DISSERTATION ON

"COMPARATIVE STUDY OF PLACENTAL VOLUME IN NORMAL PREGNANCY AND INTRA UTERINE GROWTH RESTRICTION"

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CERTIFICATE

This is to certify that this dissertation **"COMPARATIVE STUDY OF PLACENTAL VOLUME IN NORMAL PREGNANCY AND INTRA UTERINE GROWTH RESTRICTION"** submitted by **Dr.PAKKIALAKSHMI.G,** appearing for M.D. Degree Branch II Obstetrics & Gynaecology examination in April 2013 is a bonafide record of work done by her under my direct guidance and supervision in partial fulfillment of the regulations of the Tamilnadu Dr.M.G.R Medical University, Chennai. I forward this to the Tamilnadu Dr.M.G.R Medical University, Chennai, Tamilnadu, India.

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DECLARATION

I solemnly declare that this dissertation entitled comparative study of placental volume in normal pregnancy and intra uterine growth restriction was done by me at institute of Obstetrics and Gynaecology, Madras Medical College, during 2010-2013 under the guidance and supervision of professor Dr. Umashanthi MD., DGO., This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical Unviersity towards the partial fulfillment of requirements for the award of M.D. Degree in Obstetrics and Gynaecology (Branch-II).

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CONTENTS

INTRODUCTION

Fetal growth restriction continues to be one of the major complications of pregnancy affecting 5-10% of all gestation .It is associated with increased morbidity and mortality in perinatal period and in infancy .More over the adverse consequences of fetal growth restriction extend beyond early years into later life .The concept of developmental programming pioneered by Prof. David Barker & others has stimulated tremendous research into the origin of a spectrum of cardiovascular and metabolic disorders in adults .But the exact causes of fetal growth restriction in utero still remains unclear. Antenatal fetal surveillance plays a major role in identifying fetuses at risk of IUGR to offer them close monitoring to prevent the perinatal mortality & morbidity & long term consequences.

Deficiency in growth implies failure of the fetus to realise its genetically endowed growth potential .Determining the growth potential of an individual fetus however remains difficult. Many studies have produced normative gestational age specific birth weight standards that have been used to define retrospectively suboptimal fetal growth. Before the introduction of ultrasound, prospective measurement of fetal growth during pregnancy has been limited to

measuring uterine size and guessing fetal size by palpation. Over the last few decades, ultra sonogram & Doppler has come into play a major role in evaluation of fetal growth in utero.

DEFINITION;

IUGR can be defined as a condition in which the fetus fails to achieve its genetic growth potential and it is at increased risk of perinatal morbidity and mortality. A fetus is considered growth restricted when ultrasonographically measured fetal dimensions particularly AC or EFW from multiple biometric measurements, below a certain gestational age specific threshold. The most commonly used threshold is 10th percentile. This standard is arbitrary & it may lead to misdiagnosis of growth restriction. A more rigorous threshold such as 5th or 3rd percentile would be more specific but it is less sensitive.

CLASSIFICATION OF IUGR;

There are 3 types of IUGR based on time of onset & the pathological process.

TYPE 1 OR SYMMETRIC OR INTRINSIC IUGR:

Accounts for 20-30% of IUGR.

Due to growth inhibition early in pregnancy.

All parameters like BPD/ HC/AC /EFW are below 10th percentile& they have normal ponderal index.

Causes are mainly INFECTION IN UTERO (HERPES SIMPLEX, RUBELLA, CYTOMEGALO VIRUS, TOXOPLASMOSIS) Chromosomal disorders& congenital malformation.

Any insult in early phase of fetal development (4-20 wks) result in reduced number of cells in the fetus & overall reduction in growth potential.

These babies may not have immediate effect but they are at risk of long term complications like neurodevelopmental dysfunction.

TYPE 2 OR ASYMMETRIC IUGR:

Accounts for 70-80% of IUGR.

Due to placental insufficiency resulting from maternal condition or placental pathology.

Onset usually after 28 weeks.

In USG , BPD, HC remains normal, but AC& Ponderal index are low due to redistribution of blood flow from periphery to Brain and Heart.

These babies are at great risk of antepartum and intrapartum complications as well as neonatal morbidity and mortality. Moreover timely identification and interventions can reduce these complications.

TYPE 3 OR INTERMEDIATE IUGR:

Accounts for 5-10% of IUGR.

Combination of Type 1& Type 2 IUGR.

With this background this study has been conducted to know about the predictive value of placental volume that is measured antenatally by two dimensional ultrasound over the adverse prenatal outcome of the IUGR fetuses.

AIM OF THE STUDY:

1 To estimate the placental volume using 2 dimensional ultrasound.

2. To estimate the placental volume immediately following delivery.

3. To compare the placental volume measured before delivery by ultrasound with that of measured after delivery.

4. To compare the placental volume in IUGR and NORMAL pregnancy.

5. To correlate the adverse perinatal outcome with placental volume in IUGR pregnancy.

REVIEW OF LITERATURE

Fetal weight is determined by the genetic growth potential, the health of the fetus, the capacity of the mother to supply adequate substrate for growth and the ability of the placenta to transport the substrates to the fetus. Hence placenta acts as a vector for all nutrient exchange between the mother and the fetus & it has principle influence on the birth weight of the fetus.

DIAGNOSIS OF IUGR:

IUGR is suspected in patients with risk factors like preeclampsia, chronic renal disorders vasculopathy ,infections ,low pre pregnancy BMI ,poor maternal wt gain.

Determination of gestational age is the most important step in the diagnosis of IUGR.

1. Clinical method: Serial measurement of symphysio fundal height & abdominal circumference are the most common clinical methods. Symphysio fundal ht increases by 1 cm /wk& it coincides with the gestational age between 18-30 wks. A lag in the fundal ht of 4 wks is suggestive of moderate

IUGR& lag of >6 wks is suggestive of severe IUGR. This method has low sensitivity when used alone.

Both ACOG and RCOG recommend this simple technique to identify abnormal growth. ACOG suggests that symphysio fundal height measurement at 32-Both ACOG and RCOG recommend this simple technique to identify abnormal growth. ACOG suggests that symphysio fundal height measurement at 32-34 weeks has 70-85% sensitivity and 96% specificity in detecting IUGR. Whereas RCOG suggest that it has 27% sensitivity and 88% specificity in detecting IUGR.

Bakketeig et al (1984)[1] compared the clinical method with sonographic study and concluded that detection rate of IUGR for these two groups was similar (25% for ultrasound and 11% for symphysiofundal height; RR 1.36, 95% CI 0.93-1.99)

2. ULTRASONOGRAM: There are several parameters used in diagnosis of IUGR. Among that AC has highest sensitivity and greatest negative predictive value .An increase in AC less than 10mm in 2 wks has 85% sensitivity and 74% specificity in detecting IUGR. Various age independent morphometric ratios like HC/AC,/ FL/AC also been used in detection of IUGR.

Mckenna et al (2003)[2] studied ultrasound examination of the patients consisting of Estimated fetal weight, Amniotic fluid index and placental grade at 30-32 weeks and 36-37 weeks and the clinical methods like symphysiofundal height alone. They reported that the prevalence of IUGR was significantly lower in ultrasound examination (7%) than with clinical method (10%), (95% CI 0.50-0.89).

3. Doppler velocimetry: doppler has poor sensitivity in detecting IUGR. But the doppler changes correlates well with the outcome of the fetus. Changes in blood flow velocimetry of umbilical arteries is an early predictor of IUGR .Ductus venosus flow alteration is an accurate predictor of acidemia.

In idiopathic IUGR where there are no obvious fetal / maternal causes, the placenta might hold the key to the etiology. Various authors recorded contradictory histological and morphological findings while comparing the placenta of IUGR pregnancies to that of normal pregnancies.

ETIOLOGY OF IUGR:

Numerous maternal, fetal and placental disorders may interfere with normal mechanisms that regulate fetal growth resulting in IUGR.

MATERNAL FACTORS:

1. Maternal hypertensive disorders:

Hypertensive disorders present in 30-40% 0f pregnancies complicated with IUGR. Pre eclampsia, chronic hypertension with or without pre eclampsia, autoimmune disorder nephropathy ,pregestational diabetes are associated with maternal vasculopathy may lead to fetal growth restriction.

According to Odegard vattern/Nilsen et al (2000)[10], preeclampsia is associated with 4 fold increase of having IUGR babies(RR=4.2; 95% CI 2.2-8.0).

The worse the severity and the earlier the onset of pre eclampsia the lower the birth weight. Long,Abell ,Beisher (1980)[11] reported that, the decrease in birth weight was 5% in mild pre eclampsia(95% CI 3-6)& 12% with severe disease(95% CI 9-15) and it was 23% with early onset disease(95% CI 18-29).There is evidence that elevated diastolic blood pressure without proteinuria is associated with small

for gestational age but the risk is lower than that of proteinuric hypertension.According to Sibai (2002)[12] there is variable increase in small for gestational age infants with mild chronic hypertension in pregnancy(8-15.5%). Proteinuria occurring in early pregnancy is associated with elevated risk of fetal growth restriction(OR 2.8; 95% CI 1.6-5.0).

Moreover maternal antihypertensive therapy fails to improve fetal growth and some beta blockers like Atenolol increases the risk of growth restriction.

2. Maternal autoimmune disorders:

Any maternal auto immune disorders especially with vascular involvement are associated with adverse perinatal outcome.

Patients with antiphospholipid antibody syndrome shows significant increase in stillbirth.

SLE in pregnancy is associated with 3 fold increase in fetal death when APA is positive than negative.

In a prospective study by Yasudha,Takakuwa,Tokunaga et al (1995)[13] the relative risk of growth restriction with positive APA was 6.22% (95% CI 2.43-16).

3. Thrombophilia:

Controversy still remains in the association between IUGR and maternal Thrombophilia. Recent meta analysis of 10 case control studies by Howley/ Walker/ Rodger(2005)[14] showed a significant association between IUGR and presence of factor v leiden mutation(OR 2.7; 95% CI 1.3-5.5)& prothrombin gene variant (OR 2.5; 95% CI 1.3-5). The relationship between methylene tetra hydrofolate reductase mutation and IUGR still remains unsubstantiated.

4. Maternal life style:

Maternal use of various recreational drug & addictive substances is associated with IUGR. However causal relationship is difficult to establish often due to other associated confounding factors like malnutrition, multiple substance abuse ,stress and other lifestyle variables.

Maternal smoking is associated with decrease in EFW due to the carbon monoxide which interferes with fetal oxygenation and the vasoconstrictive property of nicotine/ kramer ms(1987)[15].

Cliver et al (1995)[16] noted in average birth weight reduction of 6% when smoking was continued throughout gestation compared with only 1.7% when it was stopped after 1 st trimester and this effect was appeared to be dose dependent and also increased by other cofactors like hypertension. Cnattingius,Mills et al (1997)[17] showed increased incidence of small for gestational infant when smoking was associated with hypertension than not associated with it(40% vs 5%).

Taking alcohol even 1 drink per day is associated with IUGR and low Apgar at birth(Windham et al 1995)[18].

Cocaine use in pregnancy is also associated with significant maternal and fetal effects including maternal stroke, cardiac arrhythmia, hypertension, placental abruption, fetal brain injury and still birth.

5. Therapeutic agents:

Antineoplastic agents, anticonvulsants such as phenytoin, Beta blockers and steroids are associated with IUGR.

6. Malnutrition:

The effect of maternal malnutrition on fetal growth depends upon the severity of deprivation & the period of gestation.

7. Environmental pollution:

Epidemiological investigations on the impact of environmental pollution on pregnancy outcome show significant but slight increase in the frequency of IUGR(Maisonet,Coree,Misra et al 2004)[19].

This effect was discernible even with relatively low concentration of gaseous pollutants such as So2 ,No2 ,CO, Ozone (Liu et al 2003)[20].

FETAL FACTORS:

1. Aneuploidy:

Fetal chromosomal anomalies are strongly associated with IUGR. About 7% of IUGR is attributable to aneuploidy.

Early growth restriction is associated with increased odds of trisomy 18& trisomy 13(Bagadosingh et al 1997)[21].

90% of trisomy 18 are associated with IUGR when compared to 30% in trisomy 21.

Fetuses with aneuploidy are associated with increased incidence of fetal malformations leading to higher frequency of somatic

asymmetry, increased or decreased amniotic fluid volume and normal doppler indices of umbilical and/ or uterine artery.

2. Genomic imprinting & uniparental disomy:

UPD is inheritance of both homologs of a chromosome from a single parent.

Several autosomal chromosomes and X-chromosomes have been implicated with UPD and are associated with IUGR.

Maternal UPD of chromosome 16 is the one most commonly associated with IUGR. Abnormal imprinting results in abnormal phenotypes including fetal growth restriction and dysmorphic features. In Prader willi syndrome loss of function of imprinted genes on the paternal allele in 15q11-13 leads to growth restriction in utero and associated with other developmental problems.

Maternal Uniparental disomy involving imprinted region in chromosome 7, clinically characterised by prenatal and postnatal growth deficits and dysmorphic features.

3. Fetal malformations:

A population based study conducted by CDC demonstrated >22% of infants with congenital malformations are growth restricted with relative risk of 2.6(Khoury, Erickson 1998)[22].

Multiple malformations increases the risk of IUGR and the frequency was increased from 20% in infants with two defects to 60% in infants with 9 defects. The cardiac anomalies most commonly associated with small for gestation are Tetrology of Fallot, Endocardial cushion defects, Hypoplastic left heart, Pulmonary stenosis, ventricular septal defect not only heart disease , anencephaly and anterior abdominal wall defects also associated with growth restriction in the fetus. A single umbilical artery even in the absence of other malformation or aneuploidy may be associated with fetal growth restriction.

4. Perinatal infections :

5-10% of IUGR are attributable to viral or protozoan infection in utero.

The viral infections most commonly associated with growth restriction are Rubella , Cytomegalovirus, Human immuno deficiency

virus and Varicella zoster. The early infection which leads to decrease in cell population may be the most frequently involved mechanism in growth restriction.

Protozoal infections like Malaria and Toxoplasmosis can also lead to growth restriction of the fetus. In malaria the adverse effects include maternal anemia, prematurity and growth restriction.

Bacterial infection usually not associated with growth restriction there is evidence suggest that subclinical infection and inflammation leading to chorioamnionitis may result in growth restriction. Offenbacher, Lieff et al (2001)[23] suggest that maternal periodontal disease can lead to preterm and small for gestational births and it could be a modifiable etiology of IUGR.

5. Multiple gestation:

In multiple gestation the maternal system has to provide optimum environment for individual fetus to sustain fetal growth. Individual fetuses in multiple pregnancy shows different growth profile than that of singleton pregnancy. Guenwald (1966)[24] demonstrated the growth curves of singleton and twins were same

upto 30-32 weeks after which the growth of the twins lagged behind that of singleton.

Small for gestational births are noted in 20% of dichorionic fetuses and 30% of the monochorionic fetuses. The aetiology for this is similar to that of singleton pregnancy and include hypertensive disorders , malformation, poor weight gain, low prepregnancy body mass index. An additional factor in multiple pregnancy is discordant growth before 30 weeks is associated with twin to twin transfusion syndrome and high risk of perinatal mortality.

PLACENTAL FACTORS:

Placenta being the lifeline between mother and the fetus has a critical role in IUGR. The role is however mediated by anatomic, vascular, chromosomal & morphological abnormality.

Abnormal placentation, placenta previa, chronic villitis, placental infarcts, haemorrhagic endovasculitis ,placental haemangioma, chorioangiomas are some of the placental conditions associated with IUGR.

COMPLICATIONS OF IUGR:

ANTENATAL:

Antenatal and intrapartum hypoxia ,acidosis are the most important and frequent complications of IUGR. According to Lin et al.,(1980)[3] the incidence of non reassuring fetal heart rate pattern in electronic fetal heart rate monitoring during labour is up to 40%.

STILL BIRTH:

Marana found (1980)[4] that 20% of all stillborns show evidence of IUGR. Morrisen and Olsen(1985)[5] found 26% of stillborn weighing <2.5 kgs is associated with IUGR.

OLIGOHYDROMNIOS:

Chamberlein et al (1984)[6] showed that the incidence of IUGR with normal amniotic fluid volume was <5% but when oligohydromnios was present it was up to 40%.

INTRAPARTUM COMPLICATIONS:

The incidence of intrapartum hypoxia and acidosis are high in IUGR. The incidence of caesarean section is increased due to

nonreassuring fetal heart rate pattern in electronic fetal heart rate monitoring.

EARLY NEONATAL COMPLICATIONS:

Respiratory distress syndrome: main cause of mortality and morbidity in IUGR.

Meconium aspiration syndrome also a major cause of mortality and morbidity.

persistent fetal circulation due to perinatal hypoxia and acidosis.

Intraventricular bleeding and perinatal leukomalacia are the most frequent neurological complications of preterm IUGR.

Neonatal encephalopathy is an essential component of cerebral palsy secondary to fetal asphyxia.

Hypoglycaemic episodes occur in 25% of term IUGR and 67% of preterm IUGR.

Hypocalcaemia can occur secondary to chronic hypoxia.

Hyper viscosity leading to necrotising enterocolitis, pulmonary infarcts, hyper bilirubinemia.

Hypothermia due inadequate subcutaneous fat.

LONG TERM PROGNOSIS:

Postnatal growth: Hill(1978) [7] showed that 30% of babies will remain below 30th percentile for their age and only 10-20% will be above 50th percentile.

Cerebral palsy: Follow-up studies showed that intelligence ,motor skills, speech and reading abilities are affected in IUGR babies.(Robertson et al.,(1990)[8]; Kok et al (1998),)[9.]

several studies showed incidence of chronic hypertension, abnormal lipid profile ,ischemic heart disease ,type 2 diabetes are increased in later life.

Salafia (1997)[31] proposed that not a single but several histological & morphological changes of placenta resulted in IUGR. Though the contribution of placental changes remained controversial , it was accepted that IUGR was associated with fetal hypoxia resulting partially from alteration in growth & development of placental villi & their underlying vasculature (Benrische, Kaufman 1995)[32]

PLACENTAL VASCULAR DEVELOPMENT IN NORMAL AND IUGR PREGNANCY:

Maldevelopment of uteroplacental & fetoplacental circulatory system has been shown to be associated with fetal growth compromise and pre eclampsia.

In the maternal placental circulation, a subset of trophoblasts invades the spiral endometrial arteries & remodel them into widely dilated uteroplacental arteries. As a consequence, the uteroplacental flow impendence progressively declined& the maternal blood flow through the intervillous space exponentially increases.

