COMPARATIVE STUDY OF USG ASSESMENT OF PLACENTAL VOLUME AND PLACENTAL BED VASCULARITY IN NORMAL PREGNANCY AND IUGR

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I, **Dr. A. KANIMOZHI,** solemnly declare that the dissertation titled **"COMPARATIVE STUDY OF USG ASSESSMENT OF PLACENTAL VOLUME AND PLACENTAL BED VASCULARITY IN NORMAL PREGNANCY AND IUGR"** is a bonafide work done by me at Govt. R.S.R.M Lying in Hospital, under supervision and guidance of ***Prof. Dr. KUPPULAKSHMI, M.D.,D.G.O.,*** in Department of Obstetrics and Gynaecology, Stanley Medical College, Chennai. This thesis is submitted to The Tamil Nadu Dr.M.G.R Medical University in partial fulfilment of the rules and regulations for the M.S Degree examinations in obstetrics and Gynaecology to be held in May 2019.

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PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled **"COMPARATIVE STUDY OF USG ASSESSMENT OF PLACENTAL VOLUME AND PLACENTAL BED VASCULARITY IN NORMAL PREGNANCY AND IUGR"** of the candidate **Dr. A.KANIMOZHI** with registration Number 221616053 for the award of MASTER OF SURGERY in the branch of OBSTETRIC AND GYNAECOLOGY. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 3 percentage of plagiarism in the dissertation.

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INTRODUCTION

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

Restriction in growth implies failure of the fetus to realize its genetically endowed growth potential. Growth potential determination of an individual fetus however remains difficult. Many studies 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 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. 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 10^{th} percentile. This standard is arbitrary & it may lead to misdiagnosis of growth restriction. A more rigorous threshold such as 5^{th} or 3^{rd} 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 10^{th} percentile & they have normal Ponderal index.

Causes are mainly Infection in utero (HERPES SIMPLEX, RUBELLA, CYTOMEGLO 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 neuro developmental dysfunction.

TYPES 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& placental bed vascularity that is measured antenatally by ultrasound over the adverse prenatal outcome of the IUGR fetuses.

AIM OF THE STUDY

1. To estimate the placental volume& placental bed vascularity by ultrasound.

2. To estimate the placental volume immediately following delivery.

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

4. To estimate the placental volume & placental bed vascularity in IUGR and NORMAL pregnancy.

5. To correlate the adverse perinatal outcome with placental volume &placental bed vascularity 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 low prepregnancy BMI, preeclampsia, chronic renal disorders ,vasculopathy, infections.

Gestational age determination is the most important in diagnosis of IUGR.

1. **Clinical method**: Measurement of symphysio fundal height & abdominal circumference are the most common clinical methods. Symphysio fundal height increases by 1 cm/ wk& it coincides with gestational age between 18-30weeks.Lag of fundal height of 4 wks is suggestive of moderate IUGR &lag of > 6 wks is suggestive of severe IUGR. when used alone this method has low sensitivity.

   Both RCOG and ACOG recommend this simple technique to find 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 clinical method and sonographic study and concluded the detection rate of IUGR for these two groups was similar (25% for ultrasound and 11 % for symphysiofundal height; RR 1.36, 95% CI 0.1.99)

2. **ULTRASONOGRAM** :Several parameters are used in diagnosis of IUGR.Among them 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 has also been used in detection of IUGR.

   Mckenna et al (2003)[2]done and studied ultrasound examination of patients consisting of Estimated fetal weight, Amniotic fluid index and placental grade at 30-32 weeks and 36-37 weeks and clinical methods like symphysiofundalheight alone. They reported the prevalence of IUGR was 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 related well with outcome of the fetus. Alteration 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 and maternal causes, the placenta might the etiology. Various authors recorded contradictory histological and morphological findings while comparing the placenta of IUGR Pregnancies to that normal pregnancies.

ETIOLOGY OF IUGR :

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

MATERNAL FACTORS :

1. Maternal hypertensive disorders:

Hypertensive disorders present in 30-40% of IUGR pregnancies. Pre eclampsia, chronic hypertension, preeclampsia, autoimmune disorder, nephropathy, presentational diabetes are associated with maternal vasculopathy leads to fetal growth restriction.

According to Odegardvattern / Nilsen et al (2000)[10] preeclampsia has 4 fold increase of having IUGR babies (RR= 4.2; 95% CI2.2-8.0).

The severe preeclampsia and the early onset of pre eclampsia associated with low birth weight. Long, abel, Beisher (1980)[11] reported that, Low birth weight was 5% in mild pre eclampsia (95% CI 3- 6) & 12% with severe disease (95% CI 9-15) and it is was 23% with early onset disease (95% CI 18-29). There is evidence that elevated diastolic blood pressure withoutproteinuriais associated with small for gestational age but 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 IUGR .

2. Maternal autoimmune disorders:

Maternal autoimmune 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 APLA is positive.

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

3. Thrombophilia :

There is controversy in association between IUGR and maternal Thrombophilia.Howley/walker/Rodger(2005)[14] done meta analysis of 10 case control studies.They 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 recreational drugs& addictive substances associated with IUGR. However causal relationship is difficult to establish due to other associated confounding factors like malnutrition, multiple substance abuse, street and other lifestyle variables.

Maternal smoking is associated with fall 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) noted average birth weight reduction of 6% when smoking was continued throughout gestation compared with only 1.7 % when it was stopped after 1st trimester and this effect 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 is associated with hypertension than not associated with hypertension (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 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 on the severity of deprivation & the period of gestation.

7. Environmental pollution

Epidemiological investigations on the impact of environmental pollution on pregnancy outcome shows 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 anomaliesare strongly associated with IUGR. About 7% of IUGR 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 is associated with increased incidence of fetal malformations that leads 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 most commonly associated with IUGR. Abnormal imprinting results in abnormal phenotypes including fetal growth restriction and dysmorphic features. In praderwilli syndrome loss of function of imprinted genes on the paternal allele in 15q 11-13 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 associated with increased 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 is 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 causes decrease in cell population may be the most frequently associated mechanism in growth restriction.

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

Bacterial infection is 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 gestation 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 singleton pregnancy. Guenwald (1966)[24] demonstrated the growth curves of singleton and twins were sameupto 30-32 weeks after which the growth of the twins lagged behind that of singleton.

Small for gestational birth 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,poor weight gain,lowprepregnancymass 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, chorionic villitis, placental infarcts, haemorrhagicendovasculitis, placental haemangioma, chorioangiomas are some of the placental conditions associated with IUGR.

COMPLICATIONS OF IUGR:

ANTENATAL :

Antenatal and intrapartum hypoxia, acidosis are the 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 shows evidence of IUGR. Morrisen and Olsen (1985) found 26% of stillborn weighting <2.5 kgs is associated with IUGR.

OLIGOHYDROMNIOS:

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

INTRAPARTUM COMPLICATIONS :

The incidence of intrapartum hypoxia and acidosis are higher 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 is also a major cause of mortality and morbidity.

Persistent fetal circulation due to perinatal hypoxia and acidosis.

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

Neonatal encephalopathy is an essential component of cerebral palsy secondary of 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 necrotizing 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) Kok et al (1998),)[9.]

Several studies shows 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 is accepted that IUGR was associated with fetal hypoxia resulting partially from alternation 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 result, the uteroplacental flow impendence progressivly 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 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 to 32, vasculogenesis occurs in which capillary networks formed will provide 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 characterized 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 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 shows 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 term IUGR placentas (Jansson, Yivar et al 2002)[28.]

ASSESMENT OF PLACENTAL GROWTH:

There are so many standard placental growth parameter 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/ neurologic abnormality at 7yrs.

2. **Location of umbilical cord insert in from the edge of the placenta:** Cord malposition may be due to abnormal growth of placenta towards one side or abnormal positioning of the embryo. Nayesanalysed 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 placental thickness which reflects the extent of nutrient exchange surface of the placenta essential for the successful and adequate fetal growth. Increased disk thickness decrease the placental efficiency and so abnormally thick placenta is also associated with adverse pregnancy outcome (Radio, Ghazzi et al 2004)[30.]

5. **Feto placental weight ratio.**

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

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

In 1984 the first fetal placental volumes studied by USG were constructed by Brinkley at el. After the development of 3 dimensional USG imaging assisted by computer technology it is possible to measure and calculate fetal & placental volume quickly & accurately ,measuring and monitoring the fetal and placental volume at different gestational ages may improve our understanding about pathophysiological mechanisms of fetal & placental growth. Fetal and placental volumes can be used in screening of fetuses with chromosomal anomalies ,IUGR , preeclampsia. Some reports in literatures says that increase in placental volume preceding preeclampsia & decrease in placental volume preceding IUGR & decrease in fetal volume in fetuses with chromosomal anomaly.

