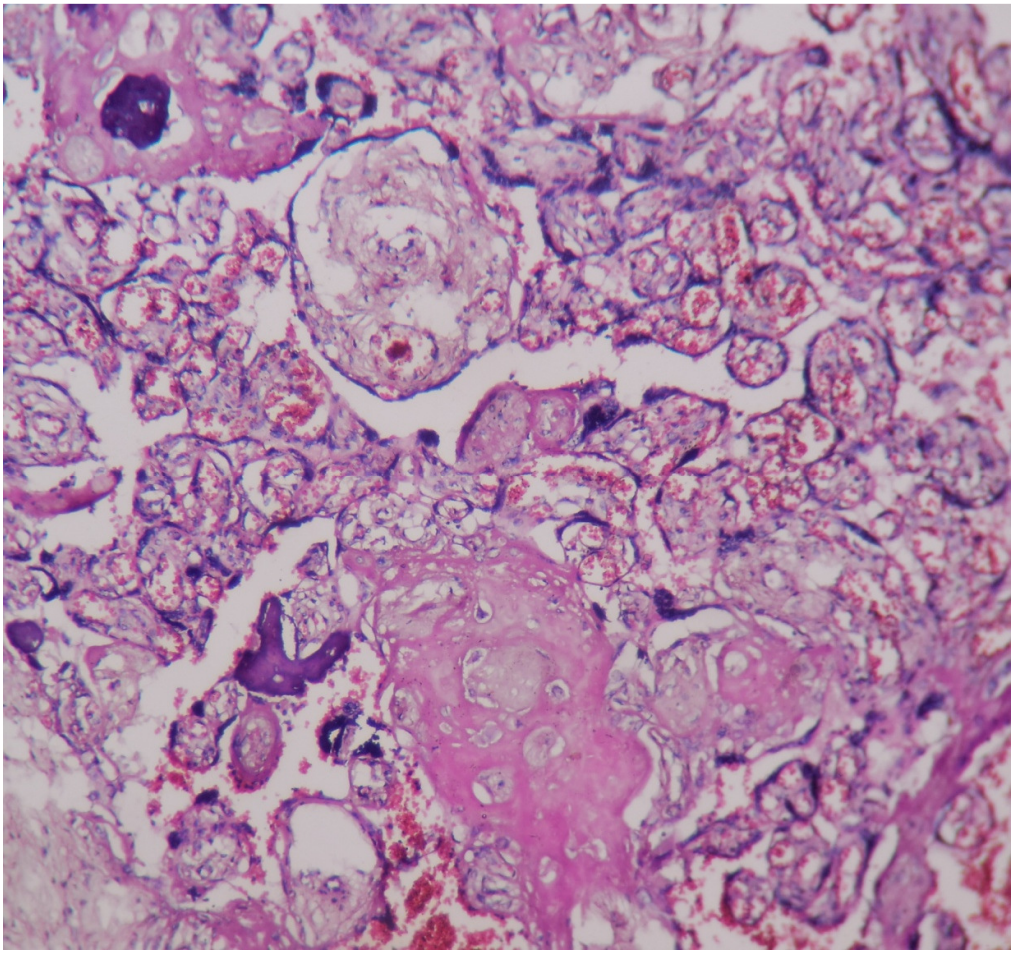
Grade III(>10%) were seen in 2 cases of PIH, 3 cases of GDM, 1 case of preterm, 1 case of postterm and nil in APH, (total- 7 cases[14%])

Fibrinoid necrosis in the normal group:

No fibrinoid necrosis in 5 cases, grade I in 35 cases, grade II in 10 cases, and nil cases in grade III.

Hence fibrinoid necrosis with grade II and grade III are more in high risk group than normal group.

Syncitial knots



FIBRINOID NECROSIS

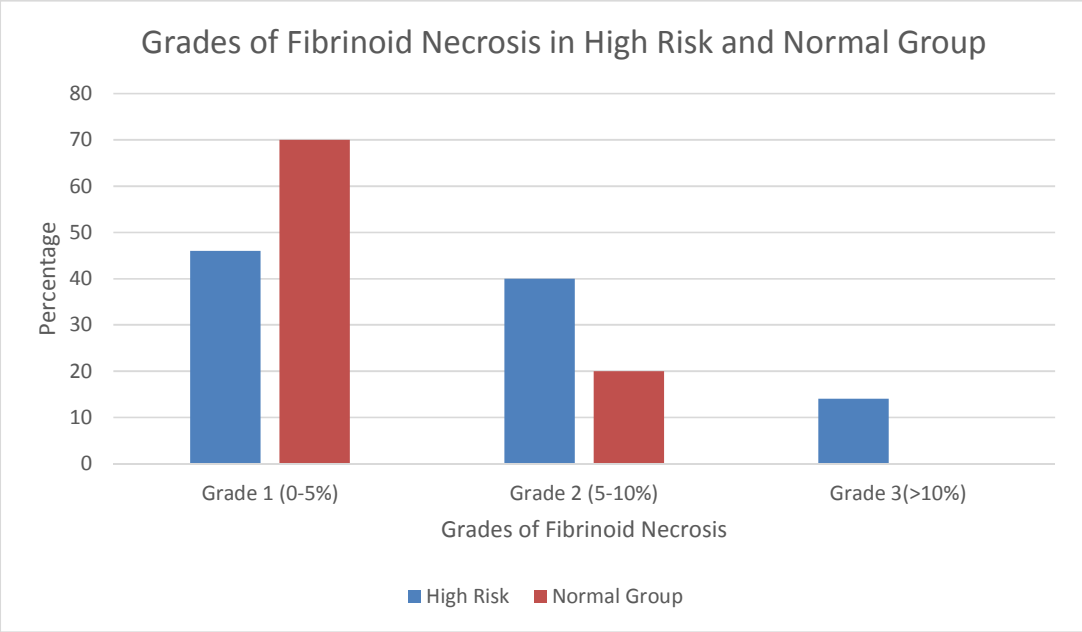

Grades of Fibrinoid Necrosis in High Risk and Normal Group
80
70
60
50
40
30
20
10
0
Percentage
Grade 1 (0-5%)
Grade 2 (5-10%)
Grade 3(>10%)
Grades of Fibrinoid Necrosis
High Risk
Normal Group

Table 11: Comparison of placental calcification score between the normal and high risk groups.

S. No	Parameter	Normal group (n=37)	High risk group (n=50)	P value	Statistical test
1	placental calcification score	3.27 ± 2.5	3.9 ± 5.7	0.485 (NS)	Mann Whitney U test

Data are expressed in mean ± SD. n= 37 in normal group (calcification was not seen in 13 subjects in normal group).

Calcification was present in 6 cases of PIH, 9 cases of GDM, 8 cases of postdated, 6 cases of APH and only in 4 cases of preterm. Calcification was absent in 17 remaining cases in the high risk group.

Whereas in normal group calcification was present in 37 cases and absent in 13 cases.

Hence there is no significant difference in the calcification score between high risk and the normal group.

Table 12: Comparison of placental calcification score between the normal and high risk groups based on the parity.

S. No	Parameter	Normal group (n=50)			High risk group (n=50)		
		Primi Gravida (n=18)	Multi Gravida (n=19)	P value	Primi Gravida (n=25)	Multi Gravida (n=25)	P value
1	placental calcification score	3 ± 1.9	3.5 ± 3	0.827 (NS)	3.3 ± 3.9	4.5 ± 7.09	0.964 (NS)

Data are expressed in mean ± SD. $P<0.05$ is considered statistically significant and unpaired 't' test was used to find the statistical difference

Table 13: Frequency distribution of placental calcification between the normal and high risk group in respect to the parity.