The changes in the uteroplacental arteries occur in 3 phases;

Before trophoblastic invasion, the arteries from both within and outside the implantation site show several changes including dilatation, vacuolation of endothelial cells and disrupted smooth muscle cells in the tunica media.

In the next phase, the interstitial trophoblasts surround the spiral arteries & induce fibrinoid deposition & other changes in the arterial media.

Finally, the trophoblasts invade the arteries & are transformed into immensely dilated conduits devoid of vasoactive capability.

These changes are more in the centre of the placenta than in the periphery.

FETOPLACENTAL ANGIOGENESIS & IUGR:

Feto placental angiogenesis is a continuous process starting soon after the implantation and evolving through pregnancy in 3 phases;

From post conception day 21-32, vasculogenesis occurs in which capillary networks are formed providing foundation for subsequent fetoplacental vascular & villous growth;

From 32nd day to 24 wks of gestation, branching angiogenesis dominates leading to the formation of 10-16 generations of stem villi.

Beyond 24 wks , the expansion of the feto placental vascular system is mainly by non branching angiogenesis characterised by elongation of the vessels rather than by branching.

According to Krebs & colleagues (1996)[25] and Todros & colleagues(1996)[26], abnormal development of villous tree has been shown to be associated with early onset pre eclampsia & IUGR.

PLACENTAL TRANSPORT MECHANISM & IUGR:

The concept of placental insufficiency in IUGR is by deficient maternal to fetal nutrient transport.

Invitro human placental experiments show diminished activity & expression of placental transporters for essential amino acids & ions in IUGR pregnancies (Cetin 2003)[27].

Deficiency in glucose transport mechanisms has been observed in preterm IUGR than in term IUGR placentas(Jansson, Yivar et al 2002)[28].

ASSESMENT OF PLACENTAL GROWTH:

There are so many standard placental growth parameters used in older birth cohorts are still in use.

1. Placental disk shape: Normal placenta is round to oval in shape. Naye(1992)[29] concluded that irregular placental shape was

associated with parent & sibling seizure disorder and adverse pregnancy outcome like preterm birth/ neurological abnormality @ 7yrs.

2. Location of umbilical cord insertion from the edge of the placenta: Cord malpositioning may be due to abnormal growth of placenta towards one side or abnormal positioning of the embryo. Nayes analysis suggested that marginal cord insertion was associated with twinning & major fetal malformation & also with maternal acetonuria during 1st trimester, Diabetes, IUGR.

3. Placental disk diameter: It determines the maximum number of spiral arteries that are involved in uteroplacental unit.

4. Disk thickness: Most of the placental growth in 3rd trimester is by increase in thickness which reflects the extent of nutrient exchange surface of the placenta essential for the successful and adequate fetal growth. Increased disk thickness decreases the placental efficiency and so abnormally thick placenta also associated with adverse pregnancy outcome(Raio,Ghazzi et al 2004)[30].

5. Feto placental weight ratio.

Only few workers had performed histomorphometric studies of the placenta associated with IUGR. Aherne & Dunnill(1996)[33] dealt with quantitative aspects of placental structure .They observed that IUGR infants born at term had placenta with reduced mean volume(350 ml). The mean values for volume proportions of chorionic villi did not differ from control.

In early 80s Geirsson et al[34] studied the use of measuring placental volumes in normal & abnormal pregnancies.

In 1984 the first fetal volumes acquired by USG were constructed by Brinkley et al. Since the development of 3 dimensional USG imaging assisted by computer technology it is possible to measure and calculate fetal & placental volume quickly & accurately .Measuring & monitoring fetal and placental volume at different gestational ages may improve our understanding about physiological and pathophysiological mechanisms in fetal & placental growth. Fetal and placental volumes have been used in screening of fetuses with chromosomal anomalies/ IUGR/preeclampsia. There are reports in literatures that increase in placental volume preceding pre

eclampsia & decrease in placental volume preceding IUGR & decrease in fetal volume in fetuses with chromosomal anomaly.

Wallace et al(2004)[35] concluded that it is the small size of the placenta per se rather than alteration in the nutrient metabolism or transferring capacity has a major limitation to fetal growth.

Thame & colleagues (2005)[36] have recently shown that the effects of maternal anthropometry on birth weight are likely to be mediated by effects of maternal anthropometry on placental volume. These effects operate early in pregnancy and alter both the absolute placental volume at 14 wks and rate of growth of placenta between 17 & 20 wks.

Clapp & colleagues (2004)[37] identified a robust relationship among the rate of increase in individual maternal IGF 1 levels after 16 wks , placental mass & neonatal fat mass.

Laviola, Perrini et al (2005)[38] showed an abnormal IGF signalling has been linked to human IUGR.

Lepereq & colleagues (2003)[39] showed Leptin may also contribute to this complex communication between mother, fetus & placenta may be an early Response Element to placental dysfunction.

I.Cetin G, Alvino (2009)[40] showed that IUGR correlates with a specific placental phenotype associated with defects in placental transport function that lead to fetal under nutrition. Both placental transport and metabolism may be affected thus modifying the nutritional supply to the fetus. In pregnancy, nutrient concentration can be measured at the time of delivery or at the time of cordocentesis. In IUGR the placental supply of aminoacid is significantly reduced independently from the severity of growth restriction and from the presence of hypoxia. Moreover maternal and fetal gradient of glucose are increased in severe IUGR.This summarizes the current knowledge about placental metabolism and transport in IUGR pregnancies and the relationship with the severity of the disease.

I Cetin,J M , Foidart, M Miazzo (2004)[41]

IUGR are associated with increased perinatal mortality and morbidity as well as cardiovascular disease and glucose intolerance in adult life. A number of genetic to metabolic , vascular , coagulative, autoimmune as well as infectious can influence fetal growth by damaging the placenta. Strict definition of IUGR and its severity are needed in order to eventually distinguish among different phenotypes such as gestational age at onset, degree of growth restriction and

presence of hypoxia. New existing findings on the genomic imprinting defects potentially associated with IUGR.

Marcus Rijken, Williams E Moroski,Suporn Kiricharo(2012)[42]

studied the effect of malaria on placental volume measured using 3 dimensional ultrasound. Malarial parasites and histopathological changes in placenta are associated with reduction in birth weight principally due to IUGR. They studied the feasibility of measuring early pregnancy volume by 3 dimensional ultrasound in malaria endemic area. They found that small placental volume in second trimester may be an indicator of IUGR and placental insufficiency.

Imdal,Aamer,Yakob, Mohammad Yawar(2011)[43]

Studied the correlation between stillbirth and IUGR.Early detection and management of IUGR can lead to reduced related morbidity and mortality. They reviewed the effectiveness of fetal movement count, doppler for detection and surveillance of high risk pregnancy and the effect of this in the prevention of stillbirth.They also reviewed the effect of Body mass index screening ,symphysio fundal height, target ultrasound in detection and triage of IUGR in the community.Finally they concluded that there is insufficient evidence

to recommend in favour or against fetal movement count for routein use of testing fetal wellbeing.Arterial doppler analysis and appropriate intervention is associated with 29% reduction in perinatal mortality (95% CI 2-48). Expert opinion suggest that detection and management of IUGR with the help of maternal Body mass index, symphysio fundal height., targeted ultrasound could be effective in reducing IUGR related stillbirth by 20%.

Hata T,Tanaka H, Noguchi J, Hata K (2011)[44]

Studied the effectiveness of conventional 2 dimensional ultrasound in evaluation of placenta during pregnancy.This 2 dimensional ultrasound evaluation includes morphology, anatomy, location, implantation, anomaly, size, power and pulsed doppler sonographic assessment of placenta. The introduction of 3 dimensional ultrasonography would facilitate the novel assessment of the placenta such as surface rendered imaging and volume assessment. The novel technique may assist in the evaluation of fetoplacental function and offer potential advantages relative to conventional 2 dimensional sonographic measurement.

Hafner,philipp schuchter (2002)[45]

Suggested that prognostic influence could be shown for placental volume , gestational age at the time of measurement and maternal weight at the time of registration.

Ferrazi,Bulfamante, Mezzopane (1998)[46]

Stated that the presence of abnormal doppler velocimetry of the uterine arteries in pregnancies with IUGR may be in fact an important indicator of hypoxic or ischemic placental lesions .This abnormal velocimetry is independent of the maternal blood pressure status.

Noguchi J,Tanaka H, Hata T (2009)[47]

Investigated placental vascular sonobiopsy using 3 dimensional ultrasound in normal and IUGR pregnancies. Placental vascular sonobiopsy using 3 dimensional power doppler ultrasound with VOCAL imaging was performed in 208 normal fetuses between 12-40 weeks and 13 pregnancies with IUGR between 22-39 weeks gestation. 3dimensional power doppler indices related to placental vascularisation were calculated. They found that placental vascular sonography may provide new information in the assessment of placental vascularisation in normal and IUGR pregnancies and placental perfusion is reduced in IUGR compared to normal.

Jang,DongGyu, Jo,Yun Sung, Lee(2011)[48]

Evaluated perinatal outcome and maternal characteristics in IUGR with absent or reversal of end diastolic flow (AEDV) independent of oligohydromnios, gestational age, and maternal factors. They compared 57 normal and 19 patients with Absent end diastolic flow. They found that gestational age was lower in AEDV group when compared to normal group.The birthweight and platelet count were lower in AEDV group and serum SGOT , non reassuring CTG were higher independent of gestational age. Perinatal outcome such as Apgar at 1 minute <4 ,use of ventilator , admission to NICU, respiratory disease, neurological disease, neonatal sepsis, anaemia, thrombocytopenia, and neonatal mortality were statistically less favourable in AEDV group.

Hafner et al (1998) revealed that the measurement of placental volume between 16 & 23 wks of gestation has a sensitivity of 53.5% for prediction of IUGR and neonatal birth wt below 10th percentile.

HAFNER, PHILIPP, SCHUCHTER(2002)[49]

Conducted prospective study in 382 women with singleton uncomplicated pregnancies at 16-23 wks to investigate the value of

2nd trimester 3-dimensional sonographic placental volume measurement to predict infants who are <10th percentile for birth weight .They inferred that placental volume estimation in predicting IUGR had 82.5% sensitivity & 52.5% specificity and prognostic influence could be shown for placental volume($p<0.0001$), gestational age at the time of measurement($p=0.0002$) & maternal weight at the time of registration($p=0.0025$).They concluded that 3 -dimensional sonographic measurement of placental volume alone is not satisfactory technique of predicting IUGR.

GIUSEPPE, RIZZO, ALESSANDRA CAPPONI(2008)[50]

Compared the efficacy of uterine artery doppler velocimetry & 3-dimensional sonographic measurement of placental volume, alone or in combination at 11-14 wks of gestation as a predictor for development of pre eclampsia. It was a prospective study involving 348 women who were scheduled for a routein prenatal ultrasonogram at 11-14 wks & the mean pulsatility index of uterine artery was calculated and, placental was volume measured using 3-dimensional sonogram. The outcome considered were development of pre eclampsia & pre eclampsia requiring delivery < 32 wks. On observation they found that the placental volume was significantly

lower in women who developed pre eclampsia later($p<0.003$).There was no relationship between placental volume & mean uterine artery pulsatility index($p=0.327$).Both showed similar sensitivities in predicting pre eclampsia(60% vs 66%) & pre eclampsia requiring delivery before 32 wks (66.7% vs 67%). The combination of both gave better results with sensitivity of 68.7% in predicting pre eclampsia& 83.3% for requiring delivery <32 wks.So they concluded that the combination of abnormal uterine artery doppler & low placental volume at 11-14 wks achieves better results than done alone.

CHRISTIANE KREBS, LENA.M MACERA, RUDOLF LEISSSER (1998)[51]

They evaluated the structure of placental terminal villi & their capillaries in pregnancies complicated by IUGR with absent end diastolic flow in umbilical artery. 10 placental specimens were taken from IUGR pregnancies and from well matched normal pregnancies as control.The structure and dimensions of 20 terminal capillary loops were determined by electron microscopic examination & their appearance were correlated with peripheral villi.The result observed was in the IUGR cases the capillary loops were sparse in no, & significantly longer than control cases(218 vs 137µm).They also

exhibited fewer branches (4/loop vs 6/loop, p<0.06) and the majority of the loops were uncoiled (79% vs 18% ,p<0.06).From this they concluded that the terminal villous compartment of the placenta appeared to be maldeveloped in IUGR with absent end diastolic flow in umbilical artery before delivery. These findings were consistent with increased fetoplacental vascular impedence at capillary level & it might account for the impaired gas and nutrient transfer across the placenta.

THAME, OSMONDE,WIKS[52]

They investigated the ability of 2nd trimester placental volume measurement by ultrasonogram in predicting the birth weight of the fetus. They selected 512 women and measured fetal anthropometry & placental volume serially at 14,17 ,20 wks . The outcome was focussed on birth weight, anthropometric measurement at birth, & placental weight. The result of the study was the placental volume positively correlated with all birth measurements. The Head circumference was the strongest predictor of birth weight at 14 wks (p=0.014) & 17 wks (p= 0.012), but at 20 wks abdominal circumference was the strongest predictor. Finally they have concluded that low birth weight was often preceded by small placental

volume in 2nd trimester. Hence placental volume may be a more reliable predictor of birth weight than fetal anthropometry & it may be useful in early identification of fetus at risk.

HUMBERTO AZPURUA, EDMUND F.FUNAI, LUISA M.CORALLUZI[53]

Conducted a prospective study involving 29 3rd trimester pregnancies & estimated placental volume with 2 dimensional ultrasonogram before 48 hrs of delivery. After delivery also they calculated placental volume, and compared these two. They found significant correlation between the estimated placental volume and actual placental volume after birth. They concluded that placental volume can be accurately predicted by 2 dimensional ultrasound with volumetric calculation.

MATERIALS AND METHODS

This prospective analytical study was conducted at The Institute of obstetrics and gynaecology, Egmore, Chennai coming under the Madras medical college, Chennai from 2011 to 2012. Ethical committee clearance was obtained to undergo the study.

The patients referred as IUGR beyond 34 wks up to term were carefully analysed. The inclusion criteria used were,

1. with singleton pregnancy

2. well known gestational age

3. without any maternal medical complications,

4. with first trimester ultrasound for confirming the gestational age and second trimester ultrasound to rule out fetal anomaly and serial ultrasound to see the interval growth.

These patients were screened with clinical method of measuring fundal height. If it was lagging behind 4 weeks for their gestational age, then they were subjected to ultrasound and fetal biometry and estimated fetal weight were measured.

Estimated fetal weight of < 10th percentile for their gestational age with ultrasound were selected for the study after getting informed consent.

Exclusion criteria:

Patients with multiple pregnancy, abnormal placentation, fetal malformation were excluded.

Patients with severe oligohydromnios in which there was difficulty in localising the placenta were excluded from this study.

And also in patients in whom there was difficulty in localising as well as measuring the placenta due to fundal or lateral wall insertion were excluded.

Detailed history was taken & patients with hypertension, diabetes, other medical disorders were excluded to avoid errors in monitoring the perinatal outcome.

Examination of the selected patients:

Name, age, unit, Registration number, Address, socioeconomic status, occupation were noted.

In multigravidas, detailed history of previous pregnancies including duration of pregnancy, mode of delivery, birth weight of the baby, perinatal outcome and pregnancy complications like gestational hypertension, pre eclampsia, gestational diabetes mellitus were elicited.

Details of present pregnancy including last menstrual period, 1st trimester ultrasonogram, any h/o bleeding episodes, / h/o fever episodes in the first trimester were noted.

Details about second trimester including the targeted ultrasound to rule out fetal anomaly, h/o iron and folicacid intake, immunisation, any history suggestive of preeclampsia were recorded.

Regarding third trimester, the follow-up ultrasound to assess the interval growth, history suggestive of pre eclampsia were recorded.

Detailed clinical examination of the patient was done & height, weight, BMI, blood pressure were noted . Routine laboratory investigations also done. Obstetric examination was done & a lag in fundal height of more than 4 weeks taken into consideration. Those patients selected for the study were subjected to ultrasound examination.

Ultrasound examination:

The machine used for 2Dimensional ultrasound examination was GE with a 5 MHz curvilinear probe.

Fetal parameters like BPD,HC ,AC, FL, were measured as described below.Estimated fetal weight was calculated with the above measurements by ultrasound and confirmed whether it was <10th percentile.

Amniotic fluid index was also done. placental localisation was done .

The probe was adjusted for seeing both edges of the placenta in the same image and the image was frozen. With this placental width and height were measured. Then placental thickness was measured possibly at the level of cord insertion.

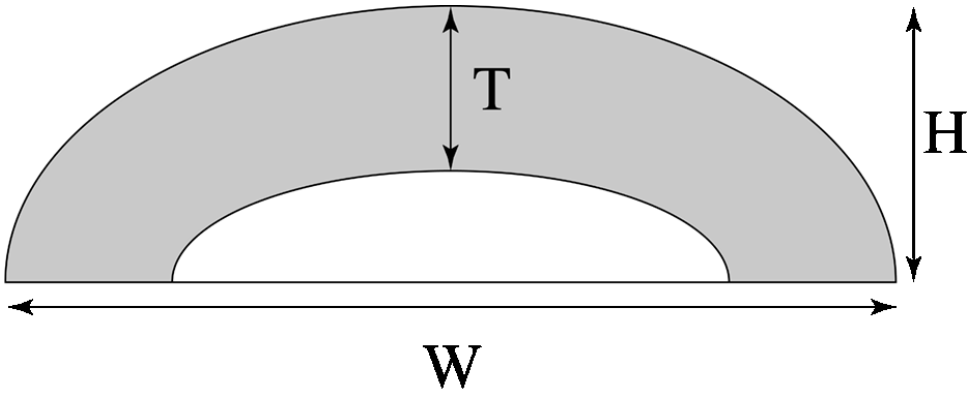
Measurement of placental volume was done by using the convex-concave shell formula.