Wallance et al (2004) concluded the small size of the placenta per se rather than alternation in the nutrient metabolism or transferring capacity has a major limitation to fetal growth.

Thame& colleagues (2005)[35] have shown the effects of maternal anthropometry on birth weight is likely to be mediated by effects of maternal anthropometry on placental volume. These effects operate in pregnancy and alter both the absolute placental volume at 14 wks and rate of growth of placenta between 17 & 20 wks.

Clap & colleagues (2004)[36] identified a 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)[37] showed an abnormal IGF signaling was linked to human IUGR.

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

I.Cetin G, Alvino (2009)[40] showed that IUGR has been linked with a specific placental phenotype associated with defects in placental transport function that lead to fetal undernutrition. Both placental transport and metabolism may be affected and modifies 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 , 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 severity of the disease.

I Cetin, J M, Foidart, M Moazzo (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 , metabolic, vascular, coagulative, autoimmune as well as infections can influence fetal growth by damaging the placenta. Strict definition of IUGR and its severity are needed so as 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 are potentially associated with IUGR.

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

Studied the effect of malaria on placental volume measured using 3 dimensional ultrasound. Malarial parasites and histopathological changes in placenta is 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 is an indicator of IUGR and placental insufficiency.

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

Studied relation between stillbirth and IUGR. Early detection and management of IUGR leads to reduced morbidity and mortality. They

reviewed the effectiveness of fetal movement count and Doppler for detection and surveillance of high risk pregnancy and the effect of it 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 routine 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 suggests that detection and management of IUGR with 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 in 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 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 than conventional 2 dimensional sonographic measurement.

Hafner, philippschuchter (2002) [45]

Suggested that the prognostic influence could be shown for placental volume, gestational age at the time of measurement and maternal weight at the time of 1st visit.

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. 3 dimensional power Doppler indices related to placental vascularisation were calculated. They found that the 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 the 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 the gestational age was lower in AEDV group when compared to normal group. The birth weight 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 la (1998) revealed the measurement of placental volume between 16 & 23 wks of gestation has a sensitivity of 53.5% in the prediction of IUGR and neonatal birth weight below 10^{th} percentile.

HLAFNER, PHILIPP, SCHUCHTER (2002) [49]

Conducted prospective study in 382 women with singleton uncomplicated pregnancies at 16-23 wksinorder to investigative the value of 2^{nd} trimester 3- dimensional sonographic placental volume measurement to predict infants who are $<10^{th}$ 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 measurements (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 a 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 subjected for a routine prenatal ultrasonogram at 11-14 wks& the mean pulsatility indexof uterine artery was calculated and, placental volume was measured using 3- dimensional sonogram. The outcome considered were development of preeclampsia & pre eclampsia requiring delivery < 32 wks. On observation they found 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 eclampia requiring delivery before 32 wks (66.7% vs 67%). The combination of both gave better results with sensitivity of 68.7 % in predicting preeclampsia & 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 had better results than done alone.

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

They studied 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 number, & significantly longer than control cases (218 vs 137 um). They also had fewer branches (4/loops vs 6/loops, $p< 0.06$) 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 impedance at capillary level & it might be account for the impaired gas and nutrient transfer across the placenta.

THAME OSMONDE, WIKS[52]

They investigated the ability of 2^{nd} trimester placental volume measurement by ultrasonogram in the prediction of birth weight of the fetus. They selected 512 women and measured fetal anthropometry &

placental volume serially at 14, 17, 20 wks. The outcome was focused 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 might be the 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 comparedthese 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.

JIE DUAN ANNE-CLAIRE CHABOT-LECOANET ESTELLE PERDRIOLLE-GALET

Evaluation of utero-placental vascular modification during pregnancy using usg recently became possible. Since 2004, it is possible to quantify placental and myometrium vascularisation by 3D power Doppler angiography (3DPD).This method allows to study the vascularisation of organ of interest. Quantification is based on calculation of the ratios of voxels with Doppler signals to the intensity of Doppler signals in the voxels. Three typical indices of a volume of interest were calculated by this method: the vascularisation index (VI), flow index (FI) and vascularisation-FI (VFI). The feasibility and reproducibility of Doppler signal quantification by calculating VI, FI and VFI were found to be satisfactory in vitro and in vivo

MATERIALS AND METHODS

This prospective analytical study was conducted at government RSRM lying in hospital, Chennai coming under the stanley medical college, Chennai from 2017 to 2018 Ethical committee clearance was obtained to undergo the study.

The patients referred as IUGR beyond 32wks up on 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 $< 10^{th}$ 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 oligohydramnios in which there was difficulty in localizing the placenta were excluded from this study.

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

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, immunization, and 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 2 Dimensional ultrasound examination was GE with a 5 MHz curvilinear probe.

Fetal parameter 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 localization was done.

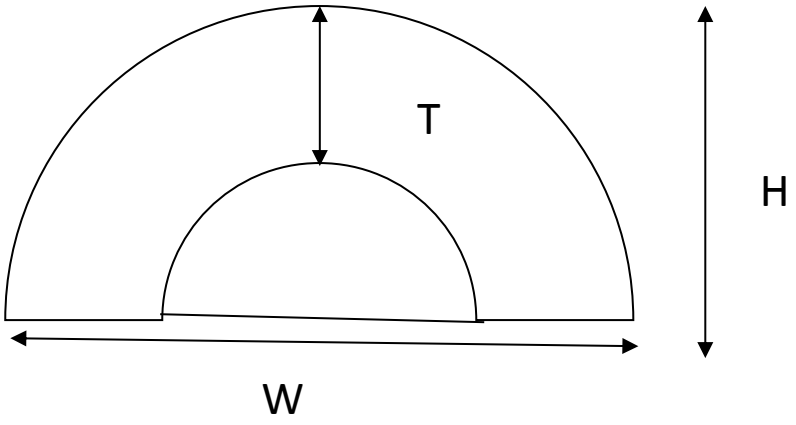
The probe was adjusted for seeing both edges of the placenta in the same image and the image was frozen. With this placental width and height were measured. Then placental thickness was measured possibly at the level of cord insertion.

Measurement of placental volume was done by using the convex-convave shall formula.

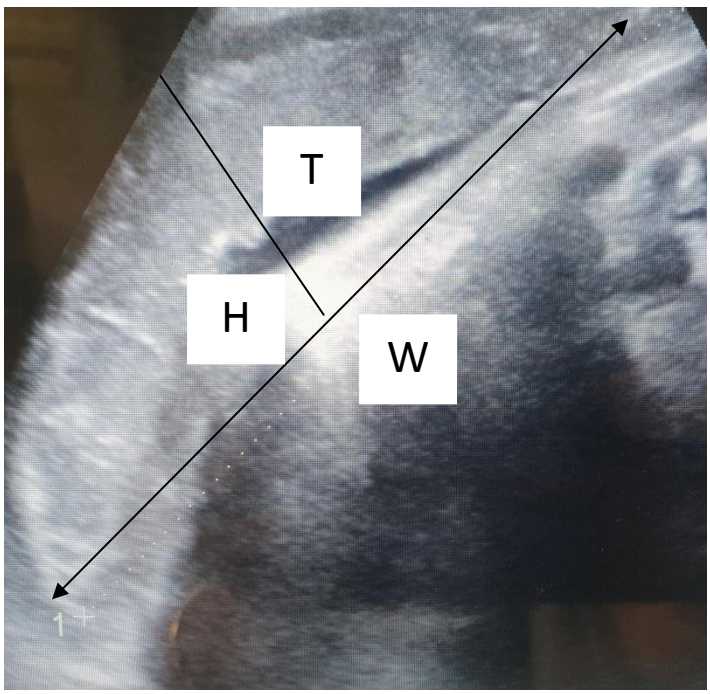
$V = \pi\ t/6\ X\ (4H(W-T) + W(W-4T)+4T^2)$;

H= PLACENTAL HEIGHT,

W= PLACENTAL WIDTH.