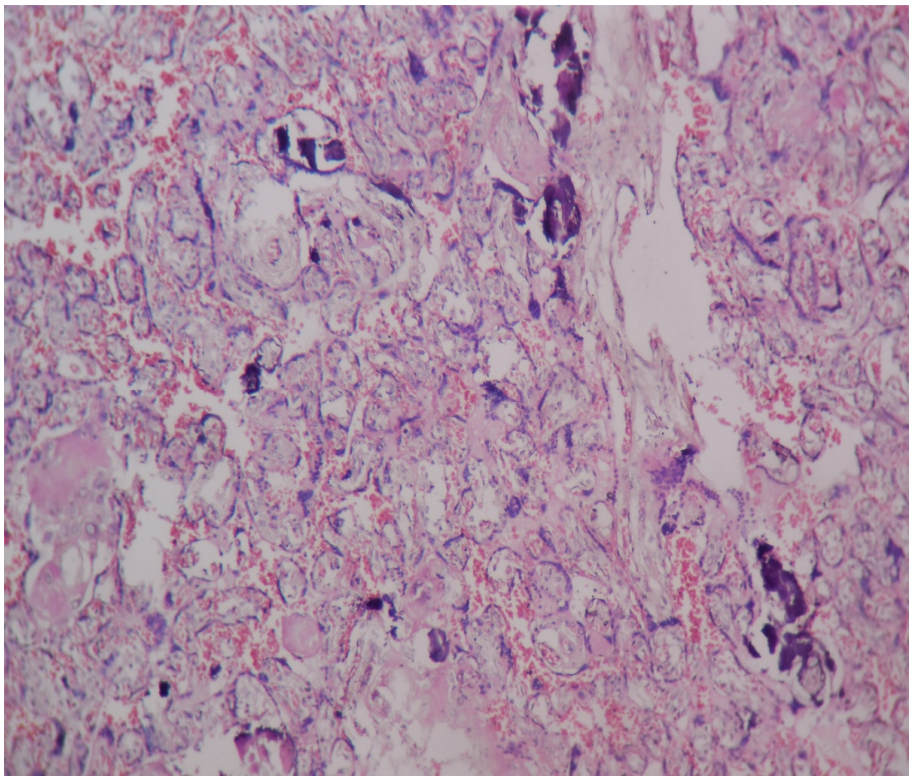
S. No	Calcification of placenta (% of area involved)	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	0	8 (35%)	12(44.4%)	20(40%)	8 (32%)	8 (32%)	16 (32%)
2	1	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (2%)
3	2	7 (30.4%)	6 (22.2%)	13 (26%)	5 (20%)	4 (16%)	9 (18%)
4	3	0 (0%)	0 (0%)	0 (0%)	2 (8%)	3 (12%)	5 (10%)
5	4	0 (0%)	0 (0%)	0 (0%)	1 (4%)	0 (0%)	1 (2%)
6	5	8 (34.8%)	7 (25.9%)	15 (30%)	7 (28%)	6 (24%)	13 (26%)
7	10	0 (0%)	2 (7.4%)	2 (4%)	1 (4%)	0 (0%)	1 (2%)
8	15	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (2%)
9	18	0 (0%)	0 (0%)	0 (0%)	1 (4%)	0 (0%)	1 (2%)
10	20	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (2%)
11	30	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (2%)

This scoring says about the percentage of area affected by calcification.

Distribution of placental calcification between the normal and high risk groups:

Calcification	High Risk group	Normal group
Absent	32%	40%
Present	68%	60%

CALCIFICATION



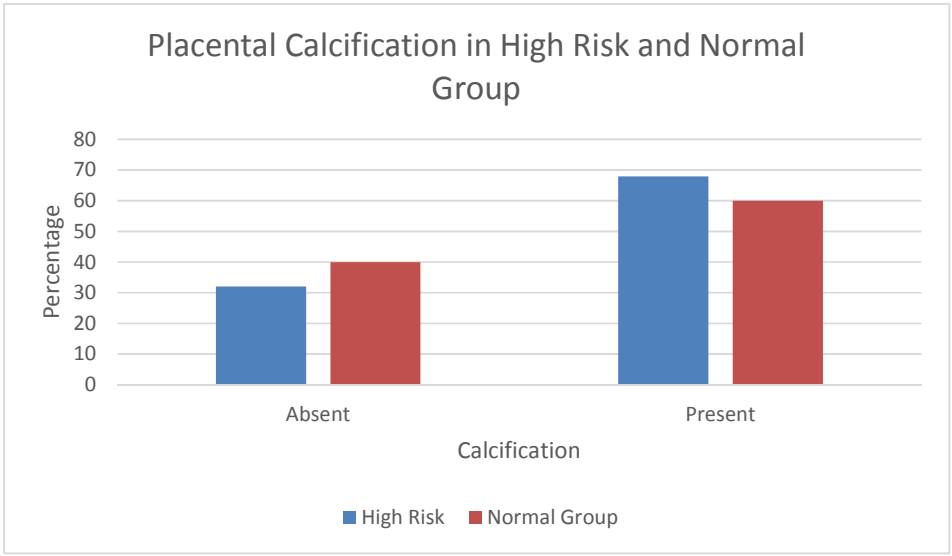


Table 14: Frequency distribution of infarction score in placenta between the normal and high risk group in respect to the parity.

S. No	Infarction in placenta (% of area involved)	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	0	12 (52.2%)	19 (70.4%)	31 (62%)	3 (12%)	4 (16%)	7 (14%)
2	2	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (4%)	2 (4%)
3	3	2 (8.7%)	0 (0%)	2 (4%)	0 (0%)	5 (20%)	5 (10%)
4	4	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (2%)
5	5	5 (21.7%)	6 (22.2%)	11 (22%)	15 (60%)	8 (32%)	23 (46%)
6	8	0 (0%)	0 (0%)	0 (0%)	1 (4%)	0 (0%)	1 (2%)
7	10	3 (13%)	1 (3.7%)	4 (8%)	1 (4%)	1 (4%)	2 (4%)
8	13	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (2%)
9	15	1 (4.3%)	1 (3.7%)	2 (4%)	3 (12%)	2 (8%)	5 (10%)
10	20	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (4%)	2 (4%)
11	25	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (4%)	1 (2%)

This scoring says about the percentage of area affected by infarction.

INFARCTION IN HIGH RISK GROUP:

Infarction was absent in 1 case of PIH, 1 case of GDM, 3 cases of postdated, 2 cases of APH.

INFARCTION of <5% was present in 1 case of PIH, 5 cases of GDM, 1 case of preterm, 1 case of APH.

INFARCTION of >5% was present in 8 cases of PIH, 4 cases of GDM, 9 cases of preterm, 7 cases of postdated, and 7 cases of APH.

INFARCTION IN THE NORMAL GROUP:

Infarction was absent in 31 cases, <5% in 2 cases and >5% in 17 cases.

Hence the percentage of area affected by infarction is more in the high risk group when compared to the normal group.

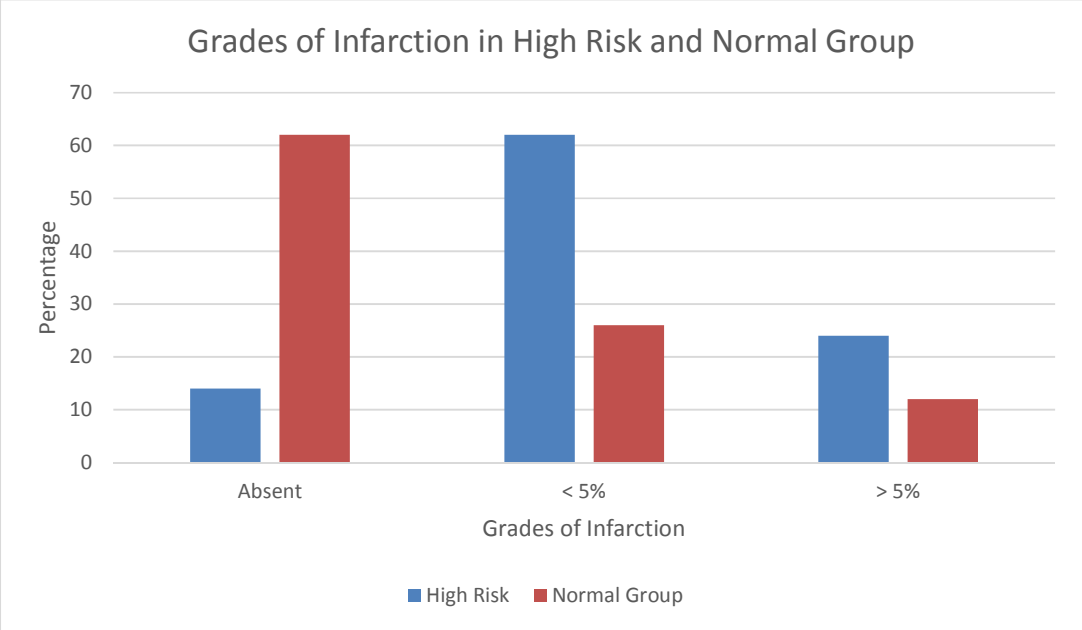


Table 15: Frequency distribution of Leukocyte infiltration in placenta between the normal and high risk group in respect to the parity.