$V=\pi T/6\times(4H(W-T) +W(W-4T)+4T^2)$;

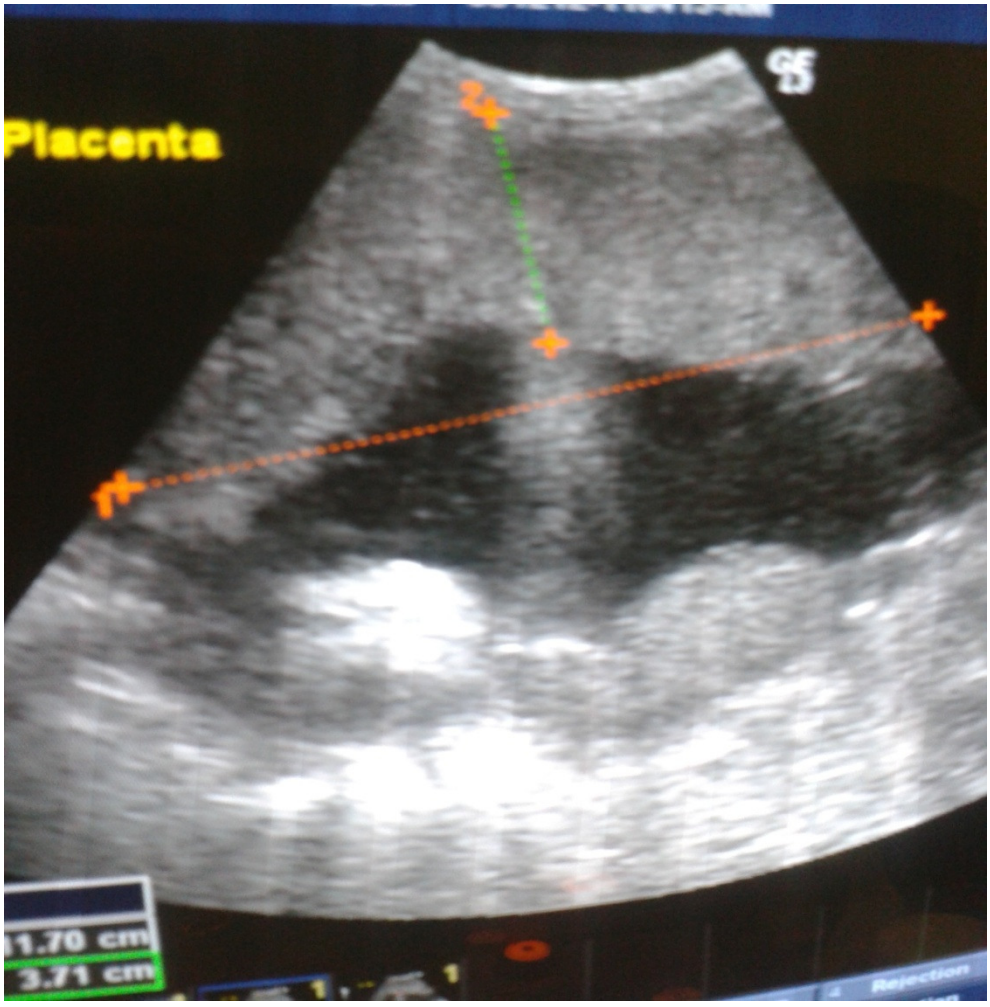
H=PLACENTAL HEIGHT,

T= PLACENTAL THICKNESS,

W= PLACENTAL WIDTH.



Diagrammatic representation of measurement of placental volume



This picture shows 2 dimensional measurement of placental width and thickness.

Doppler study was done on the umbilical artery, middle cerebral artery as described below. Umbilical cord was located in the pool of Amniotic fluid and the middle cerebral artery was localised in the transverse section of the fetal skull at the level of thalamus in the sylvian fissure. The doppler signals appropriate for the vessels were identified. The signals were recorded for a minimum of 5-8 cycles with blood flow velocity waveforms of equal shape and amplitude and of satisfactory quality were obtained. The image was frozen and the measurements of RI (RESISTANCE INDEX) was taken. Cerebroplacental ratio was calculated from the RI of umbilical and middle cerebral artery (RI of MCA/ RI of UA). Doppler was considered abnormal when the RI value above 95 th percentile for the gestational age in umbilical and middle cerebral artery or there was absent / reversal of diastolic flow in umbilical artery or CPR <1.

Patients with normal fetal growth were selected as control. The inclusion criteria for selection were same that of IUGR to avoid errors in comparison. Patients with singleton pregnancy, well known gestational age, appropriate interval growth in previous serial ultrasound, without any systemic medical disorder were included in the study.

Patients with multiple pregnancy, fetal anomaly, or abnormal placentation and with maternal complications were excluded.

In this group also detailed history was elicited. Details of this pregnancy like last menstrual period, 1st trimester ultrasonogram, 2nd trimester anomaly scan,3rd trimester interval growth were noted. In multigravidas, history regarding previous pregnancy & its outcome and any pregnancy complications were recorded.

Detailed clinical examination was done. ultrasonography was also done & the fetal biometry, AFI, placental localisation, placental volume were measured in the same way. Here also patients with difficulty in localising the placenta were excluded from this study.

All cases were observed till delivery .patients were followed up with fetal surveillance with daily fetal movement count, modified biophysical profile , repeat ultrasonogram if needed to observe the interval growth. Once decided for termination, Placental volume by 2 dimensional ultrasound was repeated if done 48 hrs before delivery.

Mode of delivery was noted. In case of vaginal delivery, careful intrapartum monitoring done. If decided for caesarean section, the indication was noted.

At delivery, baby was looked for APGAR score at 1 and 5 minutes. colour of liquor, meconium staining of umbilical cord were noted. Birth weight of the baby was taken.

After delivery of the placenta the cord was immediately tied close to the insertion to prevent the loss of blood from the placenta. The remaining cord was cut. Membranes were trimmed from the edge. The placenta was kept on a flat surface and maximum, minimal width were measured with an inch tape. Maximum height was measured. With all these measurements, placental volume was calculated by the following formula;

$V=\pi$ ABH.

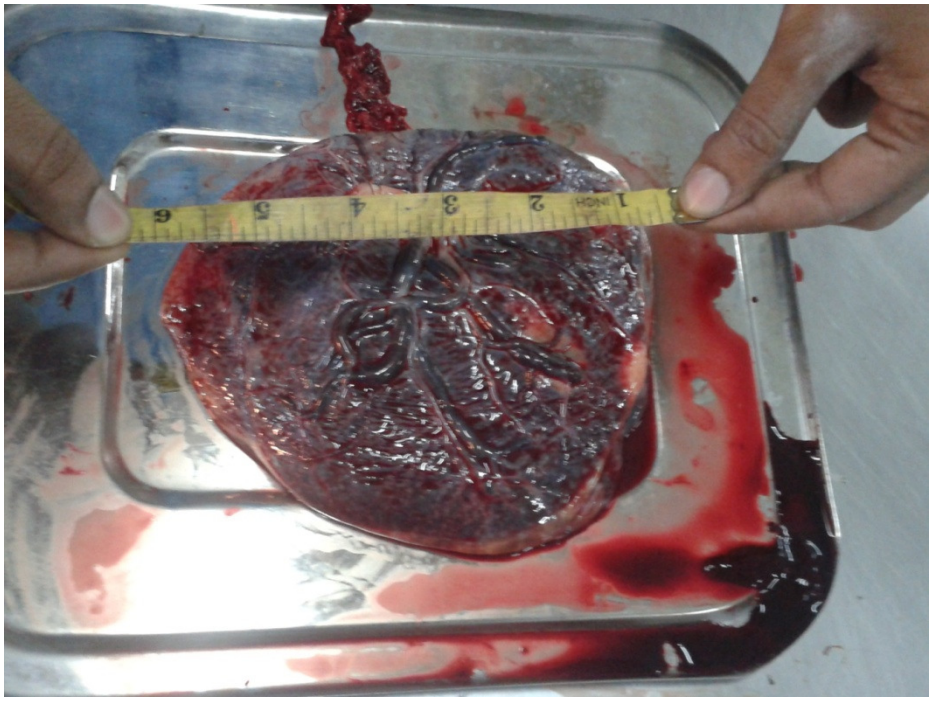
A=Major width,

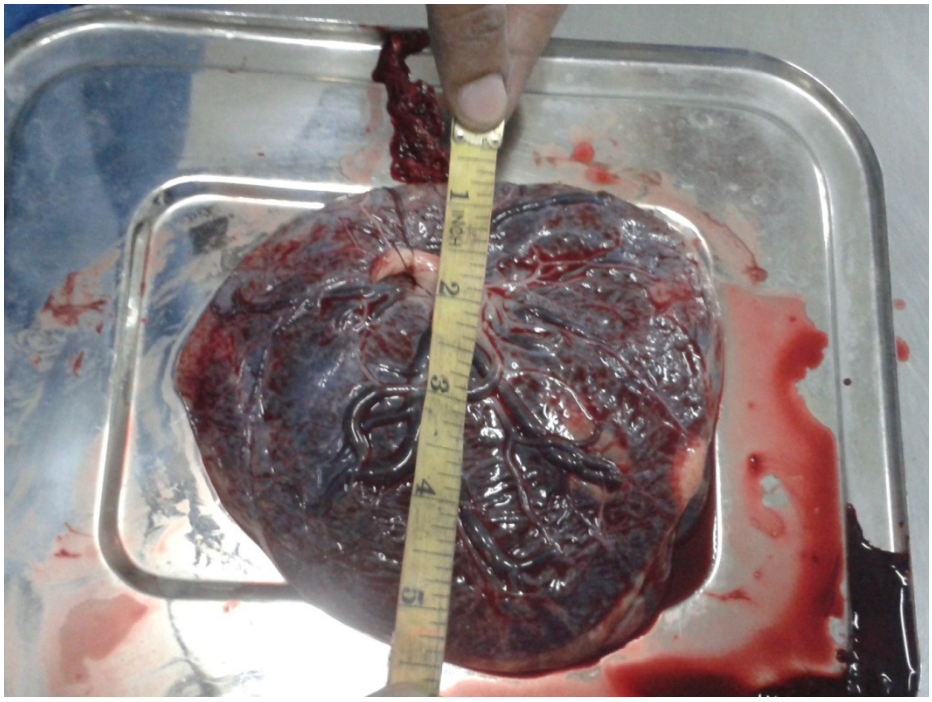
B=Minor width,

H=Height.

The placental volume measured before delivery was compared with that of after delivery.

PLACENTAL MEASUREMENT AFTER DELIVERY





METHODS OF ULTRASOUND AND DOPPLER MEASUREMENT:

BIPARIETAL DIAMETER:

Biparietal diameter helps to determine the gestational age and type of IUGR. But using BPD alone for diagnosing IUGR has poor sensitivity. According to Campbell S, Deuhurst (1971)[54] when BPD is below 5 th percentile , 82% of birth weight are below 10 th percentile. BPD may also give false positive result due to alteration in shape of the head as in brachycephaly or dolichocephaly.

It is a two dimensional measurement. Any plane of section through 360 degree arc that passes through the thalami and 3rd ventricle is acceptable for measuring BPD & it is measured from outer edge of the fetal skull on the proximal surface to the inner edge of skull on the distal surface.

HEAD CIRCUMFERENCE:

HC is better than BPD in predicting IUGR as it is not subjected to variability.

It is measured at the same level of BPD using the method of expanding ellipse.

FEMUR LENGTH:

FL is an excellent parameter to calculate gestational age, as it is not significantly affected by IUGR.

It is a single dimensional measurement. The transducer is aligned to the long axis of the diaphysis of the bone to obtain a proper plan of section .Only the ossified portions of the diaphysis and the metaphysis are measured .Proper alignment of the transducer to the long axis of the bone is ensured by demonstrating that both the femoral head or greater trochanter and the femoral condyle are simultaneously in the plane of section.

ABDOMINAL CIRCUMFERENCE:

AC has highest sensitivity and greatest negative predictive value in diagnosis of IUGR.AC value < 10 th percentile for gestational age has negative predictive value of 93% and positive predictive value of 47% in diagnosis of IUGR. AC value of < 5th percentile has negative predictive value of 93% and positive predictive value of 67%. AC of > 25 th percentile has negative predictive value of > 95%.

It is three dimensional measurement. The AC is measured at a position where the transverse diameter of the liver is greatest. It is

determined sonographically as the position where the right and left portal veins are continuous with one another.

ESTIMATED FETAL WEIGHT:

Determination of estimated fetal weight by ultrasonogram requires accurate measurement of BPD,HC , AC, FL. According to Ott, (1997)[55], fetal weight estimation has sensitivity of 89%,specificity of 88%, positive predictive value of 45%, negative predictive value of 99% in detection of IUGR.

According to Chervenac et al (1984)[56] when EFW is below 0.5% confidence limit the probability of IUGR is 82% and if it is between 0.5%-20% confidence limit, the probability is 24%.

PARAMETER	BPD	AC	FL	EFW
SENSITIVITY	75%	95%	45%	65%
SPECIFICITY	70%	60%	97%	96%
POSITIVE PREDICTIVE VALUE	21%	21%	64%	65%
NEGATIVE PREDICTIVE VALUE	96%	99%	94%	96%

DOPPLER STUDIES:

The Doppler principle was first described by Johann christian Doppler in 1842.The use of doppler in the evaluation of fetal circulation has been adequately assesed in randomized control trials and it has been found to be useful. The use of doppler in obstetrics requires adequate understanding of feto-placental and materno-placental circulation. The doppler study of arterial and venous system of the feto-placental unit has been found to be useful,

- In complementing other methods of fetal surveillance such as NST, BPP in more precisely determining the degree of fetal compromise.

- as a follow up test when other tests of fetal well being give ambiguous results,

- in identifying high risk of placental insufficiency and fetal complications,

- in evaluating the presence and severity of fetal anemia.

There are several methods of analysing doppler wave form to provide a quantitative index of vascular resistance namely S/D Ratio, PI(Pulsatility Index), RI(Resistance Index). The objective of these

indices is to obtain a numerical value from the wave form , so that we can asses the resistance to the blood flow of the vessel being studied.

S/D RATIO=Mean systolic velocity/Mean diastolic velocity.

PI=systolic velocity-diastolic velocity/mean velocity.

RI=systolic velocity-diastolic velocity/systolic velocity.

In this study we have taken the RI as an index of vascular impedence.

Umbilical artery:

The umbilical artery doppler provides the index of resistance to blood flow on the fetal side of the placenta.

A loop of umbilical cord midway between the fetal and placental insertion was located. Because measurement close to the placental insertion shows high resistance flow and close to the fetal insertion shows low resistance. That segment of umbilical cord is elongated so that 2 umbilical artery and 1 umbilical vein could be distinguished. Angle of insonation was adjusted to < 60 degrees. An optimum doppler signal was obtained and the Resistance index was measured.

GESTATIONAL AGE	RESISTANCE INDEX
34 WKS	0.62-0.74
35 WKS	0.61-0.73
36 WKS	0.59-0.72
37 WKS	0.58-0.71
38 WKS	0.57-0.70
39 WKS	0.56-0.69
40 WKS	0.55-0.68

The resistance to the blood flow through the umbilical artery decreases as the gestational age advances .Whenever there is placental insufficiency, there are certain adaptive changes that takes place in the fetal circulation which can be observed in doppler waveforms. The sequence of events are as follows.

1. Increased umbilical artery resistance without centralisation of flow.
2. Increased umbilical artery resistance with centralisation of flow.
3. Absent diastolic flow in the umbilical artery.
4. Reversed diastolic flow in the umbilical artery.
5. Alteration in venous circulation.

The initial phases indicates the fetal compensatory mechanisms to increased placental vascular resistance. When the diastolic flow in the umbilical artery becomes absent or reversed, it indicates that the fetal compensatory mechanisms exhausted and hypoxia and acidosis has set in. Alterations in venous circulation indicates the fetus is in hemodynamic decompensation and at risk of imminent death.

Middle cerebral artery:

When the placental resistance increased to a certain threshold, the fetus develops a compensatory response by increasing blood flow to the vital organs like Brain & Heart , and decreases blood flow to peripheral organs.This is evidenced in doppler study as decrease in resistance of middle cerebral artery blood flow which originally has high resistance flow. This centralization indicates the fetal compensatory mechanism to the increased resistance to the blood flow.

Section of fetal skull used for BPD measurement was obtained and then the transducer was angulated caudally till the middle cerebral artery courses along the sphenoid wings. The volume size and angle of insonation were adjusted after placing the cursor over the artery and appropriate signals were obtained and the RI was measured.

GESTATIONAL AGE	RESISTANCE INDEX OF MCA
34 WKS	0.73-0.86
35 WKS	0.72-0.85
36 WKS	0.70-0.83
37 WKS	0.68-0.81
38 WKS	0.66-0.80
39 WKS	0.63-0.78
40 WKS	0.61-0.76

The MCA resistance index also decreases with gestational age but remains higher than that of umbilical artery.

CEREBRO PLACENTAL RATIO:

It is the ratio between RI of MCA & RI of UA. According to Arias (1994)[57], CPR<1 identifies the fetuses at risk of IUGR and poor perinatal outcome.The predictive value of the CPR loses after 34 weeks (Bahado Singh et al 1999.

OBSERVATION AND RESULTS

This prospective analytical study was conducted with 100 IUGR patients as study group and 100 normal pregnancy as control group. The following observations were made.

1. GESTATIONAL AGE:

In our study IUGR above 34 weeks of gestation were taken excluding extreme prematurity. The number of patients in normal pregnancy were selected according to this gestational age for better comparison. The number of patients presented in both group were,

GESTATIONAL AGE	IUGR	NORMAL PREGNANCY
34-36 WKS	47	50
36-37 WKS	22	25
37-38 WKS	13	20
38-40 WKS	8	5

According to the above data, the commonest gestational age group presented was 34-37 weeks (n=69).

2. MATERNAL AGE:

In IUGR group ,71 patients were presented in the age group of 28-36 years. So patients in the normal pregnancy group also selected according to this to avoid errors in comparison. The age wise distribution of patients in both age group was,

AGE	IUGR	NORMAL PREGNANCY
18-22 YRS	11	13
23-27 YRS	15	20
28-31 YRS	25	21
32-36 YRS	41	44
>36 YRS	8	1

3. PARITY:

In our study both primi gravidas and multigravidas presented equally & patients in normal group were also selected like that.

PARITY	IUGR	NORMAL PREGNANCY
PRIMI	49	48
MULTI	51	52

4. PLACENTAL VOLUME:

Placental volume was measured in all the patients in our study group by 2 dimensional ultrasound as described above with in 48 hrs of delivery. The average placental volume observed according to gestational age were as follows.

GESTATIONAL AGE	IUGR	NORMAL PREGNANCY
34-36 WKS	335	552
36-37 WKS	424	578
37-38 WKS	469	604
38-40 WKS	574	647

5. DOPPLER ANALYSIS:

All patients in IUGR group were subjected to doppler study. The findings were,

CPR <1	52
CPR >1	48

6. MODE OF DELIVERY:

All patients were observed till delivery. Mode of termination was noted. Method of induction, indications for caesarean section were observed.