Diagrammatic representation of measurement of placental volume



T – Thickness

W – Width

H- Height

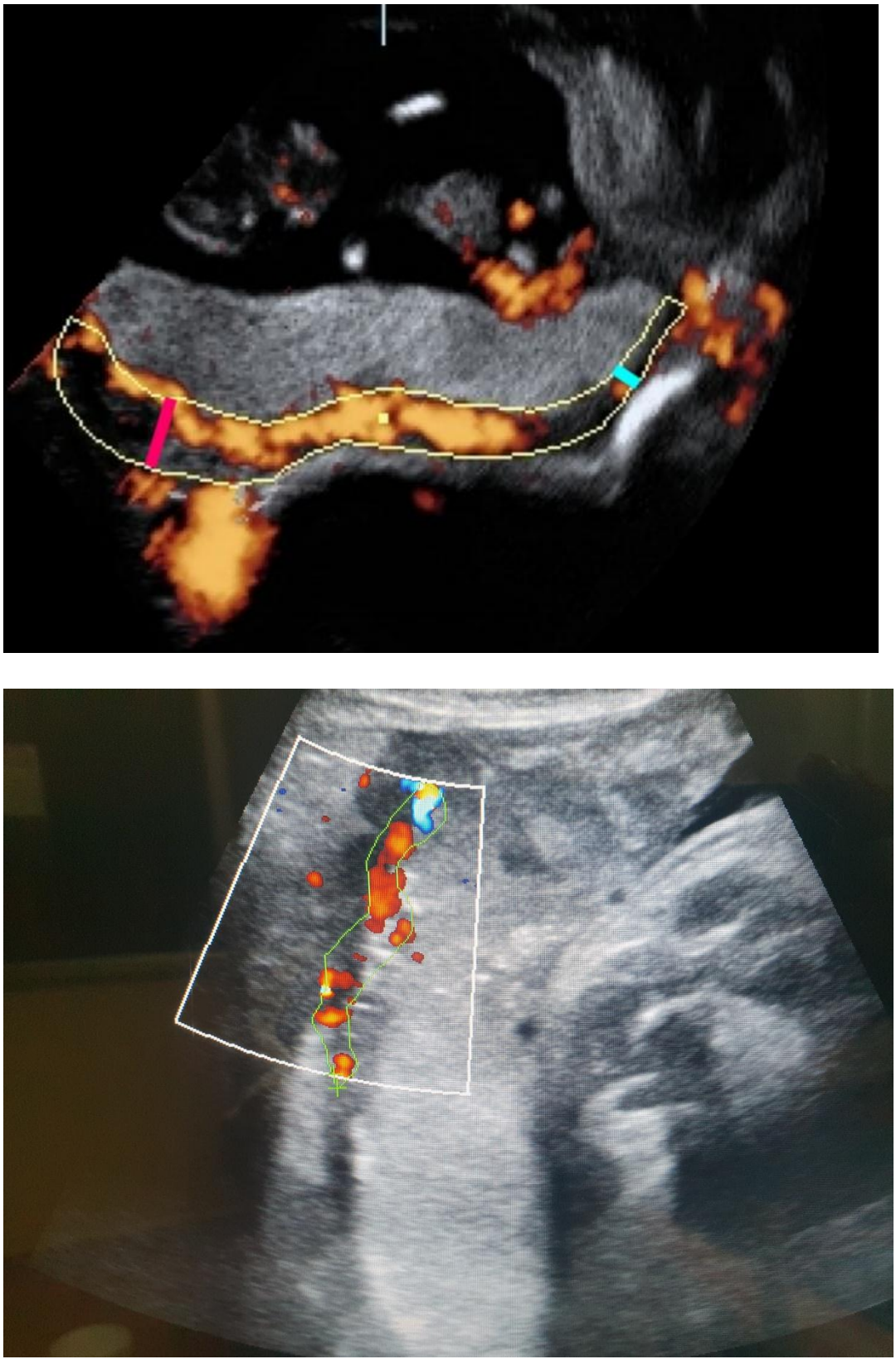
USG measurement of placental volume

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 localized 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 equals 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 calculatedfrom the RI of umbilical and middle cerebral artery (RI of MAC /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.

MEASUREMENT OF PLACENTAL BED VASCULARITY

For the estimation of number of vessels in the placental regions we used the VI to count the number of colour voxels in a particular region of interest in comparision to its grey voxels which gives the percentage of colour to grey voxels.It is known that VI is significantly influenced by power doppler settings like gain,signalpower,pulse repetition frequency.To measure the VI in the placental bed a power doppler colour box was placed over the entire placenta and the adjoining myometrium.

Inorder to calculate placental bed vascularity(PBVI),placentas were rotated in a horizontal position in both A and B plane.using an inbuilt programm for volume measurements ,the border between placenta and deciduomyometrium was carefully traced in a A-plane by caliper.The caliper was then moved into deciduomyometrium. The thickness of deciduomyometrium or placental bed measured,however, as it can differ from millimeters to centimeters.To solve this problem,placental bed was measured from its direct attachment at the placenta upto a thickness of 1 cm,which is made possibly by using the display measure ,given by the machine.only if the placental thickness is less than 1 cm this smaller value taken for measurement.The placenta was then rotated by 30 degree in a horizontal plane and tracing was repeated.As the angle size of the horizontal rotation is 30 degree it takes six cuts to completely define the placental borders.After this machine calculates the VI automatically.



USG Measurement of placental bed vascularity

Patients with normal fetal growth were selected as control. The inclusion criteria for selection were same that of IUGR to avoid errors in comparision. 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, 1^{st} trimester ultrasonogram, 2^{nd} trimester anomaly scan,3^{rd} trimester interval growth were noted. In multigravidas, history regarding pervious 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 localizing 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 the flat surface and maximum, minimal width were measured with an inch tape. Maximum height was measured. With the 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.

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 5th percentile, 82% of birth weight are below 10th 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 are that passes through the thalami and 3rd ventricle is acceptable for measuring BPD & it is measured form outer edge of the skull of the proximal surface to the inner 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 management. 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 ag 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 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. Accirdubgti 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 assessed 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 analyzing 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 in 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 impedance.

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 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 I 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	058-0.71
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 centralization of flow.
2. Increased umbilical artery resistance with centralization 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. Alternation 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 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 or umbilical artery.

CEREBRO PLACENTAL RATIO:

It is the ratio between RI of MCA & RI of UA. According to Aras (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 normal pregnancy as control group and 100 IUGR pregnancy as study group. The following observations were made.

1. GESTATIONAL AGE:

In our study IUGR above 32 weeks of gestation were taken . 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
32 -34 WKS	2	8
34-36 WKS	43	45
36-38 WKS	40	40
38-40 WKS	15	7

According to the above date, the commonest gestational age group presented was 34-36 weeks .

2 .MATERNAL AGE

In IUGR group,76 patients presented in the group of 25 to 36 years

MATERNAL AGE(YEARS)	IUGR	NORMAL PREGNANCY
18-24	24	22
25-30	28	36
31-36	48	41
>36	0	1

3. PARITY

In our study both primigravidas and multigravidas presented equally and patients in normal group also selected like that.

PARITY	IUGR	NORMAL PREGNANCY
PRIMI	37	39
MULTI	63	61

4. PLACENTAL VOLUME

The average placental volume observed according to gestational age as follows

GESTATIONAL AGE	IUGR	NORMAL PREGNANCY
32-34 WKS	325	490
35-37 WKS	492	594
38-40 WKS	586	680

5. IUGRDOPPLER ANALYSIS

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

CPR <1 48

CPR>1 52

6. PBVI

The placental bed vascularity between normal and IUGR is as follows

GA	PREGNANCY	MEAN
33-34 WKS	Normal	31.2
	IUGR	20.79
34-35 WKS	Normal	30.25
	IUGR	22.69
35-36 WKS	Normal	31.13
	IUGR	24.49
36-37 WKS	Normal	30.43
	IUGR	22.59
37-38 WKS	Normal	30.39
	IUGR	22.22
38-39 WKS	Normal	30.34
	IUGR	24.2
39-40 WKS	Normal	31.1
	IUGR	27.3

7. MODE OF DELIVERY

All patients were observed till delivery.mode of termination noted.

MODE OF DELIVERY	NO.OF PATIENT
VAGINAL	26
LSCS	74

8. BIRTH WEIGHT OF THE BABY

Birth weight of the baby in IUGR group noted

BIRTH WEIGHT	NO.OF BABIES
<1KG	2
1-1.5	20
1.6 TO 2	49
2.1 TO 2.5	29

9. OUTCOME OF THE BABY

Among 100 babies 53 babies had good outcome without any perinatal mortality or morbidity.The remaining 47 babies had adverse outcome.