S. No	Leukocyte infiltration	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	Yes	2 (8.7%)	8 (29.6%)	10 (20%)	12 (48%)	11 (44%)	23 (46%)
2	No	21 (91.3%)	19 (70.4%)	40 (80%)	13 (52%)	14 (56%)	27 (54%)

LEUKOCYTE INFILTRATION was present in more no of cases in the high risk group(23 cases) when compared to the normal group it was present only in 10 cases.

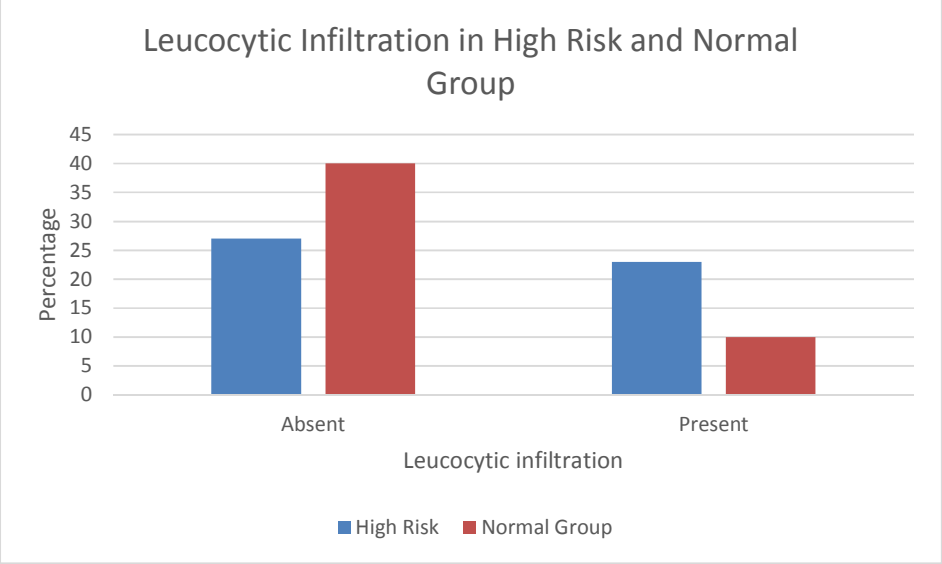


Table 16: Frequency distribution of USG placental grading between the normal and high risk group in respect to the parity.

S. No	USG placental grade	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	Grade I	2 (8.7%)	3 (11.1%)	5 (10%)	3 (12%)	5 (20%)	8 (16%)
2	Grade II	14 (60.9%)	20 (74.1%)	34 (68%)	15 (60%)	15 (56%)	30 (58%)
3	Grade III	7 (30.4%)	4 (14.8%)	11 (22%)	6 (28%)	6 (24%)	12 (26%)

PLACENTAL GRADING IN HIGH RISK GROUP:

Grade I placenta was seen in 1 case of PIH, none in GDM, 3 cases of preterm, 3 cases of postdated, and 1 case of APH.[total- 8 cases]

Grade II placenta was seen in 6 cases of PIH, 7 cases of GDM, 7 cases of preterm, 4 cases of postdated, 6 cases of APH. [total- 20 cases]

Grade III placenta was seen in 2 cases of PIH, 4 cases of GDM, NONE in preterm, 3 cases in postdated, 3 cases in APH. [total- 12 cases]

PLACENTAL GRADING IN NORMAL GROUP:

Grade I placenta was seen in 5 cases, grade II was seen in 34 cases, grade III was seen in 4 cases.

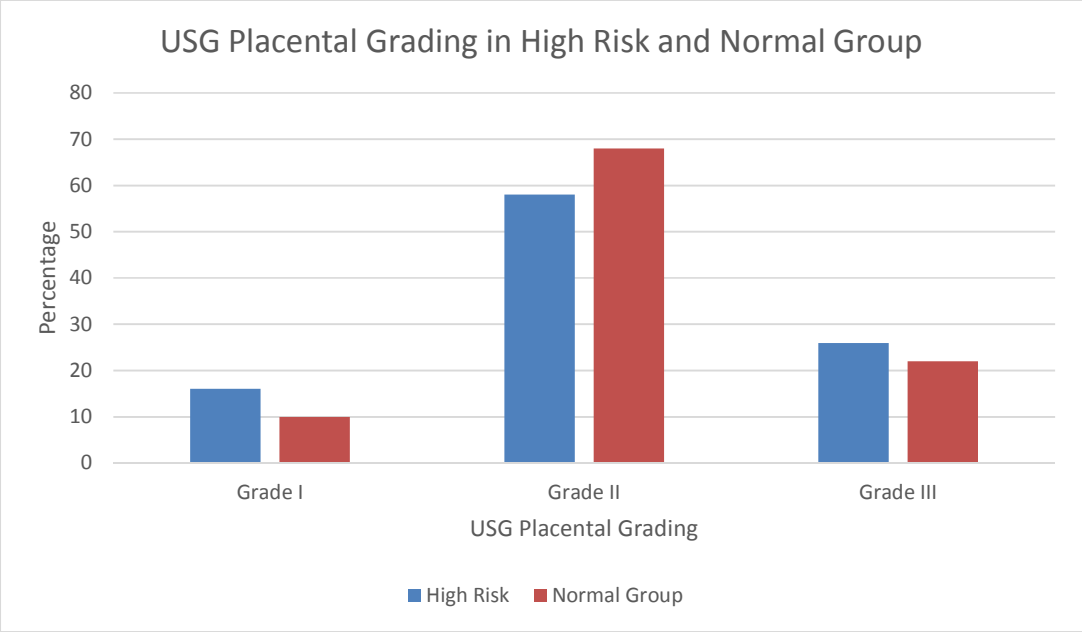

USG Placental Grading in High Risk and Normal Group
80
70
60
50
40
30
20
10
0
Percentage
Grade I
Grade II
Grade III
USG Placental Grading
High Risk
Normal Group

Table 17: Frequency distribution of mode of delivery between the normal and high risk group in respect to the parity

S. No	Mode of Delivery	Normal group (N=50)			High risk group (N=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	Total n (%)	Primi Gravida (n=25)	Multi Gravida (n=25)	Total n(%)
1	Labour Natural	18 (78.2%)	21 (77.7%)	39 (78%)	12 (48%)	14 (56%)	26 (52%)
2	LSCS	3 (13.04%)	4 (14.8%)	7 (14%)	10 (40%)	11 (44%)	21 (42%)
3	Vacuum assisted	1 (4.3%)	0 (0%)	1 (2%)	0 (0%)	0 (0%)	0 (0%)
4	Forceps assisted	1 (4.3%)	2 (7.4%)	3 (6%)	3 (12%)	0 (0%)	3 (6%)

MODE OF DELIVERY IN HIGH RISK GROUP:

No of labour natural was 26, LSCS was 21 cases, forceps delivery was 3 cases,

MODE OF DELIVERY IN NORMAL GROUP:

No of labour natural was 39 cases, lscs was 7 cases, 1 case of vaccum delivery, and 3 cases of outlet forceps delivery.

HENCE LABOUR NATURAL WAS MORE IN THE NORMAL GROUP, WHEREAS LSCS WAS MORE IN THE HIGH RISK GROUP COMPARED TO NORMAL GROUP.