MODE OF DELIVERY	NO OF PATIENTS
VAGINAL	26
LSCS	74

7. BIRTH WEIGHT OF THE BABY:

The birth weight of the baby in IUGR group was noted.

BIRTH WEIGHT	NO OF BABIES
<1 KG	2
1-1.5 KG	17
1.6-2.0 KG	47
2.1-2.5 KG	34

8. OUT COME OF THE BABY:

The outcome of the babies in IUGR group was observed. Among 100 babies 61 babies had good outcome without any perinatal mortality or morbidity. The remaining 39 babies had adverse outcome in the form of IUD(2), neonatal death (9),low apgar (18),MSAF(10).

Outcome of the babies	No of babies
Good outcome	61
IUD	2
NND	9
Low APGAR	19
MSAF	9

DISCUSSION

The above datas obtained from this study were analysed by statistical methods appropriate for the variables compared.

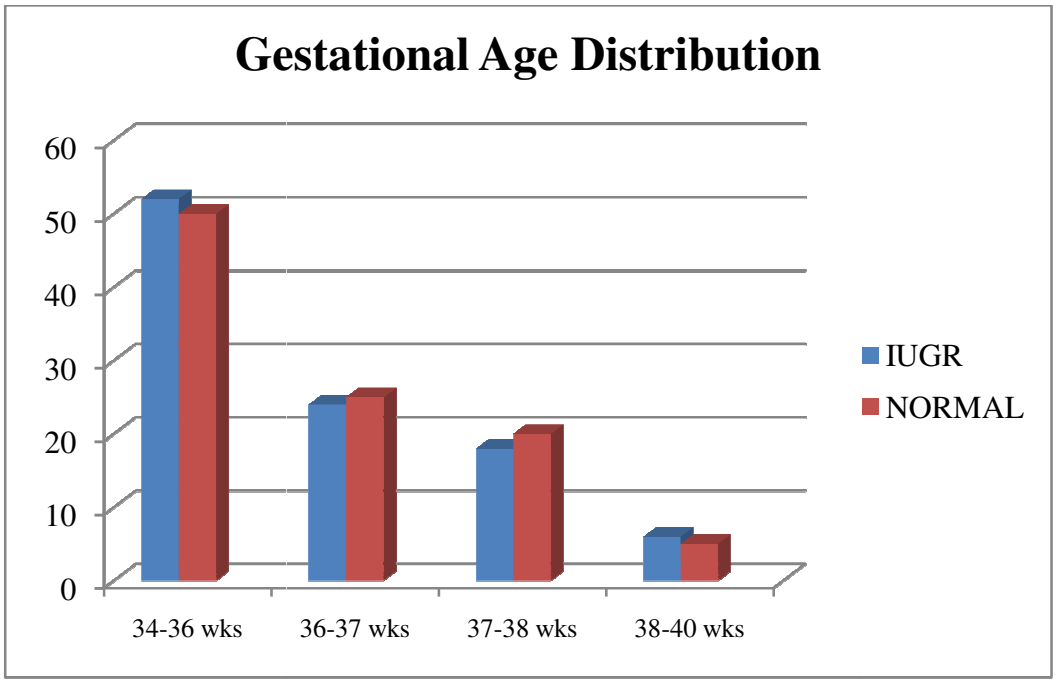
Comparison of variables between IUGR and NORMAL pregnancy groups:

1. GESTATIONAL AGE:

In this study gestational age above 34 weeks were taken. This is because most of the patients referred from periphery to tertiary care centre as IUGR for NICU care in late third trimester only. Very Preterm IUGR were excluded from the study to avoid errors in assessing perinatal outcome.

Among 100 patients with IUGR, 76 patients were between 34-37 weeks(76%).Patients between 38-40 weeks were 24 only (24%).This showed the incidence of early IUGR is more common than that of late IUGR.

The number of patients with normal pregnancy were selected similar to the number of patients with IUGR in accordance to the gestational age.

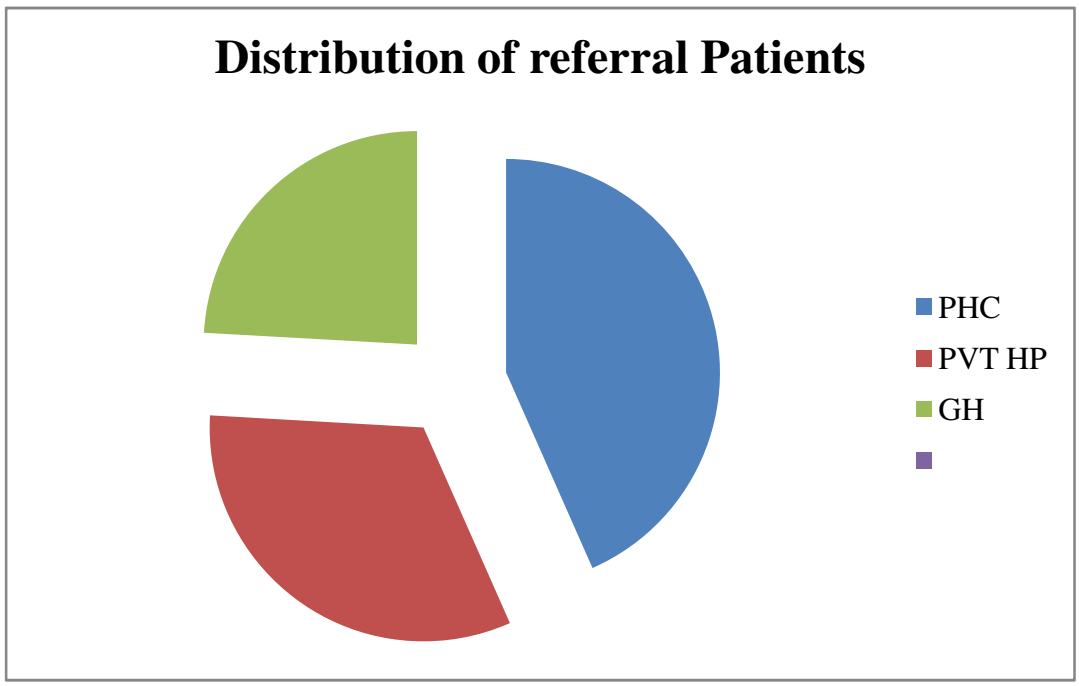


This showed the maximum number of IUGR presented in this study was between 34-36 weeks.

Among these patients, 83 patients were referred from various government and private hospitals as ?IUGR or diagnosed there as IUGR and referred here for neonatal care.

The remaining 17 patients were diagnosed as IUGR at their first booking visit at IOG in 3 rd trimester.

REFERRAL	NO OF PATIENTS
FROM PRIMARY CARE HOSPITALS AS ? IUGR	36
FROM PRIVATE HOSPITAL WITH DOPPLER CHANGES	19
FROM PRIVATE HOSPITAL WITHOUT DOPPLER STUDY	8
FROM OTHER GOVT. HOSPITALS FOR NICU CARE	20



This showed more number of patients were referred as suspected IUGR from various government hospitals including primary

health care centre as well as secondary care centre where facilities for proper evaluation of IUGR not available.

These patients were included in this study after confirming IUGR with clinical examination& previous serial ultrasonogram findings.

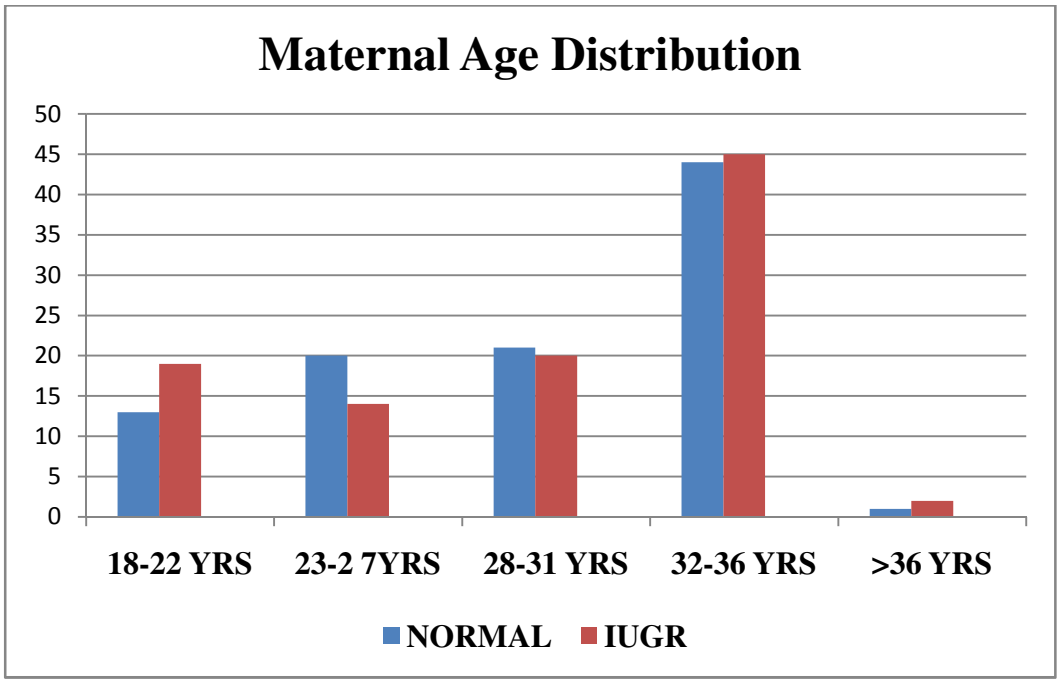
2. MATERNAL AGE:

In patients with IUGR, 71 patients were in the age group of 28-36 years.

This is comparable with the study by Odibo AO, Nelson D (2006)[58] noted that there was a positive association with increasing maternal age& IUGR. They concluded that advancing maternal age is an independent risk factor for IUGR.

The patients in the control group with normal pregnancy also selected according to this to avoid errors in comparison.

The most common age group presented was 32-36 yrs.

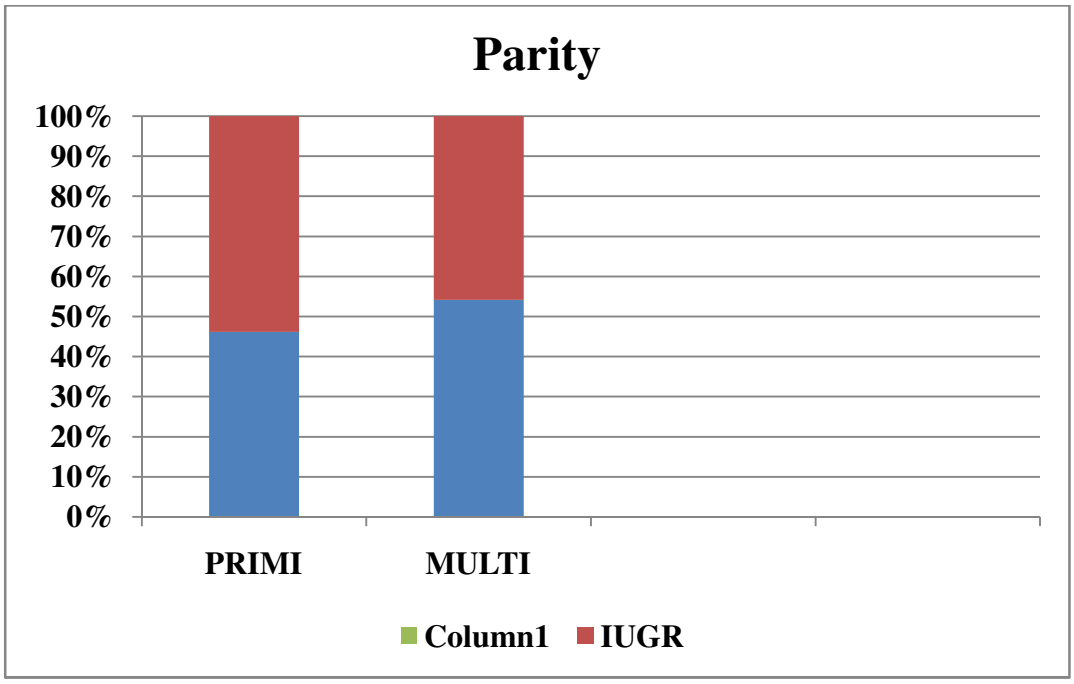


This showed there was positive correlation between advancing maternal age and IUGR. This denotes that advancing maternal age may be an independent risk factor for IUGR.

3.COMPARISON OF PARITY:

In our study both primi and multi were presented equally.

Taj mohammad, Asmat ara (2010)[59] concluded that primiparity was also a significant risk factor for IUGR . Similar findings were reported by Fikree et al[60] & Thompson et al[61].



Patterson RM, Gibbs,Woods (1986)[62] reported, the prevalence of recurrent IUGR was significantly related to the severity of growth restriction in previous pregnancy & severe placental insufficiency had 10% recurrence risk.

In our study group of IUGR, among the multigravidas 11 patients had h/o previous low birth weight babies . Among the 11 babies 3 were died in the neonatal period due to sepsis.

The rest of the multigravidas had no details regarding previous pregnancy.

PARITY	H/O IUGR	GOOD OUTCOME	NND
1 LIVE CHILD	9	8	1
>1 LIVE CHILD	-	-	-
NO LIVE CHILD	2	-	2

The recurrence rate could not be analysed properly because of insufficient datas.

4. COMPARISON OF PLACENTAL VOLUME:

De paula CF,ruano R,Campos JA (2008)[63] developed nomograms for placental volume in normal pregnancies from 12-40 weeks by measuring it with 3 dimensional ultrasonography. The placental volume measured in our study was compared with that.

Gestational Age	PV 10 th percentile (cm^3)	PV 50 th percentile (cm^3)	PV 90 th percentile (cm^3)
34 wks	189	353	530
35 wks	195	366	549
36wks	201	378	568
37 wks	207	390	587
38 wks	213	403	606
39 wks	219	415	624
40 wks	225	427	643

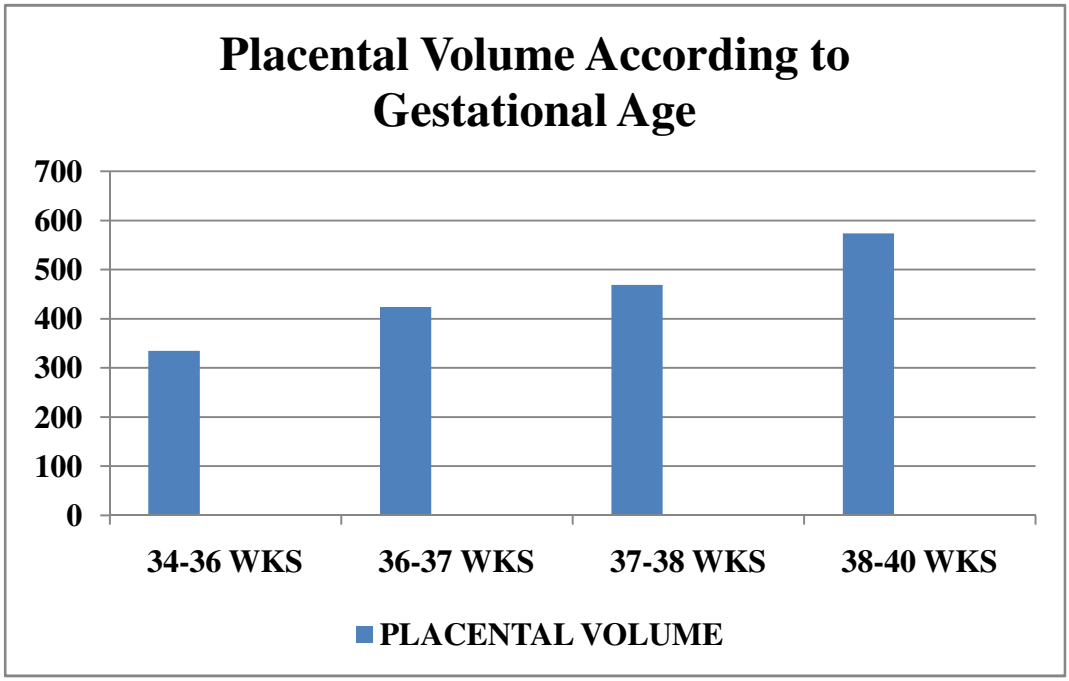
Based upon the above nomograms, the Placental volume was graded into 3 types as follows,

1. Grade -1: The placental volume falls above 50th percentile but below 90th percentile.

2. Grade-2: The placental volume falls below 50 th percentile but above10 th percentile.

3. Grade-3: There is severe reduction in placental volume & falls below 10 th percentile.

The average placental volume observed according to gestational age in pts with IUGR.

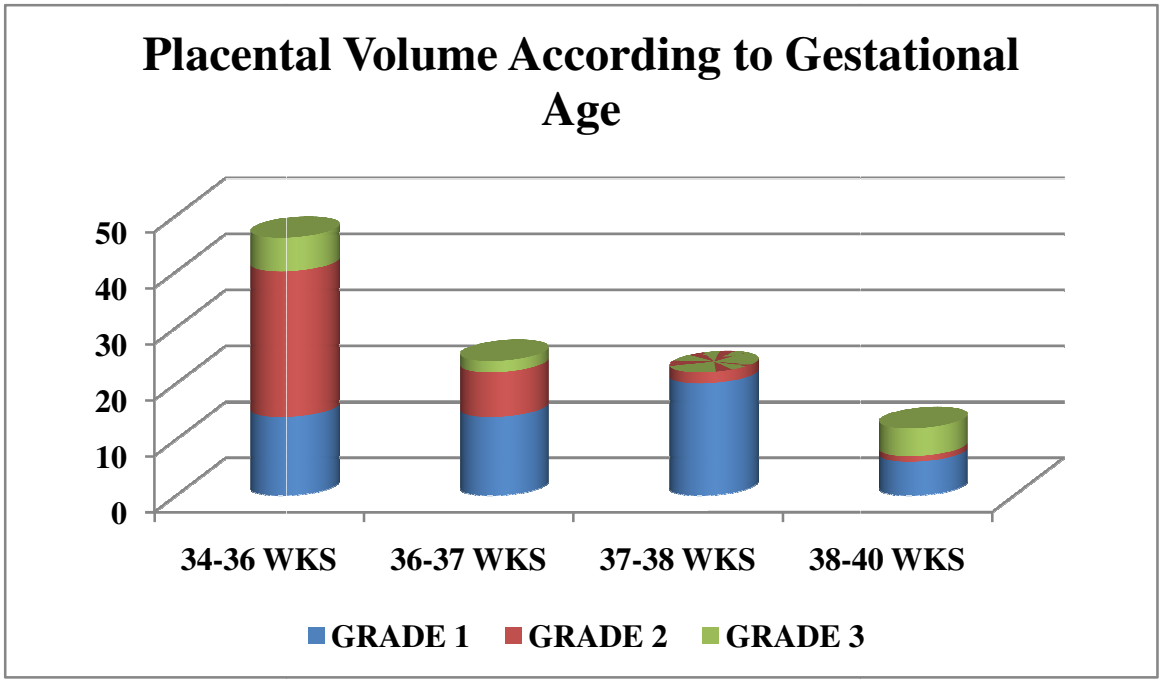


When comparing the placental volume of 34-37 weeks with that of 38-40 weeks there is more significant reduction of placental volume was noted in the early group of IUGR which were of preterm pregnancies. As the gestational age advances the reduction in the placental volume became less. This indicates the placental insufficiency may be more severe when it occurs in preterm than in term pregnancy.