OUTCOME OF THE BABY	NO.OF BABIES
GOOD OUTCOME	53
IUD	2
NND	9
LOW APGAR	24
MSAF	12

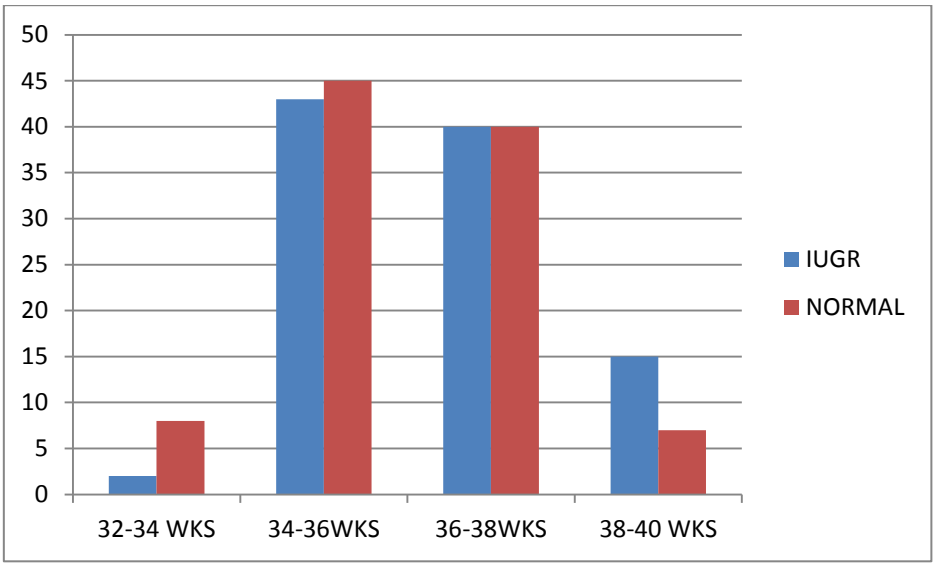
DISCUSSION

The 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 32 weeks were taken very preterm IUGR were excluded from the study to avoid errors in assessing perinatal outcome.Among 100 patients with IUGR,85 patients were between 32 to 37 weeks.patients between 38 to 40 weeks were 15only.This shows that incidence of early IUGR is more common than that of late IUGR.

GESTATIONAL AGE DISTRIBUTION

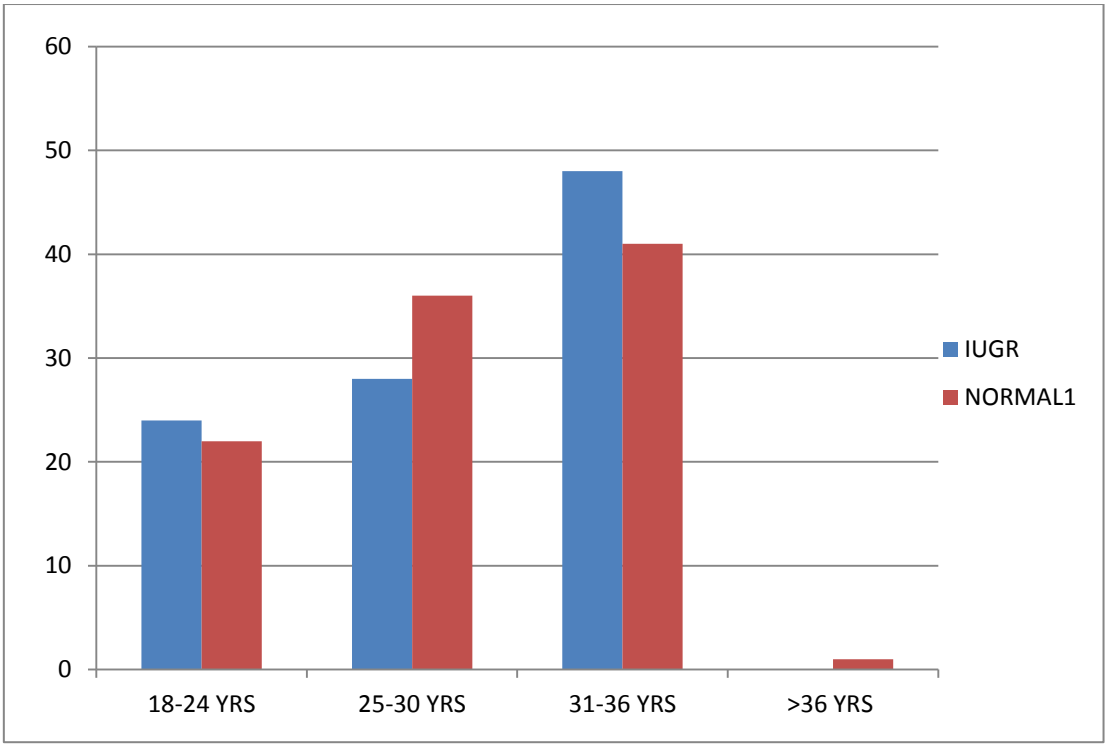


This showed maximum number of IUGR presented in this study was between 34 to 36 weeks.

2. MATERNAL AGE

In patients with IUGR,76 patients were in the age group of 25 to 36years.It shows there is positive association with increasing maternal age and IUGR.the most common age group presented were 31 to 36 years.

Maternal Age Distribution



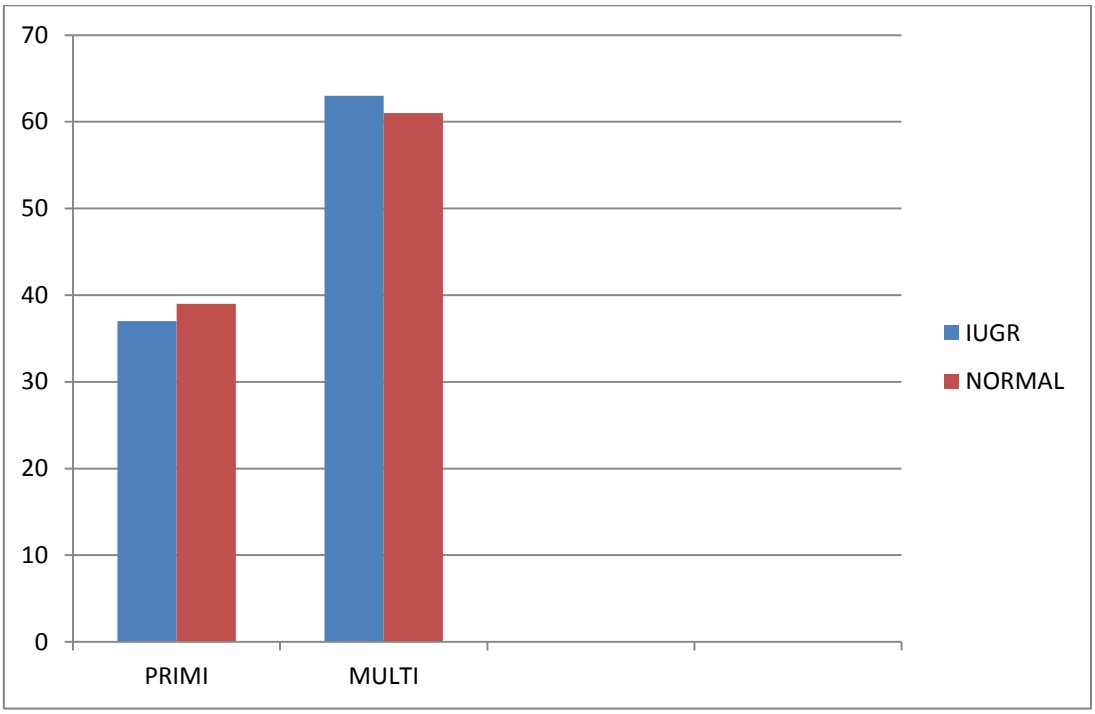
This shows there is positive correlation between advancing maternal age and IUGR. This denotes that advancing maternal age may be an independent risk factor for IUGR.

3. COMPARISON OF PARITY:

In our study both primi and multi were presented equally.

Taj mohammed, Asmat are (200) concluded that primiparity was also a significant risk factor for IUGR. Similar findings were reports by Fikree et al & Thompson et al.

Parity



Patternson RM, Gibbs, Woods (1986) reported, prevalence of recurrent IUGR is significantly related to 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 2 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	-	1

4. COMPARISON OF PLACENTAL VOLUME:

De, paula CF, ruano R, Campos JA (2008) 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)
32Wks	326	346	384
33Wks	184	315	494
34Wks	160	327	486
35Wks	186	350	584
36Wks	320	474	590
37Wks	198	520	586
38 Wks	200	577	640
39Wks	540	644	675

Based on the above nomograms, the Placental volume was graded in to 3 types as follows