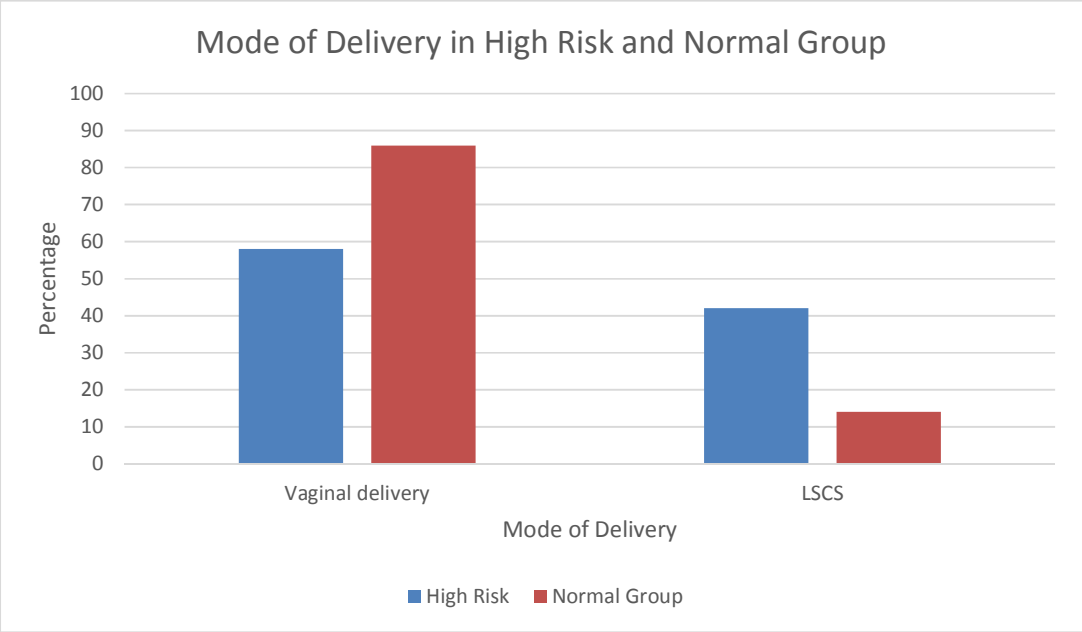


Table 18: Comparison of placental thickness in USG between the normal and high risk groups.

S. No	Parameter	Normal group (n=50)	High risk group (n=50)	P value	Statistical test
1	Placental thickness in USG (cm)	2.9 ± 0.44	3.09 ± 0.33	0.01*	Mann Whitney U test

Data are expressed in mean ± SD. * indicates p <0.05 and considered statistically significant

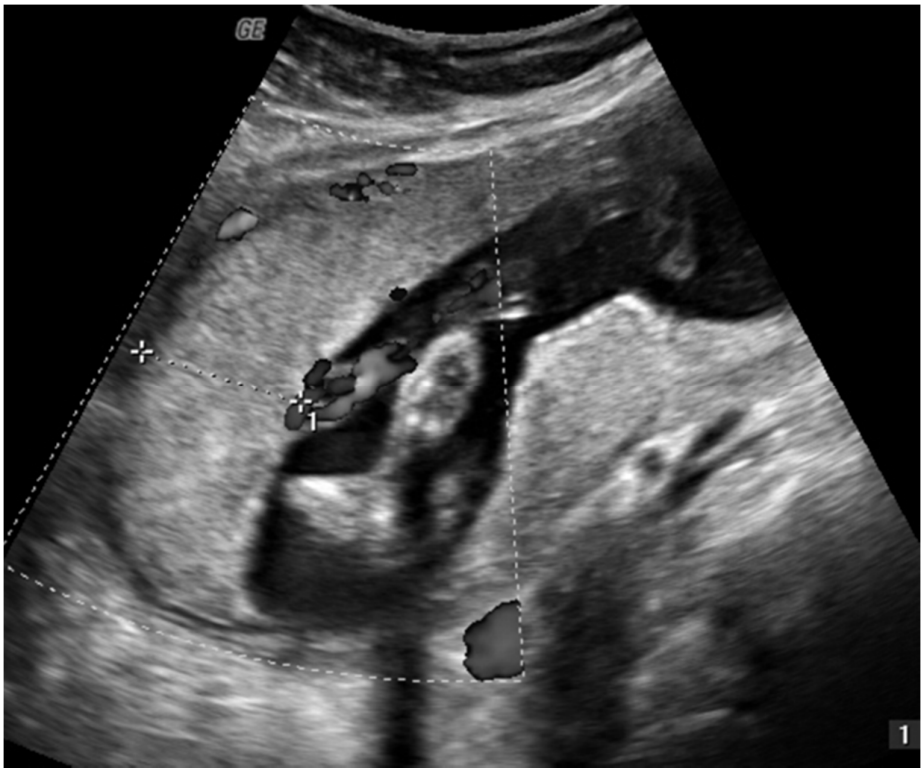
The mean placental thickness is more in the high risk group when compared to the normal group.

Table 19: Comparison of placental thickness in USG between the normal and high risk groups based on the parity.

S. No	Parameter	Normal group (n=50)			High risk group (n=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	P value	Primi Gravida (n=25)	Multi Gravida (n=25)	P value
1	Placental thickness in USG (cm)	2.9 ± 0.5	2.9 ± 0.38	0.729 (NS)	3.05 ±0.27	3.13 ± 0.38	0.526 (NS)

Data are expressed in mean ± SD. P<0.05 is considered statistically significant and unpaired 't' test was used to find the statistical difference

The below picture show 2 dimensional measurement of placental thickness. In the below picture the location of the placenta is lateral grannum grading stage 1.



PLACENTAL THICKNESS MEASUREMENT.

Table 20: Comparison of neonatal outcomes between the normal and high risk groups based on parity.

S. No	Parameter	Normal group (n=50)			High risk group (n=50)		
		Primi Gravida (n=23)	Multi Gravida (n=27)	P value	Primi Gravida (n=25)	Multi Gravida (n=25)	P value
1	Weight of the baby (in Kg)	2.8 ± 0.32	2.7 ± 0.38	0.159 (NS)	2.4 ± 0.6	2.5 ± 0.61	0.692 (NS)
2	Weight of the baby overall (Kg)	2.77 ± 0.36			2.5 ± 0.6		0.008*
3	Apgar Score						
	< 7	0 (0%)	0(0%)	<0.0001*	5 (20%)	3 (12%)	0.782 (NS)
	≥7	23 (100%)	27 (100%)		19 (76%)	19 (76%)	
	Still birth	0 (0%)	0 (0%)		1 (4%)	3 (12%)	

Data are expressed in mean ± SD except apgar score wherein data are expressed as absolute numbers with percentage. * indicates p <0.05 and considered statistically significant.

Low APGAR score is observed more in the babies of high risk group when compared to normal group.

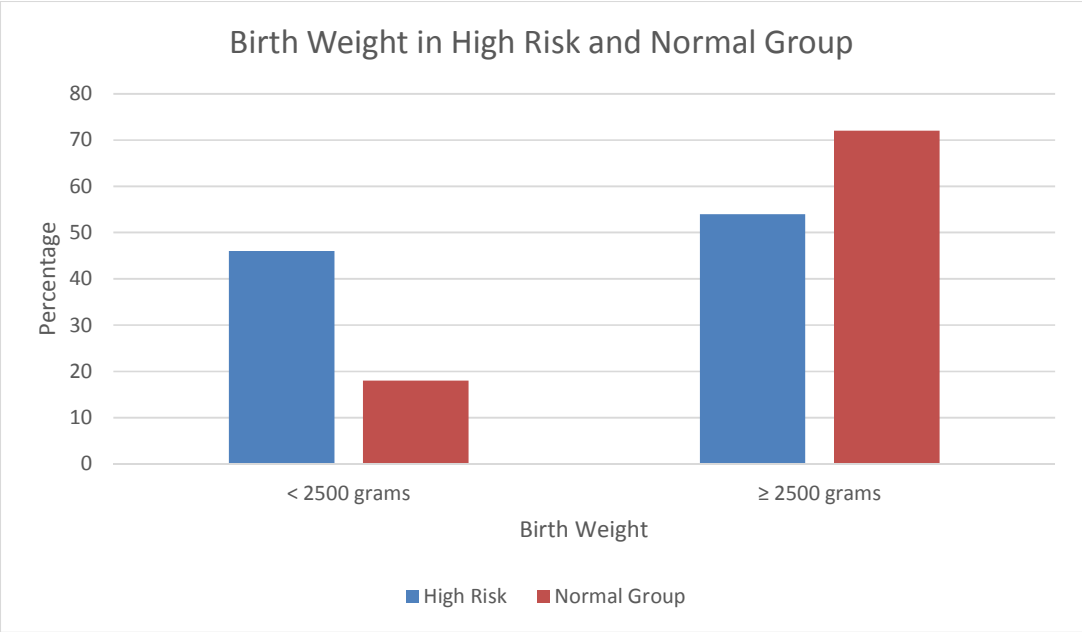
STILL BIRTH was seen in 4 cases of high risk group, whereas no still births in the normal group.