The placental volume according to the gestational age further divided into 3 grades and compared.

GESTATIONAL AGE	GRADE 1	GRADE 2	GRADE 3	TOTAL
34-36 WKS	15	26	6	47
36-37 WKS	12	8	2	22
37-38 WKS	20	1	2	23
38-40 WKS	7	-	1	8

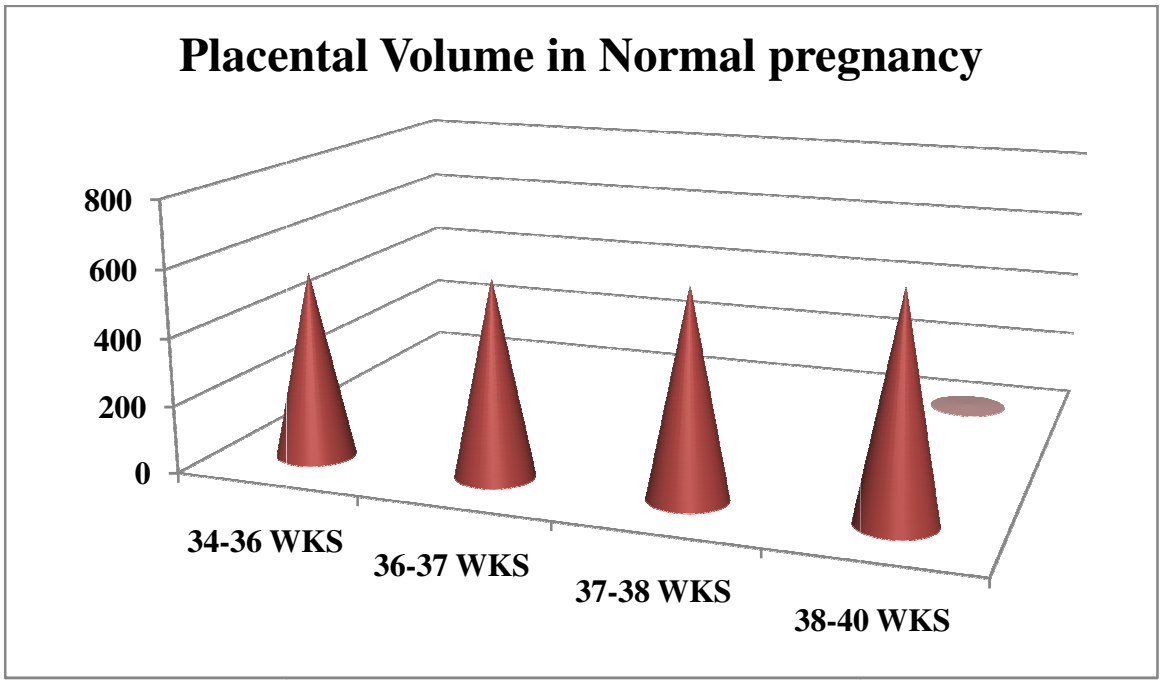
This shows the more earlier the gestational age, severe reduction in the placental volume. Near term there is only mild reduction in the placental volume.



Comparing the grading of the placental volume, most of the patients had grade1 placental volume(n=54) where the placental volume was above 50 th percentile. Severe reduction in placental volume noted only in 11 patients. In 34-36 weeks, most of the patients has grade 2, grade 3 placental volume than grade 1 placental volume. Whereas in 38-40 weeks of gestation, most of the patients had grade 1 placental volume. This indicates in the early onset IUGR, placental insufficiency is more when compared to late onset IUGR.

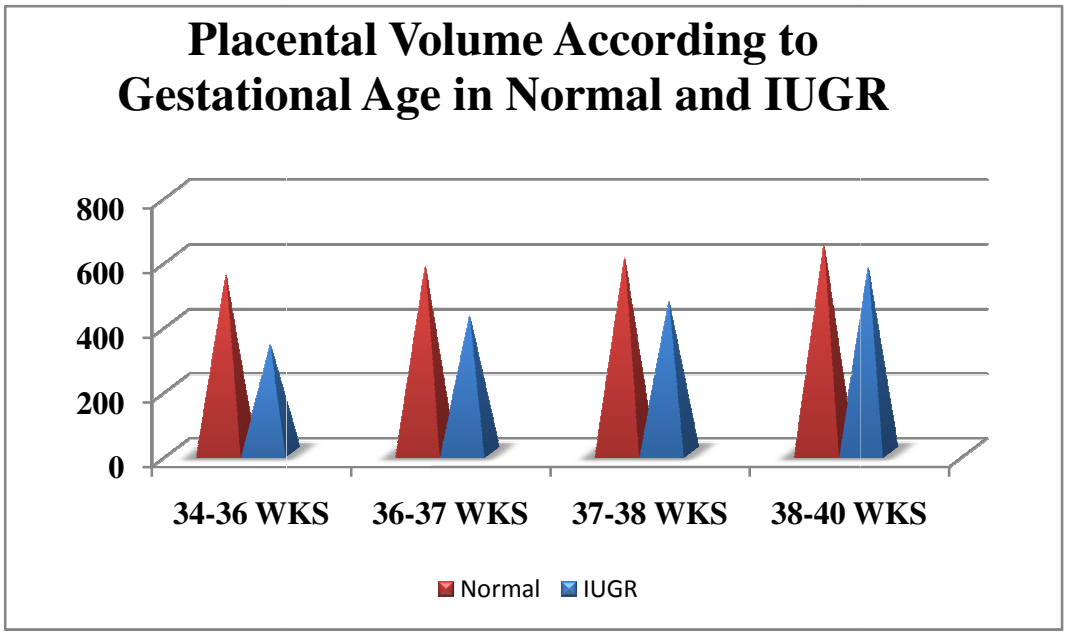
The average placental volume observed in normal pregnancy.

In normal pregnancies for all gestational age the placental volume was around 90 th percentile.



When comparing the average placental volume of normal & IUGR pregnancy , the following was observed.

GA	NORMAL PREGNANCY	IUGR	DIFFERENCE
34-36 WKS	552cm	335cm^3	217cm^3
36-37 WKS	578 cm^3	424cm^3	154cm^3
37-38 WKS	604cm^3	469cm^3	135cm^3
38-40 WKS	647cm^3	574cm^3	100cm^3



This figure shows there is significant reduction in placental volume in IUGR group when compared with normal pregnancy in all gestational age group. The reduction in placental volume is more significant in the early gestational group. As the gestational age advances the difference in placental volume between IUGR and normal pregnancy becomes less significant.

On statistical analysis the following was observed.

STUDY GROUP	MEAN PLACENTAL VOLUME	STANDARD DEVIATION	SIGNIFICANCE
IUGR	402.66	38.679	0.001
NORMAL PREGNANCY	584.4	127.924	0.001

p=0.001** Highly significant.(Levene s T-Test)

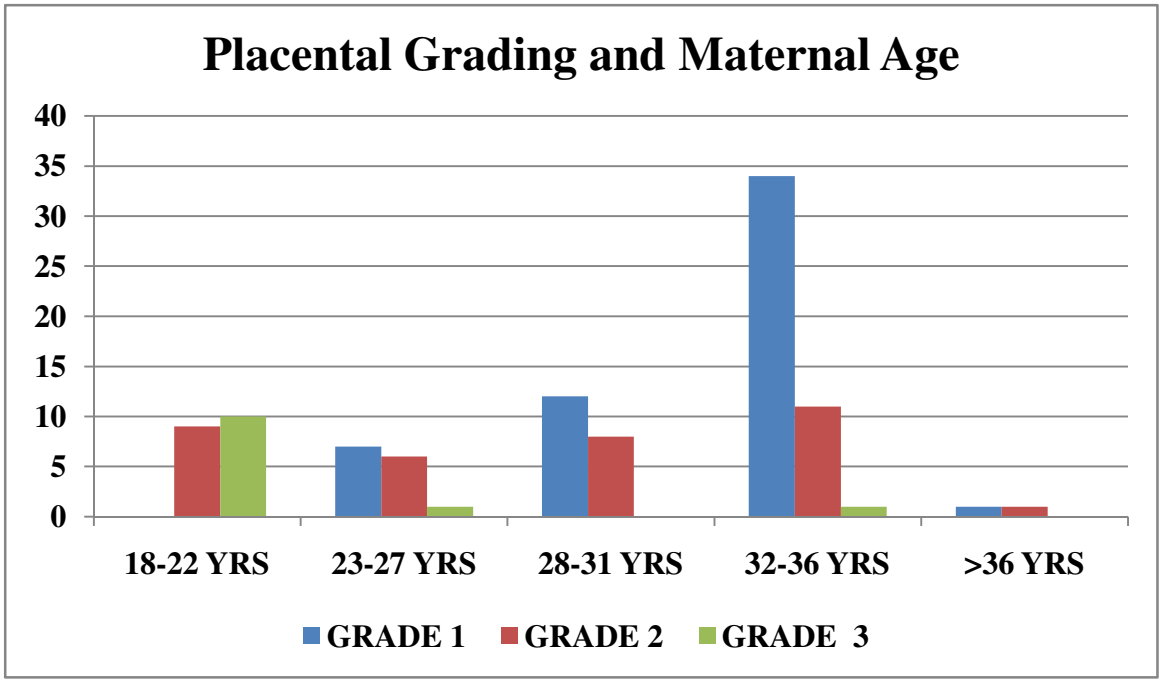
When comparing the average placental volume of all gestational age group in IUGR with that of normal group, there is statistically significant reduction is noted.

With the above findings, we can conclude that in IUGR pregnancies without any identifiable aetiology, the placental insufficiency of unknown cause plays a major role.

2. COMPARISON OF PLACENTAL GRADING WITH MATERNAL AGE:

On comparing the placental grading with maternal age the following was observed.

MATERNAL AGE	GRADE 1	GRADE-2	GRADE 3	TOTAL
18-22 YRS	-	1	10	11
23-27 YRS	7	8	1	15
28-31 YRS	13	12	-	25
32-36 YRS	30	10	1	41
>36 YRS	4	4	-	8



This diagram shows the comparison of placental grading with maternal age. Here, more severe placental volume reduction was noticed in younger age group. With advancing maternal age only mild reduction in placental volume was observed. In the commonest age group presented in this study of 32-36 years, 55.55% of these patients had only grade1 placental volume.In the contrary, 10 patients among 11 in the age group of 18-22 had grade 3 placental volume.

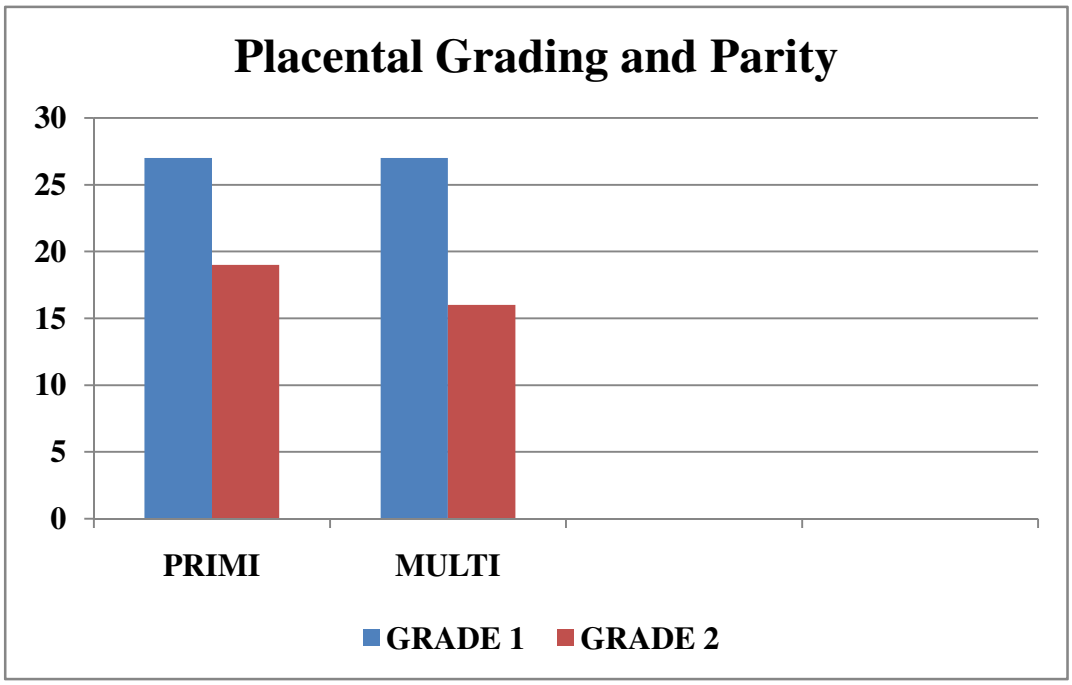
This is comparable with a study conducted by Taj Muhammad,Asmat Ara (2010)[64] who reported younger maternal age is a risk factor for IUGR by comparing with a study by Jamal et al,& Ferraz et al[65].

3. COMPARISON OF PLACENTAL GRADING WITH PARITY:

When comparing the parity with placental volume grading the following findings were noted.

PARITY	GRADE 1	GRADE 2	GRADE 3	TOTAL
PRIMI	20	19	10	49
MULTI	34	16	1	51

In our study even though both primi & multi were presented equally.



This diagram represents the comparison of placental grading with parity. Here primigravidas had severe reduction in placental volume when compared to mutigravidas. This is comparable with the

study by Taj mohammad, Amsat Ara (2010) who reported that, primiparity was also a significant risk factor for IUGR at multivariable level. Similar findings was also reported by Fikree et al & Thompson et al.

4. DOPPLER ANALYSIS:

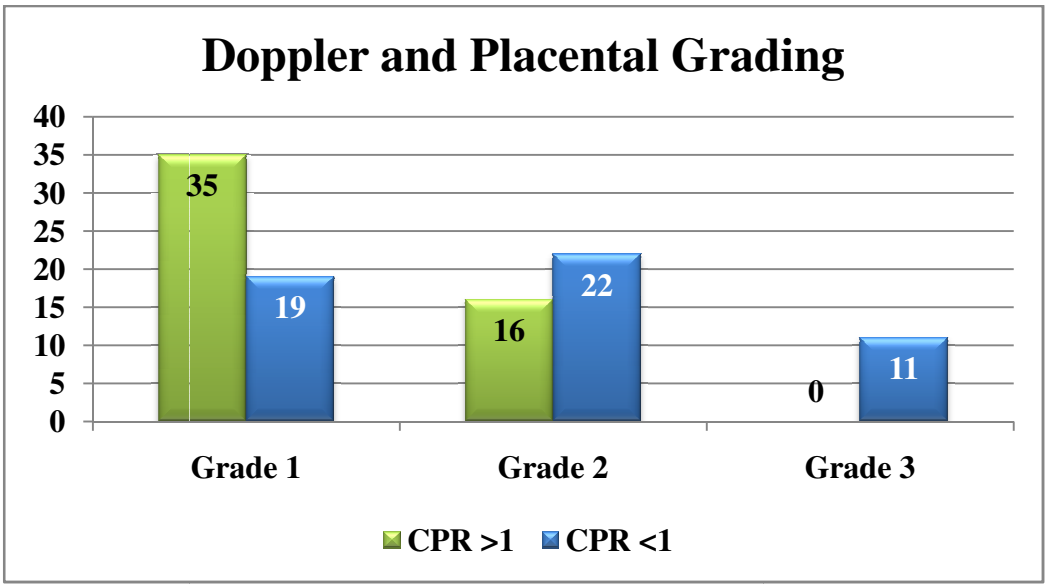
All the patients in IUGR group were subjected to arterial doppler & the Cerebroplacental ratio was calculated. Venous doppler was not done. The reports were analysed based upon the Cerebroplacental ratio.

CPR	GOOD OUTCOME	ADVERSE OUTCOME
<1	20	32
>1	31	17

In patients with CPR<1 the adverse outcome was more when compared with CPR>1.

On analysing the datas with placental volume grading, the following was observed.

PLACENTAL VOLUME	CPR <1	CPR>1
GRADE 1	22	32
GRADE 2	19	16
GRADE 3	11	-



This shows that grade 1 placental volume is associated with less doppler changes. All patients with Severe reduction in placental volume is associated with doppler changes.