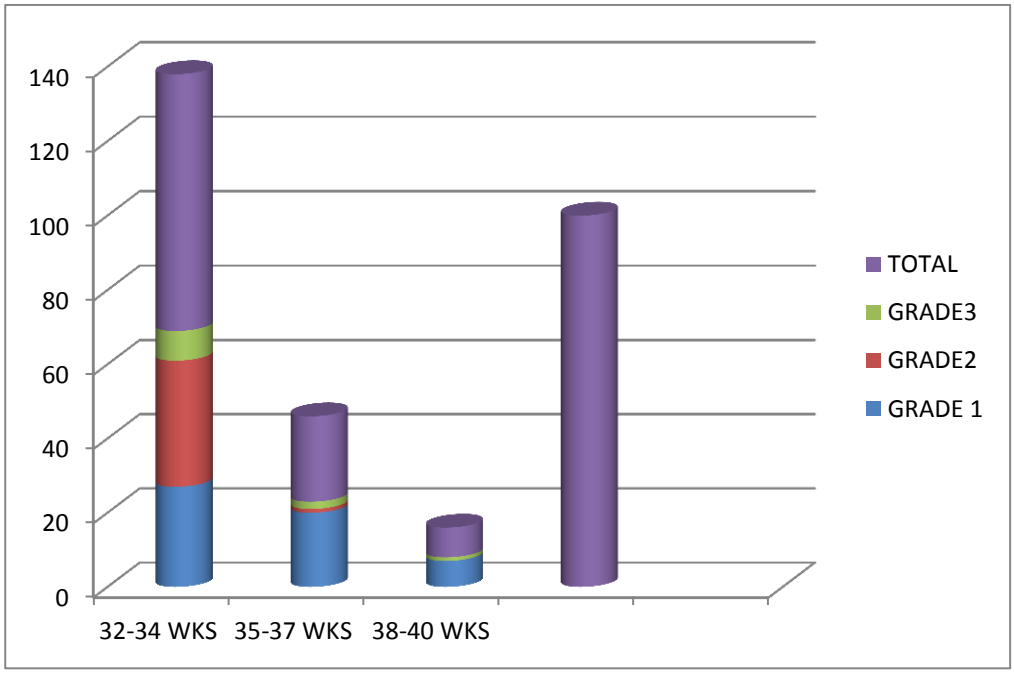
1. Grade -1: The placental volume falls above 50^{th} percentile but below 90^{th} percentile.
2. Grade -2: The placental volume falls below 50^{th} percentile but above 10^{th} percentile.
3. Grade-3 : There is severe reduction in placental volume & falls below 10^{th} percentile.

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.

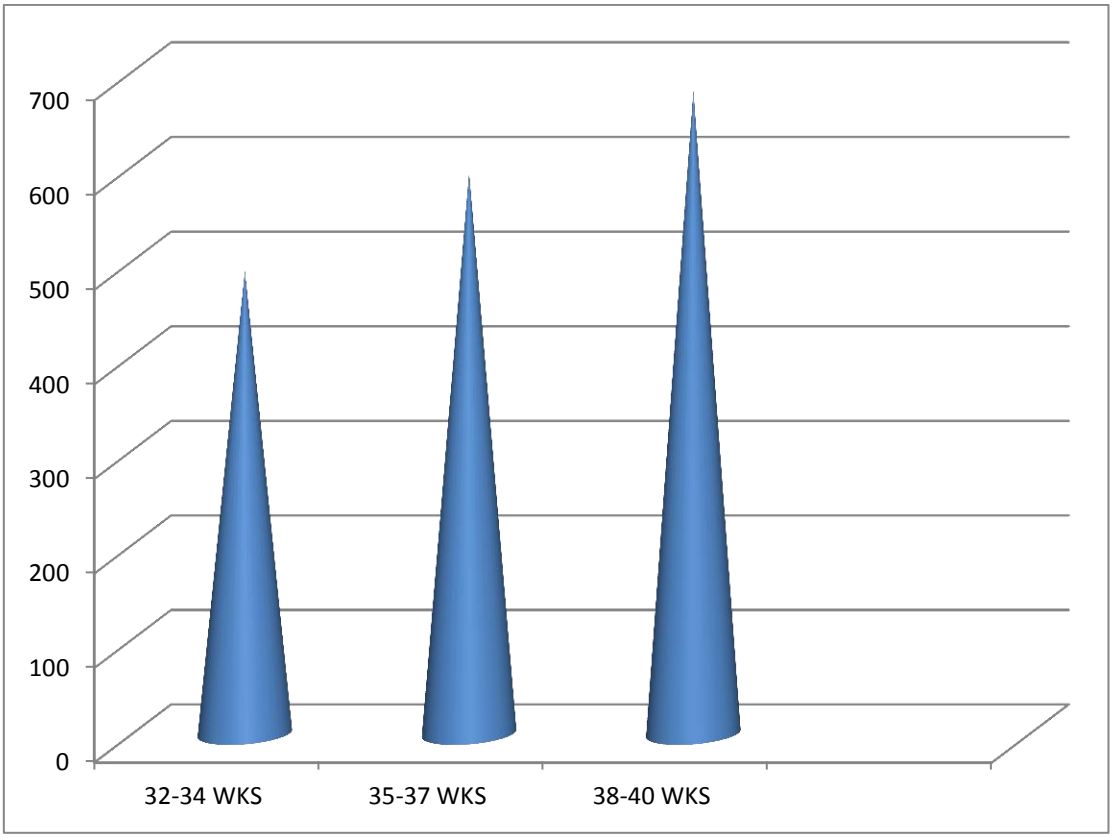
PLACENTAL VOLUME ACCORDING TO GESTATIONAL AGE



Comparing the grading of the placental volume, most of the patients had grade1 placental volume (n=54) where the placental volume was above 50th percentile. In 32-37 weeks, most of the patients has grade 2, grade 3 placental volume than grade 1 placental volume. Whereas in 38-40weeks 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 90th percentile.

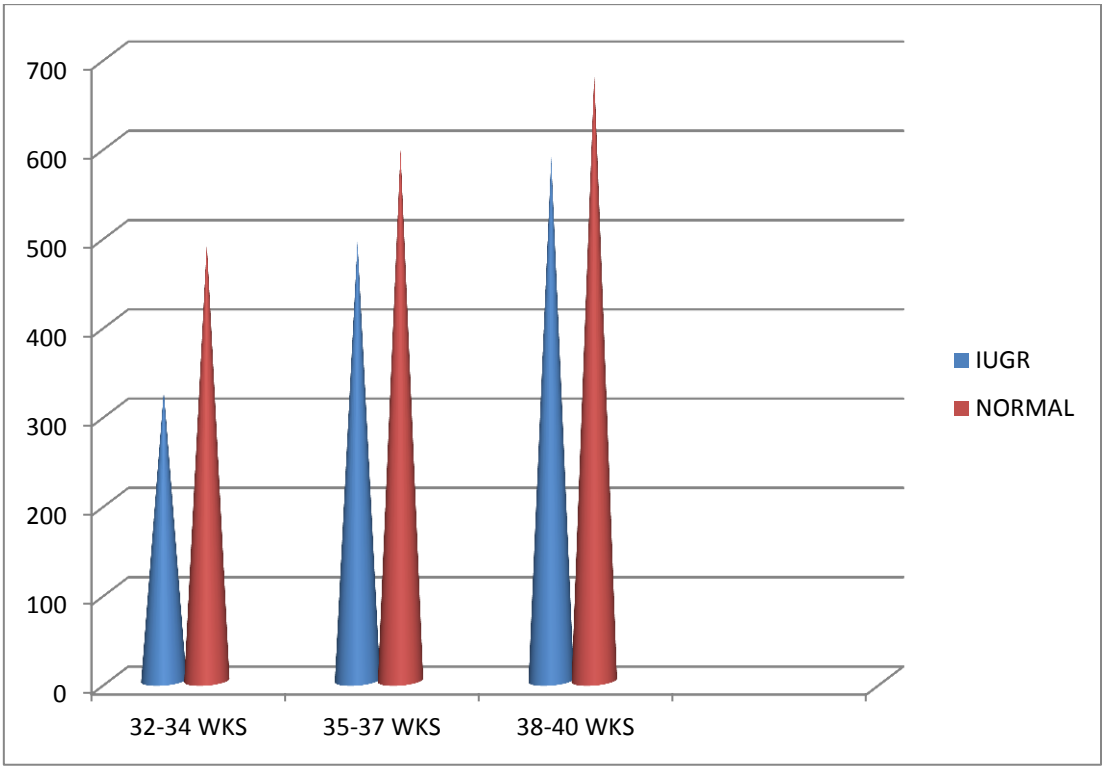
PLACENTAL VOLUME IN NORMAL PREGNANCY



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

GESTATIONAL AGE	IUGR	NORMAL PREGNANCY
32-34 WKS	325CM^3	490 CM^3
35-37 WKS	492 CM^3	594 CM^3
38-40WKS	586 CM^3	680CM^3

PLACENTAL VOLUME ACCORDING TO GESTATIONAL AGE IN NORMAL AND IUGR



This chart 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	400.91	38.177	0.001
NORMAL PREGNANCY	582.82	124.854	0.001

P=0.001** Highly significant. (leavenes 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.