Table 21. Association of high risk factors with birth weight in the high risk population group

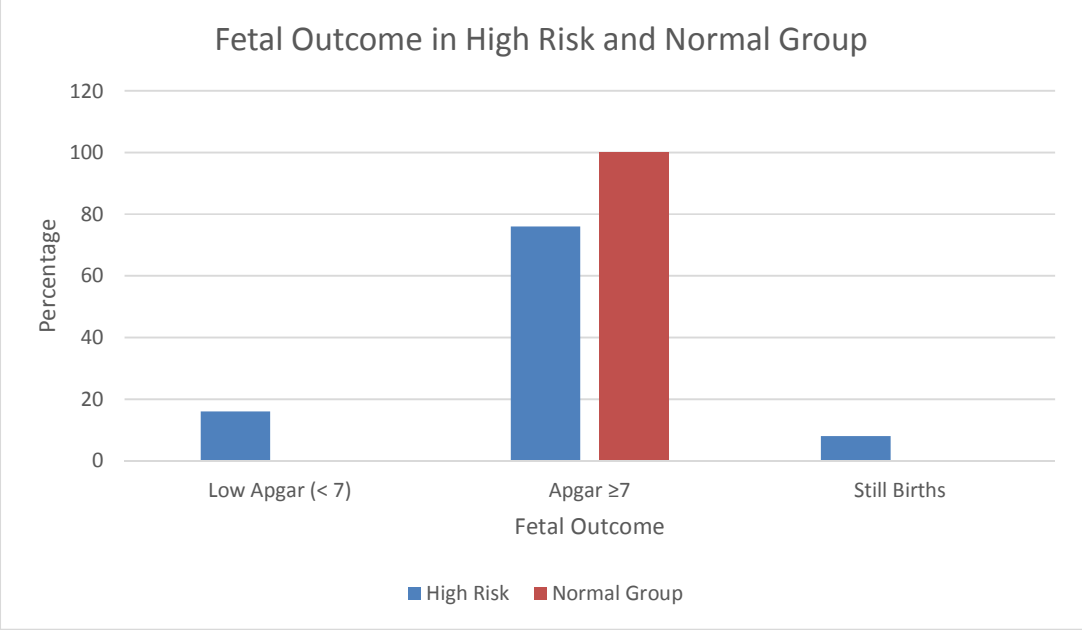
S.No	Type of birth weight	Normal Group N=50 (%)	High risk group N=50 (%)	P value
1	<2500 grams	9 (18%)	23 (46%)	0.0004*
	≥ 2500 grams	41 (72%)	27 (54%)	

Chi square test was used to test the statistical significance. * indicates $p<0.05$ and considered statistically significant. The odd's ratio is 3.88 (95% Confidence interval is 1.96 to 9.65) between the groups. Hence the odd's of getting low birth weight baby is 3.8 times higher in high risk population than the normal group.

LOW BIRTH WEIGHT was seen in more no of high risk cases when compared to that of normal group.



FETAL DISTRESS was observed more in the high risk group when compared to the normal group.



DISCUSSION

PLACENTAL ULTRASONOGRAPHY

The grading and placental thickness was compared with study done by Tewari k et al(1995 and 1997)

Placental grading and mean placental thickness- comparison of present study with other published studies

Tewari k et al. 1995

Grading	MPT(mm)
I	49.7+5.4
II	40.0+3.4
III	34.2+3.6

Tewari k et al.1997

GA	MPT(mm)
28-32	51.86+5.7
32-36	43.70+4.5
>36	37.40+5.5

Present study 2017

Grade	MPT(mm)	
	High risk	Normal
0	36	-
I	29	24.4
II	30.7	29
III	32.2	28

The mean placental thickness in grade III correlates with the study done by Tewari k et al.(1995). There was no correlation found between placental thickness and adverse fetal outcome.

Placental grading was correlated with the fetal outcome. In the high risk group, In placenta with grade I there were 8 cases out of which 1 case had fetal distress and 1 case had still birth.

In 29 cases with grade II, 5 cases had low APGAR score, 5 cases had fetal distress and there were 2 cases of still birth.

In 13 cases with grade III, only 2 cases had fetal distress and 2 cases low APGAR. There was no still birth. Cases with grade III placentas comparatively had low adverse fetal outcomes.

In the normal group, 5 cases of grade I placenta, 34 cases of grade II placenta and 11 cases of grade III had normal fetal outcome.

HISTOPATHOLOGICAL STUDY OF PLACENTA

MACROSCOPIC STUDY OF PLACENTA:

Placental weight <300 gms- comparison of present study with other published studies.

AUTHOR	HIGH RISK	NORMAL
Neerja et al. 1999	22%	0%
Banadanadas et al.1996	45.7%	0%
Anjali R et al 1985	23%	0%
Rathna R et al 2002	4%	0%
Present study	12%	6%

Only 3 cases in the normal group had placental weight of <300 gms. In high risk group, 6 cases had weight <300 gms.

1 case in the high risk group with placental weight <300 gms had fetal distress.

PLACENTAL SIZE OF >20cm- comparison of present study with other published studies

AUTHORS	HIGH RISK	NORMAL
Bandanadas et al 1996	5%	0%
Rathna R et al 2002	16%	4%
Present study	14%	6%

There was significant correlation between placental size and fetal outcome.

Here present study correlates with the study done by rathna R et al.

SYNCITIAL KNOTS OF>30%- COMPARISON OF PRESENT STUDY WITH OTHER STUDIES.

Authors	High risk	Normal
Avasthik et al 1991	40.8%	0%
Twearik et al 1997	74.5%	16.5%
neerja et al 1999	33%	3%
Rathna R et al 2002	40%	8%
Present study	88%	70%

There is a significant increase in the syncytial knotting in the high risk group of pregnancies.

FIBRINOID NECROSIS OF >5%- COMPARISON OF PRESENT STUDY WITH OTHER PUBLISHED STUDIES

Authors	High risk	Normal
Avasthik et al 1991	20	0
Twearik et al 1997	47	0
neerja et al 1999	32	0
Rathna R et al 2002	50	20
Present study	54	20

Present study correlates with study done by rathna R et al. fibrinoid necrosis of higher grades II and III was found to be in PIH cases followed by GDM, and POSTTERM.

FIBROSIS OF >3%- COMPARISON OF PRESENT STUDY WITH OTHER PUBLISHED STUDIES

Authors	High risk	Normal
Avasthik et al 1991	30%	0
Twearik et al 1997	59.5%	0
Neerja et al 1999	32%	0
Rathna R et al 2002	38%	28%
Present study	26%	14%

Fibrosis of grade II is more in the high risk group when compared to the normal group. This present study is comparable with the study done by rathna r et al.

LEUKOCYTIC INFILTRATION- COMPARISON OF PRESENT STUDY WITH OTHER PUBLISHED STUDIES

Authors	High risk		normal	
	present	absent	present	Absent
avasthi k et al 1991	20.6%	79.9%	0%	100%
Neerja et al 1999	30%	70%	0%	100%
Rathna R et al 2002	48%	52%	20%	80%
present study	46%	54%	20%	80%

Present is found to be comparable with study done by rathna r et al.

INFARCTION OF >5%- COMPARISON OF PRESENT STUDY WITH OTHER PUBLISHED STUDIES

Authors	High risk		normal	
	present	absent	present	Absent
avasthi k et al 1991	25%	75%	8%	92%
Rathna R et al 2002	66%	17%	12%	78%
present study	86%	14%	38%	62%

Present study is comparable with that of study done by rathna r et al.

CALCIFICATION- COMPARISON OF PRESENT STUDY WITH OTHER PUBLISHED STUDIES

Authors	High risk		normal	
	present	absent	present	Absent
avasthi k et al 1991	25%	75%	8%	92%
Rathna R et al 2002	64%	36%	56%	44%
present study	68%	32%	60%	40%

Present study is comparable with that of study done by rathna r et al.