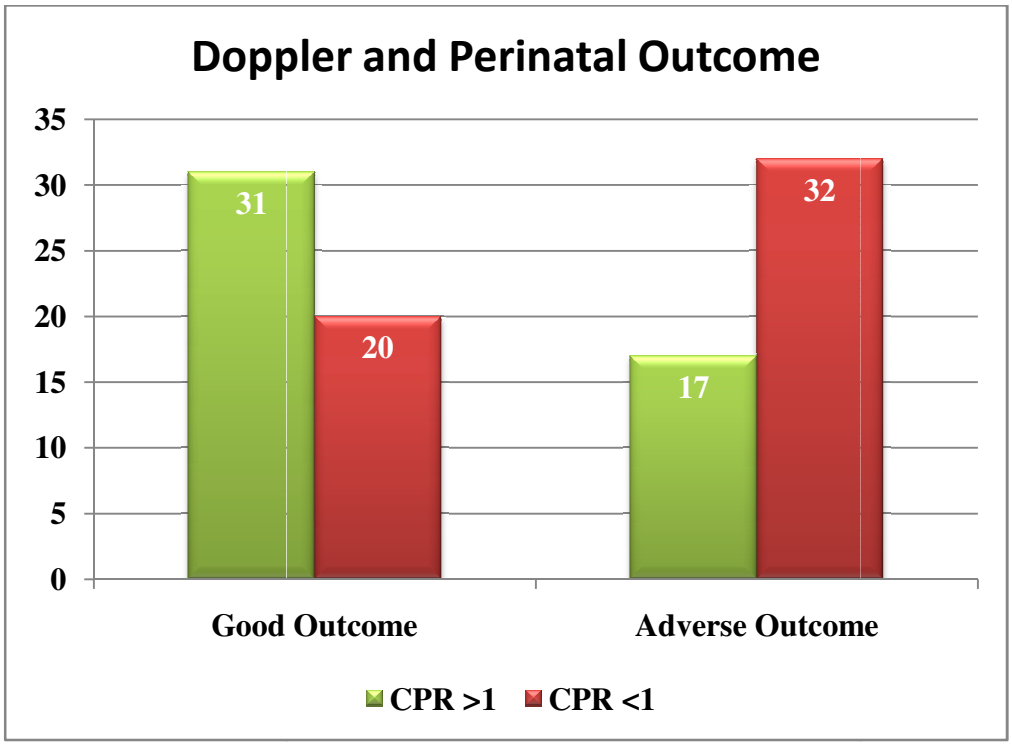
This is comparable with the study done by Dudarenicz L,Kaluzewski B (2006)[66] in which they compared placental volume with doppler study in 82 pregnancies between 14-40 wks of gestation. They concluded that PI of umbilical artery correlated negatively with

	Good Outcome	Adverse Outcome
CPR< 1	20	32
% within CPR	39.2%	60.8%
% within Outcome	38.5%	64.6%
CPR> 1	31	17
% within CPR	65.3%	34.7%
% within Outcome	61.5%	35.4%

Placental volume, PI of MCA showed no significant correlation whereas the Cerebroplacental ratio showed significant positive correlation with placental volume.

On statistical analysis of doppler changes with perinatal outcome the following was noted.

p=0.009** Highly significant. (pearsons chi-square test)



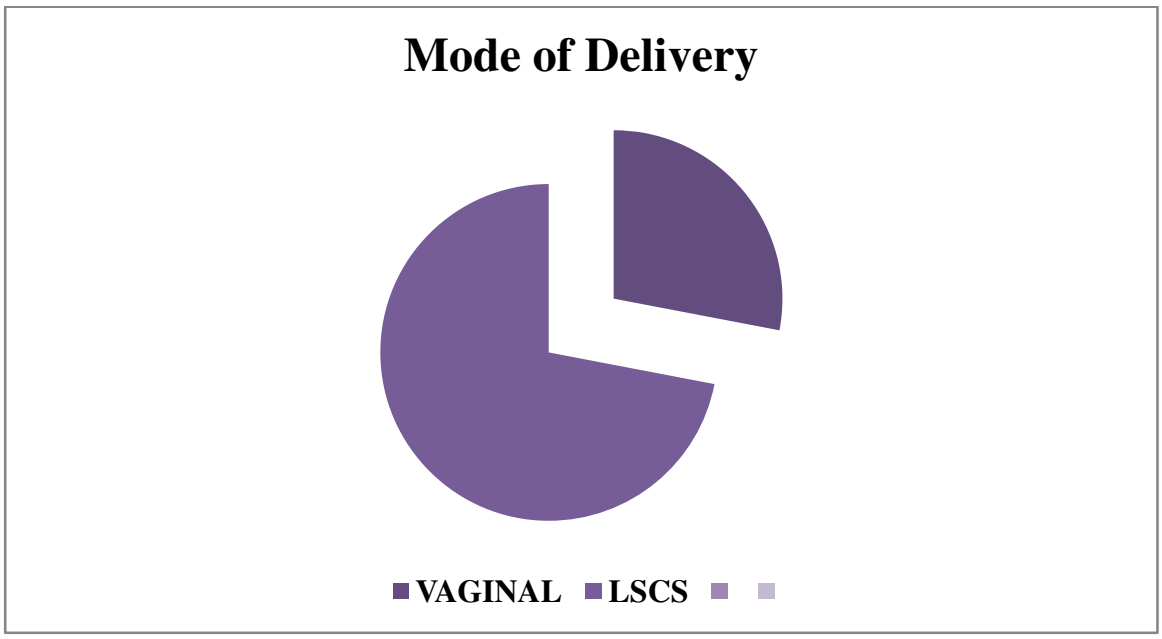
This shows the sensitivity of predicting the perinatal outcome by CPR is 60.8% and the specificity is 65.3%

5. The mode of delivery in patients with IUGR:

All the patients in the study group were observed till delivery. Patients were followed up by antenatal fetal surveillance with daily fetal movement count, Non stress test, Modified Biophysical profile, weekly doppler, serial ultrasound to monitor the interval growth. After deciding for termination of pregnancy, placental volume again measured if it was done 48 hrs before, Bishop scoring, Non stress test, Amniotic fluid index all were repeated. The mode of termination was decided based upon all these parameters. Those who were planned for

vaginal delivery were induced with cerviprime gel & were carefully monitored for signs of fetal distress.

MODE OF DELIVERY	GRADE 1	GRADE 2	GRADE 3	TOTAL
SPONTANEOUS VAGINAL DELIVERY	1	1	-	2
INDUCED VAGINAL DELIVERY	12	6	6	24
CAESAREAN SECTION	41	28	5	74
TOTAL	54	35	11	



Vaginal delivery was very low in all IUGR group irrespective of placental volume. Total no of caesarean section was high when compared to vaginal delivery.

The indications of caesarean section were the following.

INDICATIONS	NO OF DELIVERY	PERCENTAGE
FAILED INDUCTION	32	43.24%
NON REASSURING CTG	23	31.08%
SEVERE OLIGOHYDROMNIOS	8	10.81%
BREECH	11	14.86%

Among these indications, failed induction was more in primi gravida with gestational age between 34-37 wks. This was mainly due to poor Bishop score at the time of induction. Some patients in the group of induction were taken up for LSCS for the signs of intrapartum fetal distress. In the Electronic fetal heart rate monitoring ,the incidence of non reassuring heart rate pattern was observed more with placental volume <10 th percentile. The commonest non reassuring pattern observed was loss of beat to beat variability followed by absence of accelerations. Spontaneous decelerations were

observed in patients with very low placental volume. Severe oligohydromnios was also more in placental volume <50 th percentile.

Some patients were taken up for caesarean section without induction such as breech, oligohydromnios, non reassuring heart rate pattern in NST. Other patients underwent caesarean section were due to failed induction, signs of intrapartum fetal distress. The outcome of babies of these 2 groups was as follows:

Elective LSCS	29
Emergency LSCS	43

The indications for elective LSCS were Breech, oligohydromnios, non reactive CTG. The indications for emergency LSCS were non progression of labour, failed induction, intrapartum fetal distress. The outcome of these 2 groups are as follows.

Outcome	Elective LSCS	Emergency LSCS
MSAF	1 (3.44%)	9 (20.93%)
Low APGAR	6 (20.68%)	13 (30.23%)
Good	22 (75.86%)	21 (48.83%)

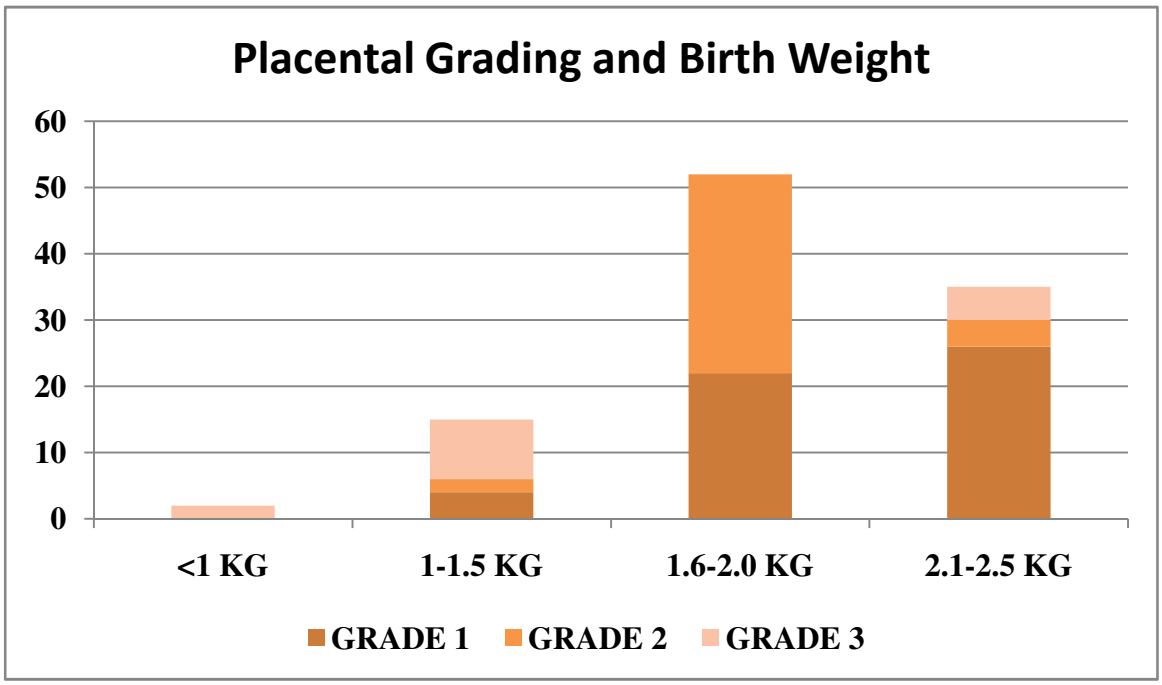
The incidence of MSAF and low APGAR were more with emergency LSCS group. The fetal outcome was better in elective LSCS when compared with emergency LSCS.

Distribution of birth weight in the IUGR group.

The birth weight of the babies were compared with placental volume and analysed.

BIRTH WEIGHT	PLACENTAL VOLUME GRADE 1	PLACENTAL VOLUME GRADE 2	PLACENTAL VOLUME GRADE 3	TOTAL	PERCENTAGE
< 1KG	-	-	2	2	2%
1-1.5 KG	6	2	9	17	17%
1.6-2.0 KG	20	27	-	47	47%
2.1-2.5KG	28	6	-	34	34%

In grade 3 placental volume the birth weight of the babies was significantly lower than that of grade 1 and grade 2 placental volume.



This diagram shows the birth weight distribution according to placental grading. Very low birth weight babies were observed in the group of severe reduction in placental volume. In patients with grade 1& grade 2 placental volume, the birth weight was 1.6-2.5 kgs. This shows a positive correlation between placental volume and birth weight.

Placental volume	Average birth weight	S.D.	Significance
Grade 1	1.99kg	0.30	0.001
Grade 2	1.82kg	0.21	0.001
Grade 3	1.25kg	0.6	0.001

p=0.001** highly significant.

When comparing the average birth weight of grade 1& grade 2 placental volume, there was no significant difference between these two. When comparing that of grade1& grade 2 with grade 3 there was significant reduction in birth weight noted.

This is comparable with a study done by Thame M,Osmond, Wilks (2001)[67] in which they concluded that low birth weight was often preceded by small placental volume in second trimester. placental volume may be a more reliable predictor of size at birth than fetal anthropometric measurements and may be useful in early identification of fetus with perinatal risk.

The perinatal outcome of the babies are as follows.

The perinatal outcome of the babies in IUGR group are analysed and the results are as follows.

PERINATAL OUTCOME	PLACENTAL VOLUME GRADE 1	PLACENTAL VOLUME GRADE 2	PLACENTAL VOLUME GRADE 3	PERCENTAGE
ADVERSE	10	18	11	39 %
GOOD	44	17	-	61%

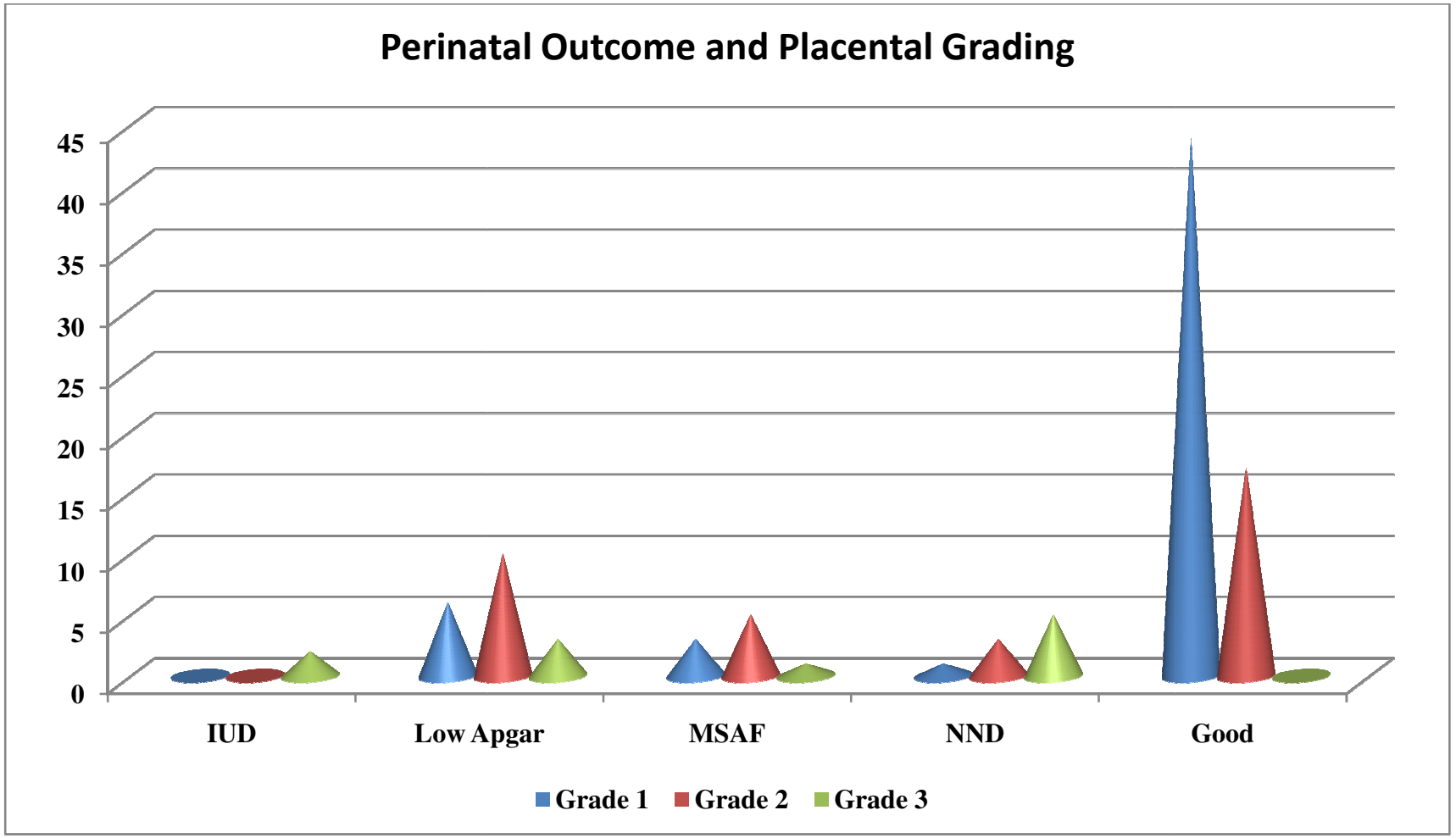
In patients with grade 1 placental volume , the overall outcome of the baby was good.

In patients with grade 2 placental volume, both good and adverse outcome were almost equal.

In grade 3 severe placental volume reduction all babies had adverse outcome only.

On analysing the adverse outcome the following was noted.

PERINATAL OUTCOME	PLACENTAL VOLUME GRADE 1	PLACENTAL VOLUME GRADE 2	PLACENTAL VOLUME GRADE 3	TOTAL
IUD	-	-	2	2
LOW APGAR AT BIRTH	6	10	3	19
FETAL DISTRESS/MSAF	3	5	1	9
EARLY NEONATAL DEATH	1	3	5	9
NO ADVERSE OUTCOME	44	17	-	39


Perinatal Outcome and Placental Grading
45
40
35
30
25
20
15
10
5
0
IUD
Low Apgar
MSAF
NND
Good
Grade 1
Grade 2
Grade 3

In patients with grade 1 placental volume the outcome of fetus was good . In this group 6 babies showed low Apgar at birth(11.11%) & 3 babies showed signs of fetal distress like MSAF (5.55%). Among these babies with perinatal morbidity,2 babies with meconium aspiration and 1 baby with low apgar at birth died in the early neonatal period after admission in the neonatal care unit (33.33% mortality).The other babies recovered well. Another 1baby was died in the early neonatal period due to very low birth weight and sepsis (1.85%) .44 babies had good perinatal outcome without any morbidity and mortality(81.48%). The overall good outcome of all babies in this group including those recovered after perinatal morbidity was 92.59%.

In patients with grade 2 placental volume, the incidence of fetal distress and low Apgar were more. Low Apgar was noticed in 10 babies(28.57%).The incidence of fetal distress with meconium aspiration was noticed in 5 babies (14.28%).Among these babies with above perinatal morbidity,3 babies with severe meconium aspiration syndrome and 2 babies with poor apgar, totally 5 babies died even with good neonatal critical care(33.33%).Other babies recovered well. 17 babies had no adverse outcome(48.57%). Moreover 3 babies were

died in the early neonatal period due to sepsis (8.57%) in this group.The overall good outcome of babies in grade 2 placental volume when considering those babies recovered from initial perinatal morbidity was 62.85% which is lower than that of grade 1 placental volume.

In patients with grade 3 placental volume all babies had adverse outcome only. There was 2 IUD (18.18%) mainly due to severe IUGR and very low birth wt (950 gms& 850 gms). 3 babies were born with low Apgar(27.27%), and 1 baby born with severe fetal distress due to meconium aspiration (9.09%) . All these 4 babies died in the early neonatal period even with good neonatal intensive care.5 babies died in the intensive care unit after admission due to delayed complications like sepsis (45.45%). The adverse outcome of babies in grade 3 placenta was 100%.

On statistical analysis the following was observed.

Placental volume	Good out come	Adverse outcome	Significance
Grade 1	81.48%	18.52%	0.003
Grade 2	48.57%	51.43%	
Grade 3	-	100%	

p=0.003** Highly significant (pearson chi-square test).

The percentage 0f good outcome in grade 1 placental volume was 81.48% and for adverse outcome it was 18.52%

The percentage of good outcome in grade 2 placental volume was 48.57% for adverse outcome it was 51.43% .This showed when the placental volume goes down there was an increase in adverse outcome.