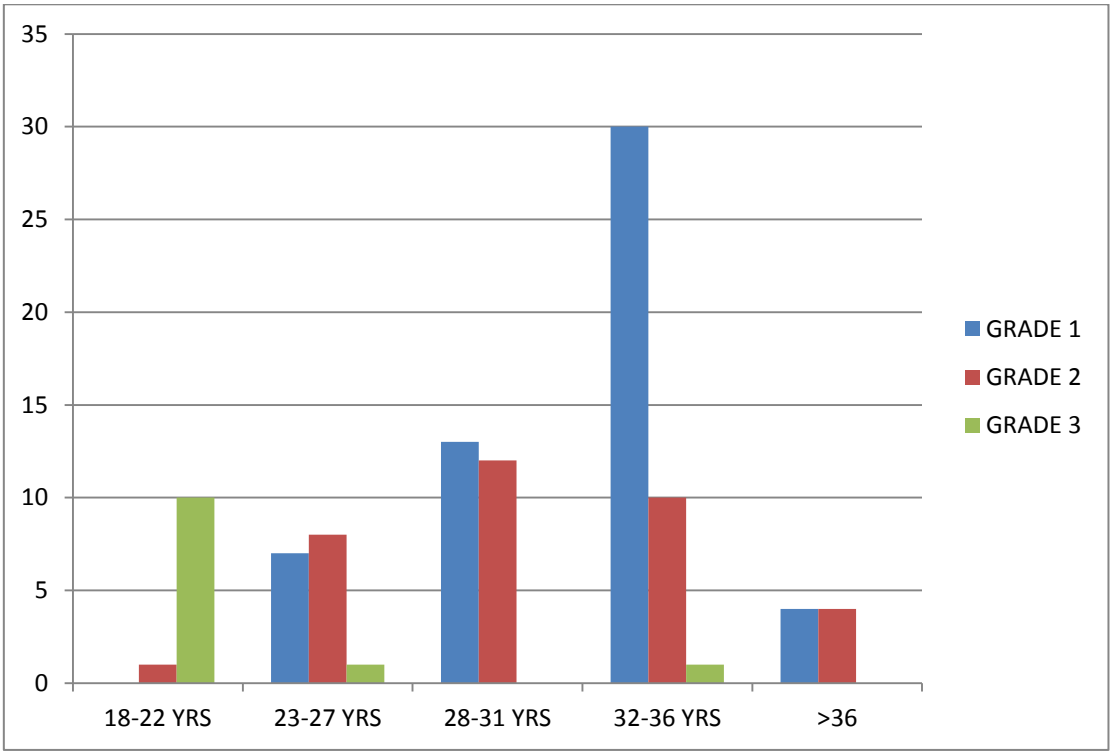
With the above findings, we can conclude that in IUGR pregnancies without any identifiable aetiology, the placental insufficiency of unknown cause plays a major role.

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

PLACENTAL GRADING AND MATERNAL AGE



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 patient had only grade 1 placental volume. In the contrary, 10 patients amount 11 in the age group of 18-22 had grade 3 placental volume.

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

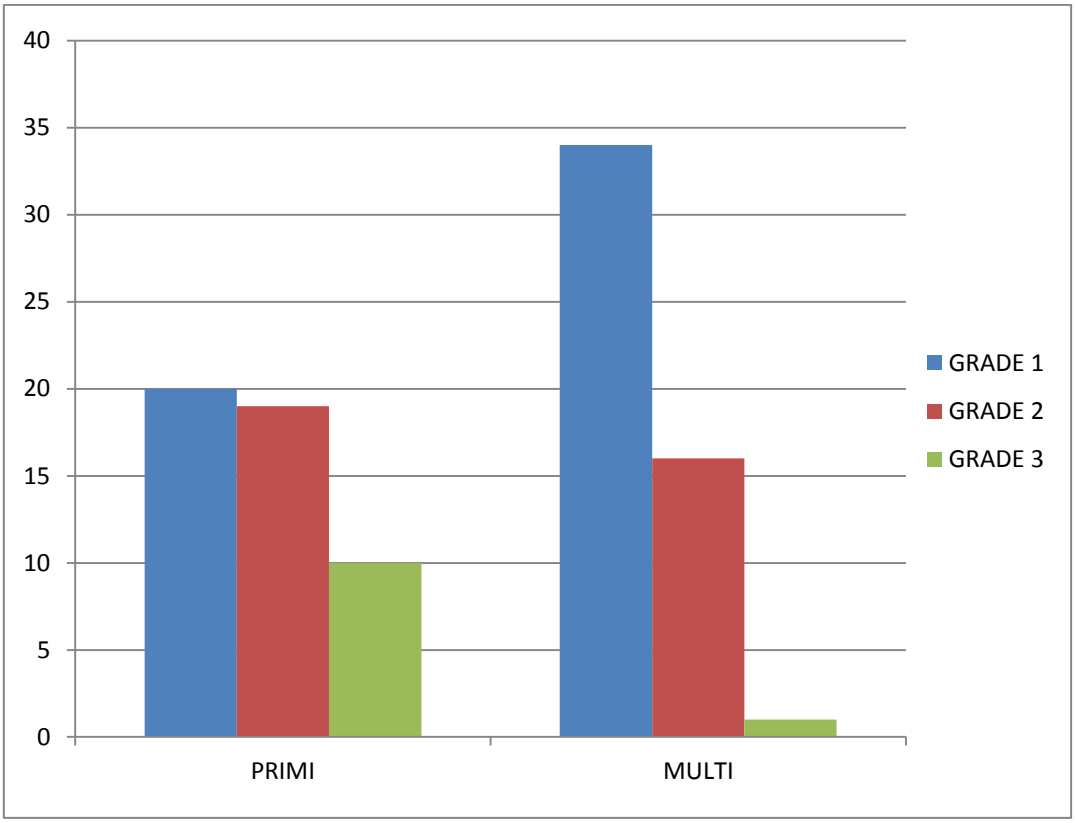
4. COMPARISON OF PLACENTAL GRADING WITH PARITY:

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

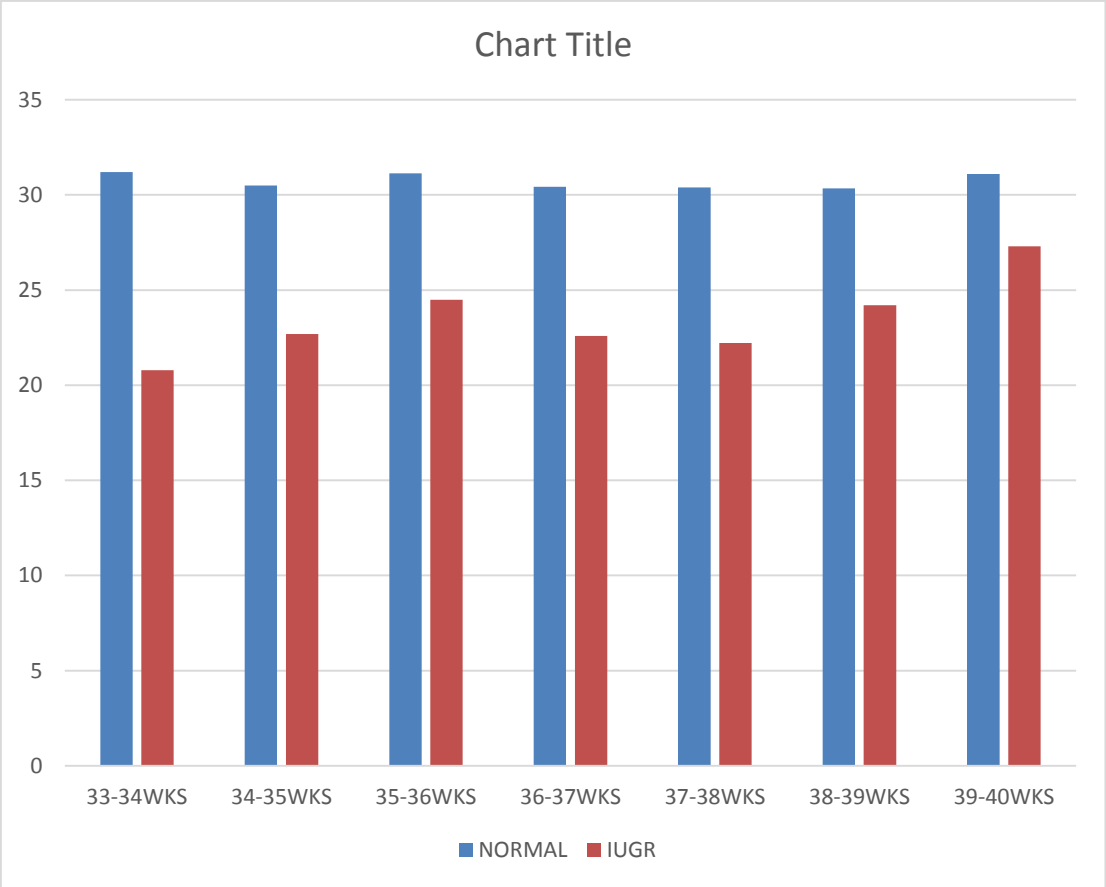
In our study even though both primi& multi were presented equally.