MODE OF DELIVERY- COMPARISON OF PRESENT STUDY WITH OTHER PUBLISHED STUDIES

Authors	VAGINAL		LSCS	
	High risk	normal	High risk	Normal
neerja et al 1999	44%	70%	56%	30%
Rathna R et al 2002	52%	64%	48%	16%
present study	58%	86%	42%	14%

Present study is comparable with that of study done by rathna r et al

FETAL OUTCOME- COMPARISON OF PRESENT STUDY WITH OTHER PUBLISHED STUDIES

Fetal outcome	Rathna r et al 2002		Present study	
	High risk	Normal	High risk	Normal
Fetal distress	32%	4%	18%	8%
LOW APGAR	24%	0%	14%	0%
Still birth	10%	0%	8%	0%

CORRELATION OF HISTOLOGIC FINDINGS WITH FETAL OUTCOME

Histological features	Fetal distress 18%	Low APGAR 14%	STILL BIRTH 8%
Syncitial knots			
I(0-29%)	16%	16%	0%
II(20-59%)	12.5%	8.3%%	4%
III(60-89%)	15%	5%	6%
IV(>90%)	Nil	Nil	Nil
Fibrinoid necrosis			
I(0-5%)	17%	4%	4%
II(5-10%)	10%	20%	10%
III(>10%)	42%	0%	14%
Fibrosis			
Absent			
I(0.3-3%)	16%	16%	Nil
II(>3%)	7%	15%	7%
Infarction			
(0-5%)	18%	5%	NIL
(6-10%)	NIL	100%	NIL
(>10%)	11%	22%	44%
Calcification			
Absent	12%	NIL	6%
Present	17%	20%	8%

HENCE ADVERSE FETAL OUTCOMES ARE ASSOCIATED WITH INCREASING GRADES OF SYNCITIAL KNOTS, FIBROSIS, FIBRINOID NECROSIS AND INFARCTION. ALSO WHEN THE LEUKOCYTE INFITRATIONAND CALCIFICATION ARE PRESENT THERE IS INCREASED PERCENTAGE OF FETEL DISTRESS, LOW APGAR AND STILL BIRTH.

SUMMARY

Infants with growth restriction have smaller placentas relative to infants with normal birth weight of similar gestational age. As growth restriction continues, the ratio of birth weight to placental weight is reduced. Progressively more placental mass is required to maintain the fetus.

Examination of the placenta gives information which will lead to a clear understanding of the origin of the abnormal outcome and is a valuable tool in predicting the outcome of future pregnancies and their management.

Sonographic placental changes in terms of thickness with advanced maturational grades correspond to histological features suggestive of villous aging, uteroplacental ischemia and hampered fetal villous perfusion, thus adversely affecting the growth of the fetus. Ultrasonographic placental grading gives an idea about the fetal maturity.

There is an exaggerated response of placenta to various disorders complicating pregnancies and the degree of pathological abnormalities may have an impact on the fetal growth.

The present study shows a significant correlation between the histological abnormalities of placenta and fetal outcome. But the pathologic changes in the placenta are not specific to a particular disorder and a variety of disorders may have similar changes

CONCLUSION

Healthy baby and healthy mother is the goal of every obstetrician. The study is a humble endeavor to assess the propable link of morphological and histopathological changes in placenta to fetal outcome and thereby pave way for better fetal outcome. Many, such studies are done all over the world and days may not be far off when a definite link is proved.

Examination of placenta with warning signals for neonatal outcome revealed within few days, in a tertiary care hospital may alert the obstetrician as well as the neonatalogist to keep such babies under careful monitoring in NICU.

The ultrasonograhic and histopathological study of placenta may suggest the possible pathogenesis due to decreased or compromised uteroplacental function. Early detection, timely intervention and good antenatal care of high risk patients may prevent the adverse fetal outcome and thus reduce the incidence of fetal growth restriction.

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PROFORMA

Name:

Age:

IP No.:

Place: Occupation:

LMP: EDD:

Diagnosis:

Menstrual history:

GENERAL EXAMINATION:

Height: cm.

Weight: kg.

BP: mm of Hg. Pulse rate:

Temp: Pallor:

Pedal edema:

CLINICAL EXAMINATION:

CVS:

RS:

P/A:

Fundal height:

INVESTIGATIONS:

ROUTINE:

Hb :

PLATELET :

BLOOD SUGAR :

UREA :

CREATININE :

LFT :

PPTCT :

Blood group and Type:

URINE: Albumin: Sugar: Deposits:

SPECIFIC (If any) :

USG on

FETAL BIOMETRY:

BPD

FL

AC

HC

HC/AC

BIOPHYSICAL PROFILE:

Breathing movements:

Movements:

Tone:

Reactive Heart rate:

EFW gms.

AFI cm

IMPRESSION:

ULTRASONOGRAPHIC STUDY OF PLACENTA:

Grading:

Thickness:

MACROSCOPIC STUDY OF PLACENTA:

Size: diameter in cms

Shape:

Weight: gms.

Colour of the membranes:

No. of cord vessels:

Cord length:

HISTOPATHOLOGICAL EXAMINATION OF PLACENTA:

Infarction: %

Calcification: %

Syncitial knots: Grade

Fibrosis: Grade

Fibrinoid necrosis: Grade

Leucocytic infiltration: + / -

MODE OF DELIVERY:

INDICATION FOR INTERVENTION:

Baby notes:

Sex: f/m

Weight: kg.

Live birth/ still birth:

APGAR: 1min 5min

Signs of fetal distress:

NICU admission: yes/ no

Reason for admission:

Outcome:

PATIENT CONSENT FORM

Study Detail: A Study on "ANTENATAL ULTRASOUND AND POSTPARTUM HISTOPATHOLOGICAL STUDY OF PLACENTA IN HIGH RISK PREGNANCIES AND ITS CORRELATION WITH FETAL OUTCOME"

Study Centre: Department of Obstetrics & Gynaecology, Thanjavur medical college, Thanjavur.

I confirm that I have read and understood the information Sheet for the above study. I have had the opportunity to ask questions and all my questions and doubt have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that i am free to withdraw at an time, without giving any reason, without my legal rights being affected.

I understand that the Clinical study personnel, the Ethics Committee and the Regulatory Authorities 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 agree to this access. However, I Understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instructions given during the study and to faithfully co-operate with the study team, and to immediately inform the study if I suffer from any deterioration in my health of well being or any unexpected or unusual symptoms.

I hereby give permission to undergo completed clinical examination and diagnostic tests including haematological, biochemical, radiological tests.

I hereby consent to participate in this study.

Signature/Thumb impression:..... Place Date of the patient

Patient's Name, Address & Ph.No:.....

Name of the Investigation:.....

Signature of the Investigator: ..... Place.....Date

Institution : .....

Signature of the Relative/Gardian.....