The percentage of adverse outcome in grade 3 placental volume was 100%.So it predicts poor perinatal outcome.

This shows the positive correlation between placental volume and perinatal outcome.

COMPARISON OF PLACENTAL VOLUME BEFORE AND AFTER DELIVERY:

Following the delivery of the placenta, the umbilical cord was tied close to its insertion preventing blood loss from the placenta. The edges were trimmed of the membranes, measurements were taken to calculate the placental volume. on comparing these two the following findings were observed.

The average placental volume measured by ultrasonogram & after delivery in IUGR group was,

GESTATIONAL AGE	PV BY USG($cm^{3)}$	PV AFTER DELIVERY cm^3
34-36 WKS	335	329
36-37 WKS	424	417
37-38 WKS	469	455
38-40 WKS	574	580

There was no significant difference noted between the placental volume measured before delivery by two dimensional ultrasound and that measured after delivery. This denotes that the measurement of placental volume by two dimensional ultrasound in the antenatal period is an effective method.

The average placental volume in normal group before & after delivery was,

GESTATIONAL AGE	PLACENTAL VOLUME BY USG	PLACENTAL VOLUME AFTER DELIVERY
34-36 WKS	552	565
36-37 WKS	578	590
37-38 WKS	604	613
38-40 WKS	647	635

In these group also both measurements were correlated well.

This was comparable with the study by Humberto Azprurua, Edmund F[68] who noticed significant correlation between placental volume measured by 2 dimensional ultrasound & placental volume measured after delivery and they found the mean error between these methods was only 16%.

SUMMARY

This was a prospective analytical study.

100 patients with singleton pregnancy after confirming IUGR were included in this study. 100 patients with singleton uncomplicated pregnancy were selected as control in such a way to match with the variables in IUGR group.

76% 0f patients in IUGR group was in 34-37 weeks of gestational age. The common maternal age group presented was 32-36 years. Both primigravida and multigravida were presented equally.

For all patients general and obstetric examinations were done.

All patients were subjected to ultrasound examination. Fetal biometry including BPD,HC ,AC ,FL, EFW and AFI were measured. Placental localisation was done. placental volume was measured.

Doppler study of umbilical and middle cerebral artery was done for all patients in the group of IUGR. Cerebroplacental ratio was calculated from the resistance index of middle cerebral and umbilical artery for all patients underwent doppler study.

All patients were followed up till delivery. The placental volume measurement was repeated if it was done 48 hrs before delivery. The mode of delivery and the indication for LSCS were noted.

Birthweight of the baby was noted. APGAR at 1 & 5 minutes were observed. All perinatal morbidities like meconium aspiration, low APGAR were noted. All babies were followed up till discharge.

After delivery again the placental volume was measured.

The placental volume measured by ultrasound was compared with that measured after delivery.

The results were compared with that of normal pregnancy.

The average placental volume in normal pregnancy was 595.25 cm^3

The average placental volume in IUGR pregnancy was 450 cm^3

This shows a significant difference in placental volume between these group. On statistical analysis, this showed significant difference.p=0.001** (highly significant; Levenes T-Test).

The placental volume done by ultrasound before delivery was compared that of measured after delivery.

The average placental volume measured after delivery in normal pregnancy was 600.75 cm^3. The average placental volume after delivery in IUGR group was 445.25 cm^3. These value did not show much difference that of ultrasound measurement before delivery.

The incidence of LSCS was high in the group of IUGR(74%).

In case of emergency LSCS the perinatal morbidity in the form of MSAF was more when compared with the elective LSCS group(20.3% Vs 3.4%).The overall good outcome in elective LSCS group was 75.86% and in emergency LSCS group it was 62.79%. This significant difference indicates intra partum fetal asphyxia that occurred with induction of labour which is a well expected complication in IUGR.

The average birthweight of the babies in grade 1 placental volume was 1.99 kgs and in grade 2 placental volume it was 1.82 kgs. These 2 did not show much difference. The average birth weight in grade 3 placental volume was 1.25 kgs. This showed significant difference in average birth weight.

When the placental volume was compared with the perinatal outcome of the baby, in grade 1 placental volume, there was 81.48% good outcome and 18.52% adverse out come & in grade 2 placental volume, the good outcome had come down to 48.57% and the adverse outcome increased to 51.43% whereas in grade 3 placental volume there was 100% adverse outcome only. This showed that the placental volume had good correlation with the fetal outcome.

This study showed positive correlation between the severity of IUGR and placental volume. It also predicted the adverse perinatal outcome of the fetus clearly. Hence this can be taken as one of the methods of predicting adverse neonatal outcome in IUGR.

CONCLUSION

Healthy baby and healthy mother are the goal of obstetrical management.

The diagnosis of Uteroplacental insufficiency , the major cause of IUGR,identifies the group of fetuses who are at incresed risk for perinatal complications.

Ultrasonography plays a major role in early diagnosis of IUGR.

Doppler ultrasonogram helps in identifying fetuses already in hypoxia and acidemia so that early interventions could be done to reduce perinatal complications. But it needs costly equipment and trained personale which limits its usefulness in developing country like India.

Placental volume has positive correlation with birthweight of the baby and perinatal complications.

Estimation of placental volume by simple 2 dimensional ultrasound could be a better alternative method of antenatal fetal surveillance in IUGR where doppler ultrasound is not available.

PROFORMA

Name : Age: Ip no:

Address: Date of admission:

Socio economic status: Education:

Obstetric code: LMP: EDD:

Menstrual history: Regular/Irregular:

Sure of LMP: Yes/No

Marital history: Md since:

Consanguinity:

Obstetric history:

Past history:

H/o HT/DM/TB/BA/ HEART DISEASE/EPILEPSY/CHRONIC RENAL DISEASE/CONNECTIVE TISSUE DISORDER.

Family history:

Personal history:

GENERAL EXAMINATION:

HT: **WT:** **BMI:**

Built: **Thin / Average / obese**

Pallor / Jaundice / clubbing / cyanosis /Pedal edema / Lymphadenopathy

VITALS: **Temp:** **Pulse rate:** **BP:** **RR:**

Breast: **Spine:** **Thyroid:**

CVS: **RS:**

Examination of the Abdomen :

Fundal ht:

FH:

Liquor adequacy:

INVESTIGATIONS:

URINE- **Alb:** **Sugar:** **Dep:**

BLOOD- **Hb:** **PCV:** **Platelets:**

BLOOD: Urea: Sugar: Creatinine:

Blood G&T: HIV: VDRL: HBSAG:

ULTRASONOGRAM:

	1TRIMESTER	2TRIMESTER	3TRIMESTER
BPD			
AC			
FL			
EFW			
GA			
PLACENTA			
AFI			

DOPPLER STUDY:

UMBILICAL ARTERY RI:

MIDDLECEREBRAL ARTERY RI:

CPR:

PLACENTAL VOLUME:

DELIVERY:

VAGINAL: SPONTANEOUS: INDUCED:

LSCS : ELECTIVE / EMERGENCY

OUTCOME:

IUD /Still born:

Birth wt: Apgar:

Liquor: clear /meconium

Placental volume:

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INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. G. Pakkialakshmi
PG in MD Obstetrics & Gynaecology
Madras Medical College, Chennai -3

Dear Dr. G. Pakkialakshmi

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " Comparative study of placental volume in normal pregnancy and intra uterine growth restriction" No.01062012.

The following members of Ethics Committee were present in the meeting held on 27.06.2012 conducted at Madras Medical College, Chennai -3.

1.	Dr. S.K. Rajan. M.D.,FRCP.,DSc	-- Chairperson
2.	Prof. Pregna B. Dolia MD Director , Inst. of Biochemistry, MMC, Ch-3	-- Member Secretary
3.	Prof. K.M. Sudha MD Prof of Pharmacology ,MMC, Ch-3	-- Member
4.	Prof. C. Rajendiran, MD Director , Inst. of Internal Medicine, MMC, Ch-3	-- Member
5.	Prof. Karkuzhali MD Director i/c, Prof of Pathology, MMC, Ch-3	-- Member
6.	Thiru. Govindasamy BA BL	-- Lawyer

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

CONSENT FORM

STUDY TITLE : COMPARATIVE STUDY OF PLACENTAL VOLUME IN NORMAL PREGNANCY AND INTRA UTERINE GROWTH RESTRICTION

STUDY CENTRE: Institute of Obstetrics and Gynaecology,Egmore,Chennai

Participant Name : Age: Sex: I.D.No.:

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

I understand that my participation in the study is voluntary and that Iam free to withdraw at any time without giving any reason.

I understand that the investigator, regularity 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 understsnd 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 results that arise from the study.

I hereby consent to participate in this study titled "COMPARATIVE STUDY OF PLACENTAL VOLUME IN NORMAL PREGNANCY AND INTRA UTERINE GROWTH RESTRICTION"

Signature of Investigator: Place:

Study Investigators Name: Date :

Signature/thumb impression of patient

Thanking you,

Yours faithfully,

MASTER CHART

S.No.	Name	Age	Obst Code	G/A (LMP)	G/A (USG)	MCA (RI)	UA (RI)	CPR	PV(USG)	PV(del)	Delivery	IND	BW	Outcome
1	Sai subha	20	Primi	34-35	26-27	0.6	0.7	<1	185 (III)	175	Lscs	Oligo	1.2	NND
2	Meera	22	G2A1	34-35	28-29	0.65	0.74	<1	195 (III)	190	LSCS	Fail ind	1.3	Low apg
3	Ansari	21	G2P1L1	35-36	28-29	0.64	0.76	<1	176 (III)	170	Vaginal		900gms	IUD
4	Ramani	20	Primi	33-34	29-30	0.7	0.8	<1	190 (III)	196	Lscs	Oligo	1.4	Low apg
5	Lakshmi	19	Primi	34-35	24-25	0.7	0.96	<1	160 (III)	150	Vaginal		850gms	IUD
6	Subha	19	Primi	34-35	26-27	0.6	0.75	<1	185 (III)	195	Lscs	Fail ind	1.4	Low apg
7.	Nabeesa	23	Primi	36-37	32-31	0.8	0.7	>1	325 (II)	315	Vaginal		1.7	Good
8.	Vimala	24	G2A1	34-35	32-31	0.76	0.64	>1	345 (II)	330	Vaginal		1.8	Good
9.	Punitha	24	G2P1L1	34-35	29-30	0.7	0.8	<1	330 (II)	325	LSCS	Oligo	1.9	Good
10.	Anitha	27	G3P1L1A1	34-35	32-31	0.7	0.68	>1	315 (II)	303	LSCS	Breech	1.6	Good
11.	Rajeswari	25	Primi	35-36	32-31	0.8	0.9	<1	324 (II)	335	LSCS	Oligo	1.8	Low Apg
12.	Vanitha	26	G2P1L0	34-35	29-30	0.7	0.82	<1	340 (II)	332	LSCS	Fail Ind	1.7	Low Apg
13.	Valli	25	G2A1	35-36	31-32	0.7	0.82	<1	345 (II)	360	LSCS	Fail Ind	1.8	Good
14.	Ranjani	25	G3P1L1A1	36-37	32-33	0.9	0.8	>1	350 (II)	355	LSCS	Breech	1.9	Good
15.	Dhanam	27	G3P1L1A1	35-36	31-32	0.86	0.74	>1	368 (I)	375	Vaginal		2.0	Good
16.	Divya	28	Primi	33-34	32-31	0.8	0.74	>1	346 (II)	340	LSCS	Faild Ind	1.8	Low Apg
17.	Kalaivani	29	G3P2L2	34-35	32-33	0.8	0.72	>1	416 (I)	400	LSCS	Fail Ind	1.9	Low Apg.
18.	Lakshmi	29	G4P1L1A2	35-36	32-31	0.9	0.82	>1	328 (II)	325	LSCS	Oligo	1.9	Good
19.	Kavitha	30	Primi	35-36	32-33	0.7	0.8	<1	315 (II)	324	LSCS	Breech	1.7	Low Apg.
20.	Sharmila	31	G2P1L1	35-36	31-32	0.7	0.68	>1	325 (II)	315	LSCS	Faild Ind	2.0	Good
21.	Vasuki	31	G3P2L1	34-35	32-31	0.8	0.9	<1	300 (II)	315	LSCS	Oligo	1.9	Low Apg.
22.	Girija	28	Primi	34-35	32-31	0.7	0.68	>1	275 (II)	280	LSCS	Faild Ind	1.8	Low Apg.
23.	Prema	29	Primi	35-36	32-33	0.8	0.7	>1	486 (I)	495	LSCS	Breech	2.2	Good
24.	Shoba	31	G5P1L1A2	35-36	31-32	0.8	0.7	>1	315 (II)	305	LSCS	Faild Ind	1.9	Good

25.	Selvi	30	Primi	35-36	32-31	0.8	0.74	>1	430 (I)	415	LSCS	Faild Ind	2.1	Good
26.	Parvathi	29	G2P1L1	35-36	31-32	0.8	0.84	<1	268 (II)	250	LSCS	Faild Ind	2	Good
27	Sangeetha	28	Primi	34-35	32-31	0.8	0.76	>1	475 (I)	482	LSCS	Oligo	1.9	Low Apg.
28.	Pushpa	29	Primi	34-35	29-30	0.7	0.84	<1	328 (II)	345	LSCS	Faild Ind	1.9	Good
29.	Kiruba	32	Primi	33-34	29-30	0.7	0.68	<1	386 (I)	370	Vaginal		1.4	NND
30.	Sumathi	34	G3P2L0	35-36	31-32	0.82	0.74	>1	480 (I)	500	LSCS	Breech	2.1	Good
31.	Shanthi	34	G2P1L0	34-35	32-31	0.84	0.76	>1	496 (I)	525	LSCS	Faild Ind	2.0	Good
32.	Sajeetha	33	G4P1L1A2	36-37	32-33	0.8	0.76	>1	502 (I)	535	LSCS	Faild Ind	2.3	Good
33.	Sagunthala	36	Primi	33-34	32-31	0.76	0.84	<1	325 (II)	330	Vaginal		1.7	Low Apg.
34.	Asha	35	G2A1	33-34	31-32	0.8	0.74	>1	326 (II)	330	LSCS	Faild Ind	1.8	Low Apg.
35.	Ramya	35	Primi	34-35	31-32	0.7	0.96	<1	388 (I)	400	LSCS	Faild Ind	1.5	NND
36.	Nithya	32	G3P1L1A1	33-34	32-31	0.8	0.76	>1	315 (II)	320	LSCS	Faild Ind	1.6	Low Apg.
37.	Jaya	33	G2A1	35-36	32-33	0.8	0.72	>1	515 (I)	545	Vaginal		2.2	Good
38.	Amutha	33	G2P1L1	34-35	31-32	0.9	1.1	<1	270 (II)	295	Vaginal		2.2	Good
39.	Malathi	35	G3P2L2	34-35	32-31	0.8	0.76	>1	383 (I)	400	LSCS	Oligo	2.0	Good
40.	Deepa	34	G3P1L1A1	34-35	32-31	0.9	0.86	>1	254 (II)	275	LSCS	Faild Ind.	1.7	Low Apg.
41.	Latha	32	Primi	35-36	32-33	0.74	0.86	<1	325 (II)	330	Vaginal		2.2	Good
42.	Selvi	33	G2A1	34-35	31-32	0.7	0.84	<1	315 (II)	310	Vaginal		2.1	Good
43.	Bala	36	G2P1L1	34-35	32-31	0.7	0.72	<1	325 (II)	335	LSCS	CTG NR	1.8	Good
44.	Malini	35	G3P1L1A1	34-35	32-31	0.8	0.76	>1	366 (I)	350	LSCS	Faild Ind	1.9	Good
45.	Sathya	33	G3A2	34-35	31-32	0.84	0.76	<1	386 (I)	395	LSCS	Faild Ind	2.0	Good
46.	Valli	34	G2P1L1	34-35	30-31	0.76	0.86	<1	278 (II)	250	LSCS	Faild Ind	1.6	NND
47.	Bhavani	37	Primi	35-36	31-32	0.7	0.86	<1	416 (I)	440	LSCS	Breech	1.9	Good
48.	Stella	32	G2P1L1	36-37	33-34	0.76	0.68	>1	505 (I)	525	LSCS	Breech	2.1	Good

49.	Jaya	37	G3P2L0	34-35	29-30	0.68	0.84	<1	290 (II)	275	LSCS	CTG NR	1.6	Low Apg.
50.	Lalitha	32	G2P1L1	34-35	32-33	0.82	0.76	>1	330 (II)	345	LSCS	CTG NR	1.8	Low Apg.
51.	Praba	35	G2P1L1	35-36	32-33	0.86	0.73	>1	414 (I)	420	Vaginal		2.1	Good
52.	Saraswathi	36	Primi	36-37	33-34	0.8	0.74	>1	496 (I)	515	Vaginal		2.0	Good
53.	Neela	21	Primi	36-37	33-34	0.74	0.86	<1	188 (III)	200	Vaginal		1.2	NND
54.	Devi	21	Primi	36-37	30-31	0.8	0.96	<1	190 (III)	210	LSCS	CTG NR	1.3	MSAF.
55.	Suganya	20	Primi	36-37	33-34	0.8	0.76	>1	366 (II)	375	LSCS	Faild Ind	2.1	Good
56.	Rekha	24	G2A1	36-37	32-33	0.72	0.86	<1	588 (I)	595	LSCS	CTG NR	2.2	Good
57.	Lally	27	G2PlL1	36-37	33-34	0.86	0.7	>1	590 (I)	605	Vaginal		2.4	Good
58.	Sivakami	27	G3P1L1A1	36-37	34-35	0.96	0.72	>1	582 (I)	575	Vaginal		2.3	Good
59.	Radhi	28	G3A2	37-38	33-34	0.92	0.76	>1	586 (I)	575	LSCS	Breech	2.3	Good
60.	Zeenath	30	Primi	36-37	32-33	0.76	0.92	<1	550 (I)	565	LSCS	CTG NR	2.1	Good
61.	Selvi	30	G2P1L1	36-37	33-34	0.8	0.76	>1	344 (II)	360	LSCS	Faild Ind	2.0	Good
62.	Thilaka	31	G4P1L1A2	37-38	33-34	0.78	0.96	<1	572 (I)	580	LSCS	Faild Ind	2.2	MSAF
63.	Chitra	29	Primi	36-37	32-33	0.9	0.8	<1	580 (I)	595	Vaginal		2.3	Good
64.	Thangam	28	G2A1	37-38	34-35	0.96	0.72	<1	586 (I)	600	Vaginal		2.2	Good
65.	Maheswari	29	G3P1L1A1	37-38	32-33	0.86	0.73	>1	550 (I)	575	Vaginal		2.4	MSAF
66.	Vijayalakshmi	32	G2P1L1	36-37	32-33	0.8	0.76	<1	566 (I)	550	LSCS	Breech	2.4	Good
67.	Kumari	36	Primi	36-37	31-32	0.76	0.84	<1	320 (II)	335	LSCS	Faild Ind	2.1	MSAF
68.	Jayanthi	35	Primi	36-37	32-33	0.8	0.72	<1	335 (II)	340	LSCS	Faild Ind	2.1	Good
69.	Sathya	33	G2P1L1	36-37	31-32	0.9	0.72	>1	525 (I)	520	LSCS	CTG NR	2.1	MSAF
70.	Kala	32	G3A2	37-38	32-33	0.74	0.96	<1	540 (I)	550	LSCS	Breech	2.1	Good
71	Manjula	32	Primi	37-38	31-32	0.74	0.82	<1	475 (I)	480	LSCS	Faild ind	1.8	Low apg
72	Rani	32	G3P2L2	37-38	30-31	0.88	0.78	>1	440 (I)	450	Vaginal		1.6	Low apg