PLACENTAL GRADING AND PARITY



This diagram represents the comparison of placental grading with parity. Here primigravidas had severe reduction in placental volume when compared to multigravidas. This is comparable with the study by tajmohammed, Asmat Ara (2010) who reported the primiparity was also a significant risk factor for IUGR at multivariable level. Similar findings was also reported byFikree et al &Thompsond et al.

PLACENTAL BED VASCULARITY INDEX



A Comparative analysis of PBVI revealed statistically significant differences between normal and IUGR pregnancies.In normal pregnancies PBVI is on range of 30 to 32 than IUGR(20 to 24)

VI was 6-8 times higher for normal than IUGR pregnancies.It clearly shows that placenta of IUGR has fewer blood vessels and decreased blood flow.

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 report were analysed based upon the Cerebroplacental ratio.

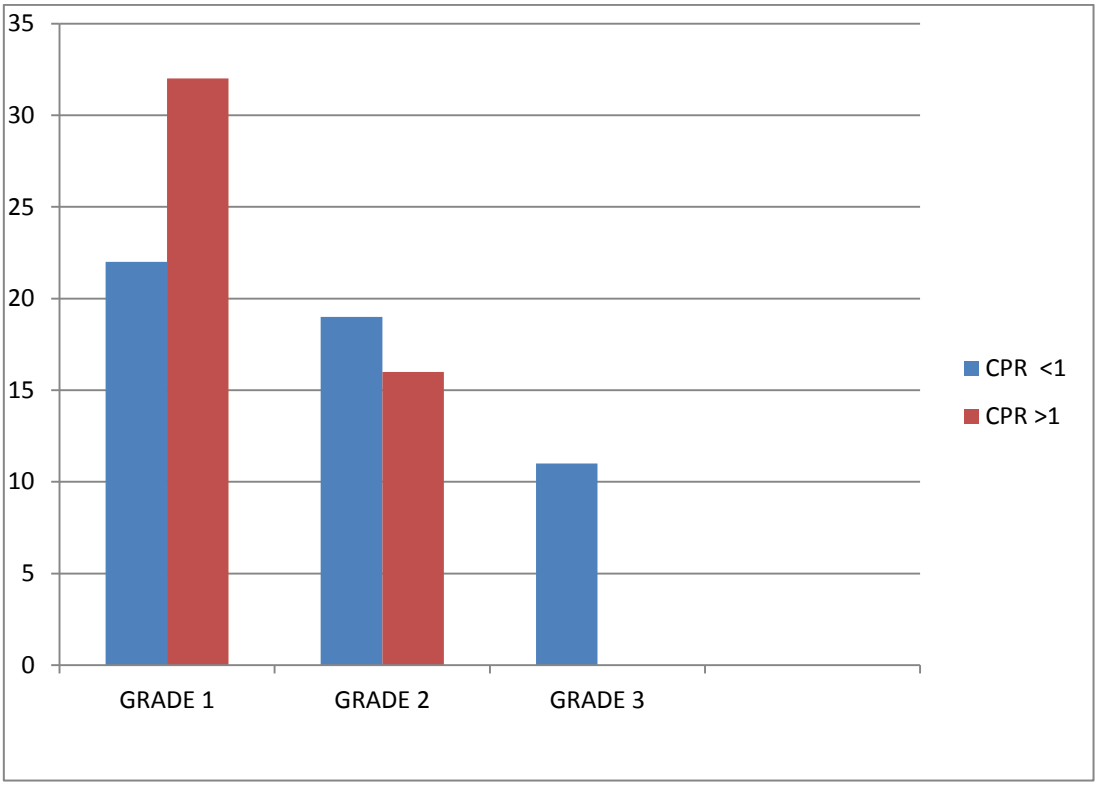
CPR	GOOD OUTCOME	ADVERSE OUTCOME
< 1	20	32
> 1	31	17

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

On analyzing the dates with placental volume grading following was observed.

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

DOPPLER AND PLACENTAL GRADING



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.

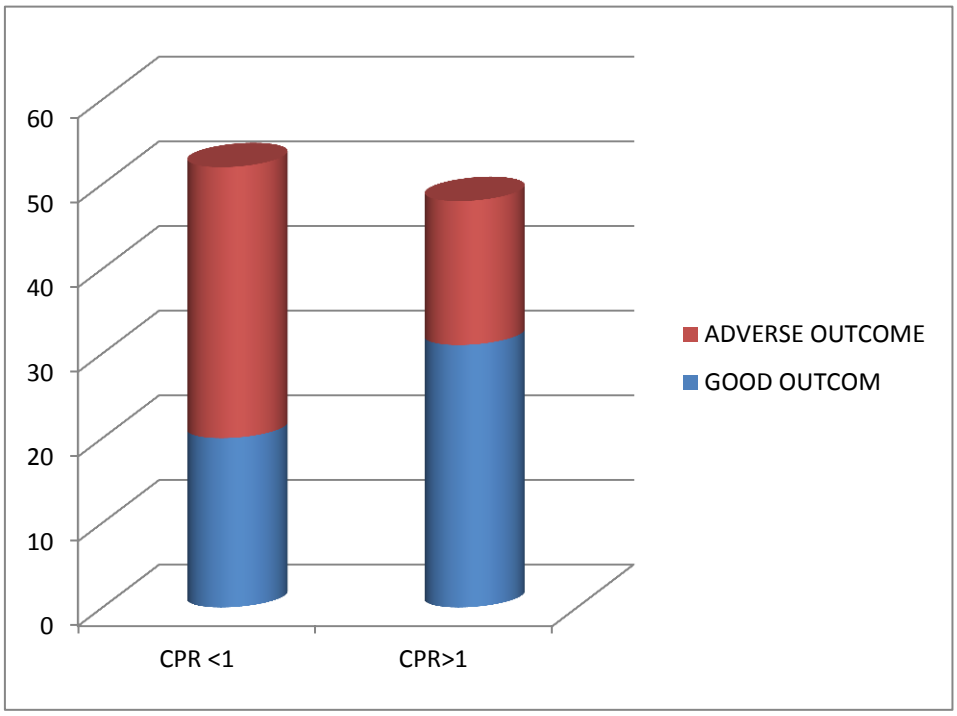
This is comparable with the study done by Dudareniex L, kaluzewski B (2006) in which they compared placental volume with Doppler study in 82 pregnancies between 14-40 wks of gestation. They concluded that PI of umbilical artery correlated negatively with Placental volume, PI of MCA showed no significant correlation whereas the Cerebroplacental ratio showed significant positive correlation with placental volume.

	Good Outcome	Adverse Outcome
CPR <1	20	32
% within CPR	39.2%	60.8%
% within Outcome	38.8%	64.6%
CPR >1	31	17
% within CPR	65.3%	34.7%
%within Outcome	61.5%	35.4%

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

P= 0.009** Highly significant. (Pearsons chi-square test)

DOPPLER AND PERINATAL OUTCOME

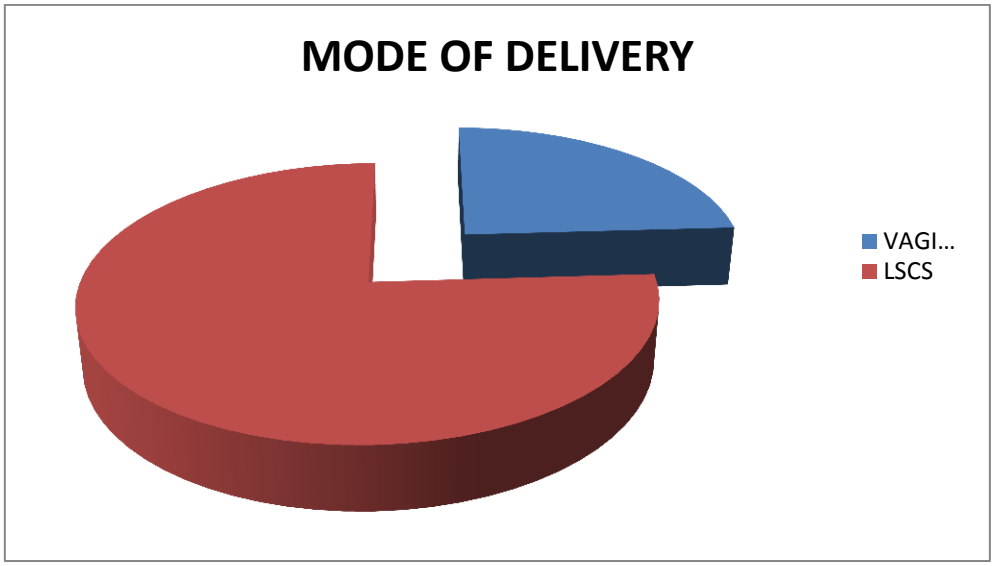


This shows the sensitivity of predicting the perinatal outcome by CPR is 60.08% and the specificity is 65.3%

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 profile, after decision 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 sign of fetal distress.