KEY TO MASTER CHART

GHT: gestational hypertension

GDM: gestational diabetes mellitus

APH: antepartum haemorrhage

D: discoid

B: bilobed

C: club shaped

Lscs: lower segment caesarean section

CPD : cephalopelvic disproportion

MSAF: meconium stained amniotic fluid

S.No	Name	Age	IP No	Parity	High risk factor	placental weight g	placental size in cm	shape of placenta	synctitial knots	fibrosis	fibrinoid necrosis	calcification	infarction	leuckocyte infiltration
1	Jacquelin sandhanmary	28	468201	Multi	GHT	350	24	D	III	II	II	0	0	+
2	Abirami	20	469499	Primi	GHT	450	15	D	II	0	III	5	15	+
3	Suseela	24	469546	Primi	GHT	250	15	D	I	0	II	5	5	+
4	Kokila	19	469859	Primi	GHT	320	18	D	II	0	II	5	8	+
5	Rekha	24	465239	Multi	GHT	340	18	D	I	0	I	0	3	+
6	Nivetha	24	465150	Primi	GHT	600	20	D	II	0	III	0	5	+
7	Arokya ramya	22	462015	Primi	GHT	400	25	D	I	II	I	5	5	-
8	Malathi	28	468146	Multi	GHT	300	14	D	II	0	II	5	5	-
9	Kala	30	479168	Multi	GHT	280	18	D	II	I	II	2	15	+
10	Valarmathi	29	470684	Primi	GHT	600	24	D	III	0	II	5	5	+
11	Libiya	29	479499	Multi	GDM	230	20	C	III	II	III	2	5	-
12	Jeyanthi	32	479522	Multi	GDM	350	22	D	II	II	I	30	3	-
13	Vijaya	24	480612	Primi	GDM	500	24	D	III	0	II	0	5	-
14	Kavitha	30	481718	Multi	GDM	450	16	D	II	I	III	1	3	-
15	Selvi	27	484702	Multi	GDM	400	20	D	II	II	I	5	3	-
16	Anandharuby	29	489245	Primi	GDM	380	15	D	III	0	I	2	0	-
17	Mallika	25	480486	Multi	GDM	350	14	D	II	0	I	15	5	+
18	Vanathi	29	487124	Primi	GDM	500	18	D	III	I	II	2	2	+
19	Kauveri	31	492196	Multi	GDM	420	20	C	III	I	II	2	2	-
20	Manimala	26	487418	Primi	GDM	450	22	D	II	II	III	18	5	-
21	Sumathi	19	474016	Primi	preterm	450	9	D	II	I	I	10	5	+
22	Valli	28	474289	Primi	preterm	390	10	D	I	0	I	5	5	-
23	Uma	26	481268	Multi	preterm	300	10	D	III	II	III	2	20	+
24	Banumathy	21	480855	Multi	preterm	250	8	D	II	0	I	0	5	-
25	Minnalkodi	25	480888	Primi	preterm	350	12	D	III	0	II	0	20	+
26	Shanthi	24	480802	Multi	preterm	300	9	D	II	0	I	0	5	-
27	Sasikala	31	483701	Primi	preterm	400	15	D	II	0	I	0	5	-
28	Kalaivani	22	492614	Primi	preterm	300	10	D	III	0	II	2	5	-
29	Keerthika	20	484321	Primi	preterm	280	12	D	II	0	I	0	5	-
30	Amudha	25	481602	Multi	preterm	250	14	D	II	0	I	0	3	+
31	Manimegalai	32	477664	Multi	post dated	650	18	D	I	0	I	0	0	-
32	Kowsalya	28	476999	Primi	post dated	400	6	D	II	0	II	0	0	-
33	Sudha	24	482142	Primi	post dated	350	15	D	III	II	I	3	5	+
34	Selvi	27	478387	Multi	post dated	450	15	D	III	II	I	0	0	+

S.No	Name	Age	IP No	Parity	High risk factor	placental weight g	placental size in cm	shape of placenta	synctitial knots	fibrosis	fibrinoid necrosis	calcification	infarction	leuckocyte infiltration
35	Vijayalakshmi	18	481245	Multi	post dated	400	16	D	III	II	II	3	15	-
36	Gunasundari	19	480657	Multi	post dated	400	15	D	II	I	II	5	13	-
37	Mahalakshmi	26	478557	Multi	post dated	550	25	D	III	0	III	20	25	+
38	Vennila	29	477286	Primi	post dated	500	20	C	II	0	II	4	15	+
39	Vimala	24	488635	Primi	post dated	450	15	D	III	0	I	3	5	+
40	Suseela	28	484574	Multi	post dated	550	20	D	II	0	I	5	5	-
41	Thilagavathy	30	499053	Multi	APH	550	20	D	I	0	II	5	5	-
42	Vimala	24	480699	Multi	APH	350	15	D	II	0	I	0	5	-
43	Rajakumari	22	480601	Multi	APH	450	20	D	III	0	II	3	10	+
44	Kala	28	488630	Primi	APH	500	18	D	II	II	I	0	0	-
45	Sevanthiamal	26	483046	Primi	APH	450	18	D	III	0	I	0	5	-
46	Renuka	26	488055	Primi	APH	500	14	D	III	0	II	2	15	+
47	Annalakshmi	29	483046	Multi	APH	400	12	D	III	II	II	3	4	+
48	Chitra	28	481284	Primi	APH	450	18	D	III	0	II	2	5	-
49	Usharani	24	479267	Primi	APH	400	20	D	II	II	I	5	10	-
50	Priya	20	481450	Multi	APH	450	14	D	II	0	I	5	0	+
51	Gomathi	24	499692	Primi	nil	450	24	D	III	II	II	0	15	-
52	Gnanamani	22	475004	Primi	nil	400	8	D	II	0	II	0	0	-
53	Sumathi	30	480527	Primi	nil	500	13	D	II	0	I	nil	3	+
54	Tamilselvi	27	485624	Primi	nil	500	13	D	I	0	I	2	0	-
55	Visalakshi	29	481234	Primi	nil	450	12	D	I	0	I	5	0	-
56	Rajeshwari	26	476125	Multi	nil	500	14	D	II	I	I	2	0	-
57	Senthamilselvi	24	481298	Multi	nil	500	15	D	I	0	I	5	0	+
58	Palaniyammal	19	470654	Multi	nil	400	15	D	II	0	I	nil	0	-
59	Muthamilselvi	24	476134	Primi	nil	450	15	B	II	0	I	2	0	-
60	Pushpalatha	29	480679	Primi	nil	450	18	D	I	0	I	nil	0	-
61	Nirmala	27	480888	Multi	nil	500	20	D	I	0	I	nil	0	-
62	Radha	24	471965	Multi	nil	500	19	D	II	0	I	nil	0	-
63	Nagammal	25	478564	Primi	nil	500	9	D	II	II	II	5	0	-
64	Sumathi	28	474421	Multi	nil	450	18	D	II	II	I	nil	5	-
65	Selvi	23	473428	Multi	nil	550	14	D	I	0	0	5	10	+
66	Sundari	24	484123	Primi	nil	550	18	D	II	0	I	5	0	-
67	Rajalakshmi	23	476942	Multi	nil	600	15	D	II	0	I	2	5	+
68	Kala	24	476134	Multi	nil	500	14	D	III	I	II	5	0	-
69	Uma	28	489256	Primi	nil	400	18	D	II	0	I	5	10	-

S.No	Name	Age	IP No	Parity	High risk factor	placental weight g	placental size in cm	shape of placenta	synctitial knots	fibrosis	fibrinoid necrosis	calcification	infarction	leuckocyte infiltration
70	Revathy	34	490154	Primi	nil	450	18	D	II	0	I	nil	0	-
71	Anjammal	29	482453	Primi	nil	550	16	D	I	I	I	2	5	-
72	Thamaraiselvi	27	489269	Primi	nil	450	12	D	II	0	I	5	0	-
73	Parvathi	26	478405	Primi	nil	400	20	D	II	0	0	2	0	-
74	Usha	24	481297	Multi	nil	550	14	D	I	0	I	0	0	+
75	Vijayalakshmi	25	473468	Multi	nil	450	16	D	II	0	I	5	0	-
76	Chitra	23	474281	Multi	nil	450	14	D	III	II	II	0	15	-
77	Nandhini	21	477419	Multi	nil	400	12	D	II	0	0	nil	0	-
78	Pavithra	22	482352	Primi	nil	500	18	D	II	0	I	2	3	-
79	Suganya	20	477545	Multi	nil	500	20	D	II	0	I	5	0	-
80	Kokilam	18	477781	Multi	nil	450	16	D	I	0	I	2	5	-
81	Malathy	24	470166	Primi	nil	500	14	D	II	I	I	5	0	-
82	Parimala	22	482432	Multi	nil	500	18	D	II	0	II	nil	0	+
83	Angelin	21	484331	Multi	nil	450	20	D	I	0	I	2	0	-
84	Kasthuri	23	478631	Multi	nil	380	22	D	II	0	I	nil	0	-
85	Udhaya	20	477664	Primi	nil	250	16	D	III	0	I	nil	5	-
86	Abirami	28	476669	Primi	nil	260	16	D	II	0	I	2	0	-
87	Abinaya	27	482142	Multi	nil	320	18	D	II	0	II	5	0	+
88	Vidhya	29	478387	Primi	nil	400	20	D	I	II	0	5	10	-
89	Sathya	28	482115	Multi	nil	450	24	D	I	II	I	10	0	-
90	Suseela	28	471657	Multi	nil	520	12	D	I	I	II	nil	0	-
91	Jayakodi	26	478587	Primi	nil	500	14	D	II	0	I	2	5	-
92	Valli	27	477286	Multi	nil	380	16	D	II	0	I	2	0	-
93	Sandhya	31	488635	Multi	nil	280	20	D	II	0	I	0	5	+
94	Saranya	30	484574	Primi	nil	290	20	C	III	I	I	5	5	+
95	Nithya	25	483085	Multi	nil	300	18	D	II	0	II	0	5	-
96	Kanagam	28	499053	Primi	nil	450	12	D	I	0	I	0	10	-
97	Kavitha	24	480699	Multi	nil	400	14	D	II	0	I	2	0	-
98	Kalpana	29	480601	Multi	nil	220	14	D	II	II	0	5	0	-
99	Parvathi	20	488921	Multi	nil	280	16	D	II	0	I	10	5	+
100	Menaka	22	488630	Primi	nil	300	18	D	I	I	II	nil	5	-