73	Sandhya	36	G2P1L1	36-37	31-32	0.76`	0.92	<1	446 (I)	450	LSCS	Failed ind	1.6	MSAF
74	Poongodi	35	G2P1L0	36-37	30-31	0.92	0.86	>1	395 (II)	402	LSCS	CTG NR	1.2	NND
75	Vanaja	33	Primi	37-38	30-31	0.78	0.88	<1	415 (I)	410	LSCS	CTG NR	1.4	MSAF
76	Sarala	32	G2P1L1	37-38	30-31	0.74	0.88	<1	402 (I)	415	LSCS	CTG NR	1.75	LOW APG
77	Indra	18	Primi	37-38	30-31	0.8	0.92	<1	202 (III)	195	Vaginal		1.3	NND
78	Uma	20	Primi	37-38	31-32	0.96	1.2	<1	198 (III)	210	Vaginal		1.25	NND
79	Devagi	25	Primi	37-38	32-33	0.82	0.76	>1	420 (I)	430	LSCS	CTG NR	1.5	MSAF
80	Rajathi	24	Primi	36-37	33-34	0.84	0.76	>1	430 (I)	415	Lscs	CTG NR	1.7	MSAF
81	Geetha	27	G2P1L1	37-38	34-35	0.82	0.78	>1	465 (I)	475	LSCS	CTG NR	1.7	GOOD
82	Kanmani	28	G2A1	37-38	33-34	0.68	0.86	<1	342 (II)	335	Vaginal		1.5	MSAF
83	Gandhimathi	29	G3P1L1A1	38-39	31-32	0.72	0.84	<1	410 (I)	420	LSCS	CTG NR	1.4	Low apg
84	Suganthi	31	G2P1L1	37-38	34-35	0.82	0.76	>1	550 (I)	560	Lscs	Failed ind	2.2	Good
85	Hema	31	G2P1L0	36-37	31-32	0.72	0.84	<1	340 (II)	350	Lscs	CTG NR	1.6	MSAF
86	Amudha	30	Primi	37-38	33-34	0.82	0.76	>1	490 (I)	500	Lscs	CTG NR	1.8	GOOD
87	Mahalakshmi	32	G3P1L1A1	37-38	34-35	0.78	0.86	<1	486 (I)	475	LSCS	CTG NR	1.9	GOOD
88	Ambika	36	G2P1L1	37-38	34-35	0.86	0.75	>1	501 (I)	496	LSCS	CTG NR	1.9	LOW APG
89	Akila	35	G2P1L1	37-38	32-33	0.84	0.78	>1	475 (I)	485	LSCS	FAILED IND	1.7	LOW APG
90	Jesinda	34	G4P1L1A2	37-38	32-33	0.74	0.88	<1	480 (I)	470	LSCS	FAILED IND	1.8	GOOD
91	Meera	33	Primi	37-38	34-35	0.68	0.82	<1	492 (I)	480	Lscs	Failed ind	1.9	Good
92	Noorjahan	32	G4P2L2A1	38-39	34-35	0.74	0.68	>1	530 (I)	525	LSCS	CTG NR	2.2	GOOD

93	Kala	33	G2P1L1	37-38	35-36	0.86	0.76	>1	565 (I)	579	LSCS	Breech	2.4	Good
94	Mary	34	G2P1L0	37-38	33-34	0.86	0.72	>1	440 (I)	450	LSCS	CTG NR	1.4	Low apg
95	Mangai	32	Primi	38-39	30-31	0.64	0.88	<1	202 (III)	220	Vaginal		1.3	NND
96	Bagyalakshmi	35	Primi	38-39	34-35	0.86	0.74	>1	630 (I)	640	LSCS	CTG NR	2.1	Good
97	Pattu	34	G3P2L2	39-40	35-36	0.76	0.84	<!	646 (I)	630	LSCS	CTG NR	2.3	MSAF
98	Sudha	35	G3P1L0A1	39-40	35-36	0.74	0.88	<1	675(I)	675	Vaginal		2.4	MSAF
99	Priya	33	G2A1	38-39	36-37	0.84	0.72	>1	680(I)	690	Vaginal		2.5	Good
100	Mariammal	35	Primi	39-40	35-36	0.82	0.76	>1	660(I)	675	Lscs	CTG NR	2.4 GOOD	

S no	NAME	AGE	OBST CODE	GA(LMP)	GA(USG)	PV(USG)	PV(DELI)	DELIVERY	BW	OUTCOME
1.	Jeyanthi	18	Primi	34-35	35-36	536	546	Vaginal	2.3	LOW APGAR
2.	Nanthini	19	Primi	34-35	34-35	586	595	Vaginal	2.6	Good
3.	Meenakshi	21	Primi	35-36	34-35	590	580	LSCS	2.6	Good
4.	Anitha	22	Primi	35-36	36-37	575	586	LSCS	2.5	RESP DISTRESS
5.	Selvi	19	G2A1	34-35	35-36	568	550	Vaginal	2.4	Good
6.	Chellammal	20	Primi	34-35	34-35	545	530	Vaginal	2.5	Good
7.	Dhanalakshmi	23	G2P1L1	34-35	35-36	560	575	LSCS	2.6	Good
8.	Sudha	27	G2P1L1	35-36	36-37	572	585	Vaginal	2.7	Good
9.	Mallika	23	G3P1L1A1	36-37	35-36	570	585	lscs	2.9	Good
10.	Shanthi	25	Primi	35-36	36-37	555	570	Vaginal	2.6	Good
11.	Thilaka	24	G2A1	34-35	34-35	545	560	Vaginal	2.5	RESP DISTRESS
12.	Mala	25	G2P1L1	36-37	36-37	588	595	Vaginal	2.7	Good
13.	Saritha	26	Primi	35-36	36-37	590	585	LSCS	2.6	Good
14.	Jothi	24	Primi	34-35	35-36	540	555	Vaginal	2.7	Good
15.	Punitha	23	G2A1	34-35	34-35	552	565	lscs	2.8	Good
16.	Nagalakshmi	23	Primi	35-36	36-37	564	585	Vaginal	2.7	Good
17.	Mariammal	28	G3P2L0	34-35	35-36	515	525	lscs	2.4	LOW APGAR
18.	Umarani	30	Primi	35-36	34-35	546	555	Vaginal	2.7	Good
19.	Malathi	31	G3P2L2	36-37	35-36	546	585	Vaginal	2.9	Good

20.	Ambika	31	Primi	35-36	36-37	552	575	LSCS	2.8	Good
21.	Gomathi	29	G3P1L1A1	34-35	34-35	536	545	lscs	2.3	RESP DISTRESS
22.	Vennilla	28	G2P1L1	35-36	34-35	542	565	vaginal	2.4	Good
23.	Lakshmi	29	G2P1L1	35-36	36-37	570	580	LSCS	2.6	Good
24.	Shanthini	30	Primi	36-37	35-36	575	590	Vaginal	2.5	MSAF
25.	Jaya	31	G3P1L1A1	35-36	34-35	580	595	Vaginal	2.7	Good
26.	Raji	30	G3P2L0	34-35	33-34	530	555	Vaginal	2.6	Good
27.	Arifa	29	G2A1	35-36	35-36	545	560	LSCS	2.5	Good
28.	Vimala	32	Primi	35-36	36-37	555	565	Vaginal	2.4	Good
29.	Thara	36	G3P2L2	34-35	33-34	525	540	LSCS	2.3	Good
30.	Eswari	32	Primi	35-36	36-37	568	580	LSCS	2.7	Good
31.	Ponni	33	G4P1L1A2	36-37	35-36	580	575	LSCS	2.8	Good
32.	Bhavani	32	G2A1	34-35	34-35	536	545	Vaginal	2.5	Good
33.	Lakshmi	33	G2A1	33-34	34-35	538	545	LSCS	2.6	Good
34.	Malar	34	G3P2L1	35-36	36-37	542	555	Vaginal	2.9	Good
35.	Hema	32	Primi	34-35	35-36	520	530	Vaginal	2.2	RESP DISTRESS
36.	Nalini	33	G3P1L1A1	35-36	35-36	550	560	LSCS	2.6	Good
37.	Sangeetha	34	G2P1L1	36-37	36-37	568	580	LSCS	2.9	MSAF
38.	Saranya	36	G4P2L2A1	35-36	35-36	578	590	Vaginal	2.8	Good
39.	Santha	34	G2P1L0	34-35	35-36	525	540	LSCS	2.4	Good
40.	Vasantha	33	Primi	36-37	35-36	584	575	LSCS	2.5	Good
41.	Saheeba	32	G2P1L1	35-36	34-35	575	585	vaginal	2.6	Good
42.	Menaka	32	Primi	34-35	34-35	538	545	Vaginal	2.5	Good
43.	Kanchana	37	G5P1L0A3	36-37	36-37	574	590	LSCS	2.9	Good
44.	Sri Devi	38	G3A2	35-36	34-35	568	575	LSCS	2.7	Good
45.	Kamatchi	36	G2P1L1	34-35	34-35	555	560	Vaginal	2.7	Good
46.	Usha	32	G2A1	36-37	35-36	585	600	Vaginal	2.9	MSAF
47.	Josephin	33	G2P1L1	35-36	36-37	590	610	Vaginal	3.0	Good
48.	Deepa	33	Primi	34-35	35-36	530	545	LSCS	2.6	MSAF
49.	Manjula	33	G2P1L0	35-36	34-35	525	540	Vaginal	2.4	Good
50.	Priya	34	Primi	33-34	34-35	548	565	Vaginal	2.6	Good
51.	Lakshmi	18	Primi	36-37	36-37	605	610	LSCS	3.1	MSAF
52.	Kajalakshmi	19	Primi	36-37	37-38	595	580	LSCS	2.9	Good
53.	Vasantha	21	Primi	36-37	36-37	585	575	Vaginal	2.8	Good

54.	Shanthini	21	Primi	37-38	36-37	595	580	LSCS	2.9	Good
55.	Sharmila	23	G2A1	37-38	36-37	602	615	Vaginal	3.2	Good
56.	Lalitha	27	G2P1L1	37-38	37-37	610	620	Vaginal	3.1	Good
57.	Jaya	26	Primi	36-37	35-36	592	610	LSCS	3.0	Good
58.	Vanaja	25	G2P1L1	37-38	36-37	598	585	Vaginal	2.9	Good
59.	Patchiammal	28	G2P1L1	36-37	37-38	575	602	Vaginal	3.3	Good
60.	Sabana	28	G3P1L1A1	36-37	37-38	610	625	Vaginal	3.5	MSAF
61.	Rajalakshmi	31	Primi	36-37	35-36	600	590	Vaginal	2.9	MSAF
62.	Poornima	30	G2P1L1	36-37	35-36	582	590	Vaginal	2.8	Good
63.	Geetha	29	G3P1L1A1	35-36	36-37	594	594	LSCS	3.0	Good
64.	Ambarasi	32	G3A2	36-37	35-36	588	555	Vaginal	2.8	Good
65.	Padma	33	G3P1L0A1	36-37	37-38	576	592	Vaginal	3.2	Good
66.	Suguna	34	G3P2L2	37-38	36-37	594	602	Vaginal	3.0	Good
67.	Sarojini	33	Primi	37-38	38-39	598	615	LSCS	3.1	Good
68.	Ponni	32	Primi	36-37	37-38	570	590	Vaginal	3.0	MSAF
69.	Sarulatha	32	G2P1L1	36-37	35-36	575	590	Vaginal	2.9	Good
70.	Parimala	32	Primi	37-38	38-39	590	610	LSCS	3.2	MSAF
71.	Kumari	36	G3P1L1A1	36-37	37-38	575	600	vaginal	2.9	Good
72.	Sheela	34	G3P1L0	37-38	36-37	615	630	LSCS	3.4	Good
73.	Rani	35	G3A2	36-37	37-38	587	602	LSCS	3.1	Good
74.	Prabavathy	33	G3P2L1	37-38	36-37	588	595	Vaginal	3.2	Good
75.	Devi	18	Primi	37-38	38-39	625	633	Vaginal	3.1	Good
76.	Lakshmi	20	Primi	38-39	37-38	630	645	LSCS	3.2	Good
77.	Malarvizhi	20	G2A1	38-39	39-40	635	650	Vaginal	3.3	LOW APGAR
78.	Nagamani	23	Primi	37-38	38-39	604	615	Vaginal	3.1	Good
79.	Ramani	26	Primi	38-39	37-38	612	630	Vaginal	3.2	Good
80.	Jothi	27	G3A2	37-38	38-39	598	615	Vaginal	3.1	Good
81.	Pushpa	23	G2P1L1	37-38	37-38	630	645	LSCS	3.3	Good
82.	Anandhi	27	G3P1L1A1	38-39	38-39	640	625	Vaginal	3.1	Good
83.	Devi	28	Primi	38-39	37-38	625	610	LSCS	3.2	MSAF
84.	Esthar	30	Primi	37-38	37-38	595	615	Vaginal	2.9	Good
85.	Nagajothi	30	G2P1L1	38-39	38-39	610	600	Vaginal	3.0	Good
86.	Sobana	29	G2A1	37-38	36-37	605	620	Vaginal	3.1	MSAF
87.	Kokila	30	Primi	37-38	37-38	588	602	LSCS	2.8	Good
88.	Kousalya	32	G2A1	37-38	36-37	595	615	Vaginal	3.1	LOW APGAR
89.	Chinnamma	33	G3P2L2	38-39	37-38	625	610	Vaginal	3.2	Good

90.	Vanaja	32	G2P2L2	38-39	37-38	630	645	Vaginal	3.5	Good
91.	Bharathi	33	G4P1L1A1	37-38	38-39	610	625	LSCS	3.1	Good
92.	Soraja	34	G3A2	38-39	37-38	620	635	Vaginal	3.25	MSAF
93.	Dhakshyani	35	G2P1L1	37-38	36-37	605	630	Vaginal	3.1	Good
94.	Kalavathy	36	G3P1L1A1	38-39	38-39	626	645	Vaginal	3.25	Good
95.	Krishnaveni	36	G2P1L1	39-40	38-39	680	695	LSCS	3.6	Good
96.	Arthy	32	G2A1	39-40	39-40	676	695	Vaginal	3.4	Good
97.	Saratha	33	Primi	39-40	38-39	685	670	LSCS	3.6	Good
98.	Seetha	33	G2P1L1	39-40	39-40	690	685	LSCS	3.75	MSAF
99.	Thilakavathy	34	Primi	39-40	38-39	682	670	LSCS	3.2	Good
100.	Umamaheswari	35	G3A2	39-40	39-40	690	685	LSCS	3.3	Good

ABBREVIATIONS:

IUGR: Intrauterine growth restriction.

CPR: cerebro placental ratio.

LSCS: lower segment caesarean section.

MSAF: meconium stained amniotic fluid.

USG: Ultrasonogram.

NND: Neonatal death.

IUD: Intrauterine death.

NICU: Neonatal intensive care unit.

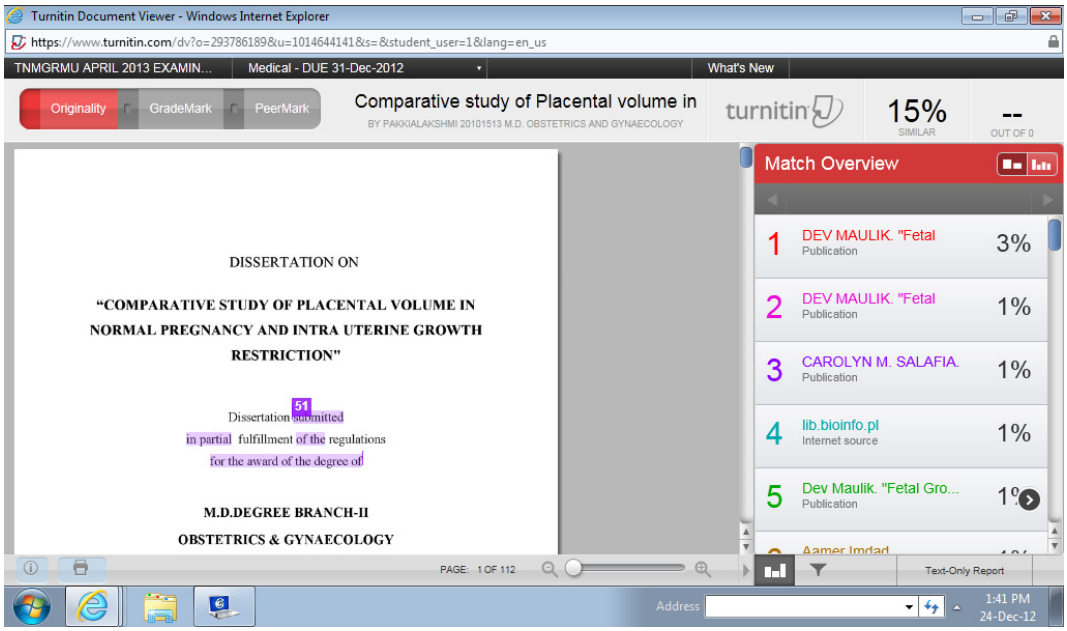
BPD: Biparietal diameter.

HC: Head circumference.

AC: Abdominal circumference.

FL: Femur length.

EFW: Estimated fetal weight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