S.No	Name	USG placental grade	Placental thickness mm	Mode of delivery	Indication for operative procedures	Baby weight kg	apgar score
1	Jacquelin sandhanmary	II	3.4	LSCS	Previous LSCS/CPD	3.1	8
2	Abirami	II	3	Vaginal		2.6	9
3	Suseela	II	3.5	Vaginal		2.5	3
4	Kokila	II	3	Forceps	Fetal distress	2.5	5
5	Rekha	II	3.5	LSCS	Fetal distress	2.2	8
6	Nivetha	II	3.8	LSCS	Failed induction	3.5	8
7	Arokya ramya	III	3	Vaginal		2.8	9
8	Malathi	III	2.6	Vaginal		2.75	8
9	Kala	I	3	Vaginal		1.6	0
10	Valarmathi	III	3.2	LSCS	Failed induction	3.25	8
11	Libiya	III	4.2	LSCS	Fetal distress	3.98	8
12	Jeyanthi	II	3.2	Vaginal		3	9
13	Vijaya	II	3.1	Vaginal		2.5	8
14	Kavitha	II	2.8	Vaginal		2.4	7
15	Selvi	II	3.4	LSCS	Previous LSCS/CPD in labour	2.25	8
16	Anandharuby	III	3	Forceps	Failed maternal effects	3	8
17	Mallika	II	3	LSCS	Fetal distress	3.1	9
18	Vanathi	II	2.8	Vaginal		2.7	8
19	Kauveri	III	3.8	Vaginal		2.4	8
20	Manimala	II	3	LSCS	MSAF/Fetal distress	3.5	9
21	Sumathi	II	3.1	Vaginal		1.9	8
22	Valli	II	2.8	Vaginal		1.8	9
23	Uma	II	3	Vaginal		1.8	0
24	Banumathy	II	3	Vaginal		1.5	8
25	Minnalkodi	II	3	Vaginal		2	0
26	Shanthi	I	3.6	Vaginal		1.75	8
27	Sasikala	I	3.4	Vaginal		2.4	9
28	Kalaivani	II	3.2	LSCS	Fetal distress	1.9	8
29	Keerthika	II	3	Vaginal		1.7	8
30	Amudha	I	3	Vaginal		1.8	8
31	Manimegalai	II	2.8	LSCS	Failed induction	3	9
32	Kowsalya	I	2.4	Vaginal		2.1	9
33	Sudha	III	3	Vaginal		1.2	8
34	Selvi	I	3.4	Vaginal		2.75	9

S.No	Name	USG placental grade	Placental thickness mm	Mode of delivery	Indication for operative procedures	Baby weight kg	apgar score
35	Vijayalakshmi	II	3.2	Vaginal		2.75	8
36	Gunasundari	III	3	LSCS	CPD	2.1	4
37	Mahalakshmi	II	2.8	LSCS	Fetal distress	3.25	0
38	Vennila	II	3.4	Forceps	Failed maternal effects	2.6	9
39	Vimala	I	3	LSCS	Fetal distress	3.5	8
40	Suseela	III	3	LSCS	Failed induction	3.4	8
41	Thilagavathy	II	2.4	LSCS	Placenta previa with bleeding	2.75	8
42	Vimala	I	3.2	LSCS	Previous LSCS/Placenta previa	2	8
43	Rajakumari	II	3	Vaginal		2.4	2
44	Kala	III	2.8	LSCS	Abruption/Fetal distress	3	8
45	Sevanthiamal	III	3	LSCS	Abruption/Unfavourable cervix	2.6	2
46	Renuka	III	3	LSCS	Placenta previa with bleeding	2.3	6
47	Annalakshmi	II	2.8	Vaginal		2.75	5
48	Chitra	II	3	LSCS	Previous LSCS/Placenta previa	2.2	8
49	Usharani	II	2.8	LSCS	Placenta increta	1.8	5
50	Priya	III	3.2	Vaginal		2.8	9
51	Gomathi	III	4.5	Vaginal	Prolonged 2nd stage	2.6	9
52	Gnanamani	III	2.6	Vaginal		2.9	9
53	Sumathi	II	2.8	Vaginal		3	8
54	Tamilselvi	III	2.4	Vaginal		3	8
55	Visalakshi	III	2.4	Vaginal		2.4	9
56	Rajeshwari	I	2.5	Vaginal		2.8	7
57	Senthamilselvi	II	3	Vaginal		2.5	8
58	Palaniyammal	III	2.5	LSCS	Severe Oligohydramnios	2.5	8
59	Muthamilselvi	II	2.5	Vaginal		2.7	9
60	Pushpalatha	II	3	Vaginal		3	7
61	Nirmala	II	3.2	Vaginal		2.8	8
62	Radha	II	3.2	Forceps	Fetal distress	3	8
63	Nagammal	III	3.5	Vaginal		2.2	9
64	Sumathi	II	4	LSCS	CPD	2.6	9
65	Selvi	II	3	Vaginal		2.9	8
66	Sundari	II	3.4	Vaginal		3	8
67	Rajalakshmi	III	3.6	Vaginal		3.25	9
68	Kala	II	2.8	Vaginal		3.5	8
69	Uma	II	3.6	Vaginal		2.8	8

S.No	Name	USG placental grade	Placental thickness mm	Mode of delivery	Indication for operative procedures	Baby weight kg	apgar score
70	Revathy	II	3	Vaginal		2.6	9
71	Anjammal	II	2.8	Vaginal		2.5	9
72	Thamaraiselvi	II	2.4	LSCS	CPD	3.4	8
73	Parvathi	II	3	Vaginal		3.2	9
74	Usha	II	3.2	Vaginal		2.8	7
75	Vijayalakshmi	II	2.6	Forceps	Fetal distress	2.1	8
76	Chitra	II	2.8	Vaginal		2.9	8
77	Nandhini	II	2.4	Vaginal		3.4	8
78	Pavithra	II	2.4	Vaccum	Prolonged 2nd stage	3.2	9
79	Suganya	II	2.5	LSCS	Non reactive CTG	2.7	7
80	Kokilam	III	3	Vaginal		3.1	8
81	Malathy	I	2.5	Vaginal		2.8	8
82	Parimala	II	2.8	Vaginal		2.5	9
83	Angelin	II	3.2	Vaginal		2.5	9
84	Kasthuri	II	3.3	Vaginal		2.6	9
85	Udhaya	III	3.4	Forceps	Fetal distress	3	8
86	Abirami	II	2.8	LSCS	CPD	2.9	8
87	Abinaya	II	3	Vaginal		2.25	8
88	Vidhya	II	3.2	Vaginal		2.8	9
89	Sathya	I	3.3	LSCS	Previous LSCS/CPD in labour	2.5	8
90	Suseela	II	2.5	Vaginal		2.4	9
91	Jayakodi	II	2.6	Vaginal		3.25	9
92	Valli	III	2.8	Vaginal		3.1	9
93	Sandhya	II	3	Vaginal		2.2	9
94	Saranya	III	3	Vaginal		2.8	9
95	Nithya	I	3.2	Vaginal		3.2	8
96	Kanagam	II	3	LSCS	Fetal distress	3.4	9
97	Kavitha	II	2.5	Vaginal		2.6	7
98	Kalpana	II	2.4	Vaginal		2.1	8
99	Parvathi	II	2.8	Vaginal		2.4	8
100	Menaka	I	2.6	Vaginal		2.25	9