MODE OF DELIVERY	GRADE 1	GRADE 2	GRADE 3	TOTAL
SPONTANEOUS VAGINAL DELIVERY	1	1	-	2
INDUCED VAGAINAL 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	PRECENTAGE
FAILED INDUCTION	32	43.24 %
NON REASSRING 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 to induction. Some patients in the group of induction were taken up for LSCS for the signs of intrapartum fetal distress. In the Electronic fetal hart rate monitoring, the incidence of non reassuring heart rate pattern was observed more if the placental volume <10th percentile. The commonest non reassuring pattern observed was loss of beat variability followed by absence of accelerations. Spontaneous deceleration were observed in patients with very low placental volume. Severe oligohydramnios was also more in placental volume < 50^{th} percentile.

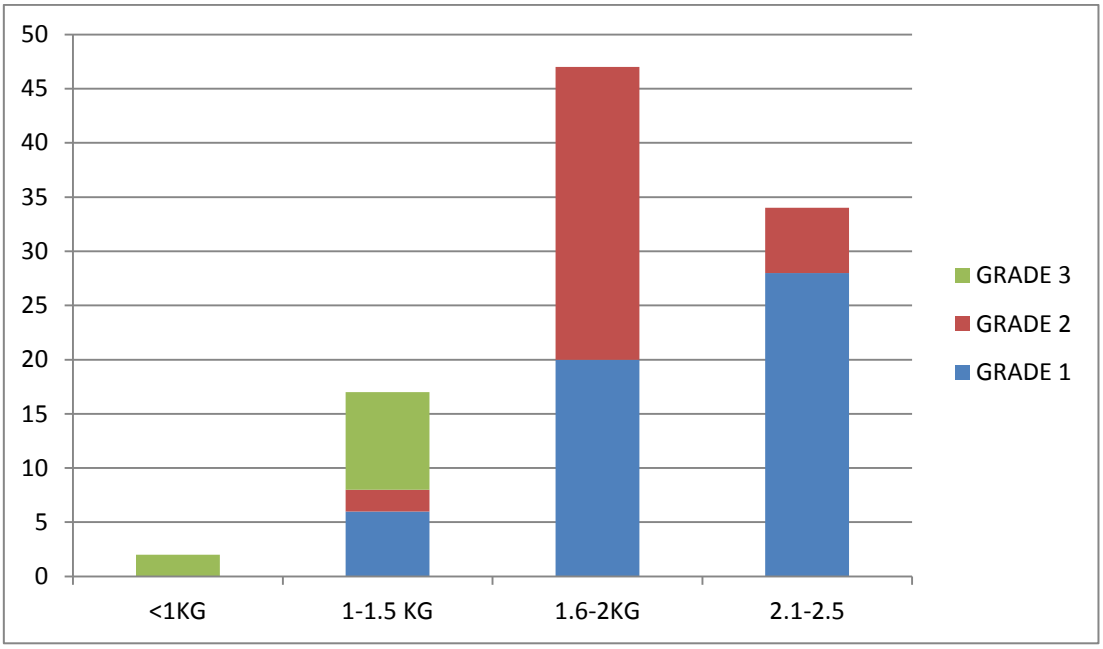
Distribution of birth weight in the IUGR group.

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**	**%**
<1KG	-	-	2	2	2%
1-1.5 KG	6	2	9	17	17%
1.6-2.0	20	27	-	47	47%
2.1-2.5 %	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.

PLACENTAL GRADING AND BIRTH WEIGHT



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	1.99 kg	0.30	0.001
Grade 2	1.82 kg	0.21	0.001
Grade 3	1.25 kg	0.6	0.001

P= 0.009** 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 grade 1 & 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) 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 fetal 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.

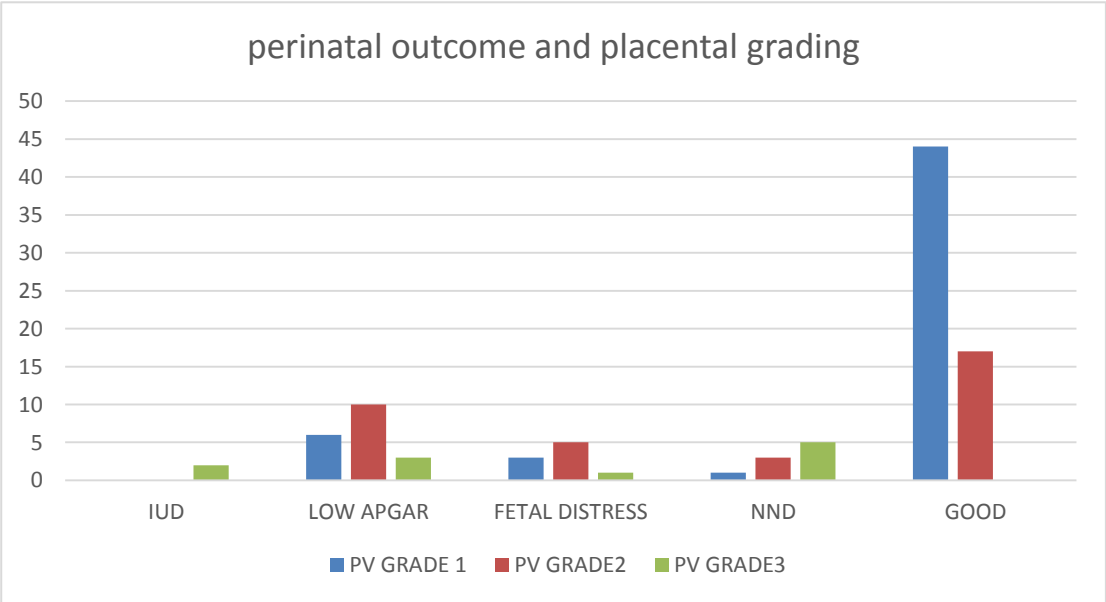
PERINATAL OUTCOME	PLACENTAL VOLUME GRADE 1	PLACENTAL VOLUME GRADE 2	PLACENTAL VOLUME GRADE 3	%
ADVERSE	10	18	11	39%
GOOD	44	17	-	61%

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

In patients with grade 2 placental volume, both good and adverse outcome of the baby was equal. In patients with 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



In patients with grade 1 placental volume the outcome of fetus was good.In this group babies showed low APGAR at birth and 3 babies suffers fetal distress because of MSAF.Among this 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.The other babies recovered well.babies had good perinatal outcome without any morbidity and mortality.

In patients with grade 2 placental volume, the incidence of fetal distress and low apgar were more. Low APGAR noticed in 10 babies.The incidence of fetal distress with meconium aspiration was noticed in 5 babies.Among these babies 3 babies with meconium aspiration syndrome and 2 babies with poor APGAR totally 5 babies died even with good neonatal critical care.other babies recovered well.17 babies had no adverse outcome.moreover 3 babies died in early neonatal period due to sepsis.The overall good outcome of babies in grade 2 placental volume when considering those babies recovered from initial perinatal morbidity was which is lower than grade 1 placental volume.

In patients with grade 3 placental volume all babies had adverse outcome only.There was 2 IUD mainly due to severe IUGR and very low birth weight.3 babies born with low APGAR,1 baby with severe fetal distress due to meconium aspiration.all these babies died in the early neonatal period even with good intensive care unit after admission due to delayed complications like sepsis.The adverse outcome of the babies in in grade 3 placenta was 100%

On statistical analysis the following was observed

Placental volume	Good outcome	Adverse outcome	significance
Grade 1	81.48%	18.52%	.003
Grade 2	48.57%	51.43%	
Grade 3		100%	

The percentage of 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.

The average placental volume measured by usg and after delivery in IUGR group was

GESTATIONAL AGE	PV BY USG	PV AFTER DELIVERY
32-34 WKS	325	330
35-37WKS	492	490
38-40WKS	586	588

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

The average placental volume in normal group before and after delivery was

GESTATIONAL AGE	PLACENTAL VOLUME BY USG	PLACENTAL VOLUME AFTER DELIVERY
32-34 WKS	490	496
35-37WKS	594	600
38-40 WKS	680	676

In these group also both measurements were correlated well.

This was comparable with the study by Humberto Azprurua,Edmund F who noticed significant correlation between placental volume measured by usg and placental volume measured after delivery.

SUMMARY

This is a prospective analytical study.

100 patients with singleton uncomplicated pregnancy selected as control .100 patients with singleton IUGR pregnancy were included in this study inorder to match the variables in IUGR group.

85% of IUGR patients was in 32 to 37 weeks of GA.The common maternal age group is 31 to 36 years.

General and obstetric examination done for all the patients.By doing USG fetal biometry including BPD,FL,AC,EFW,AFI were measured.Doppler study of umbilical artery and middle cerebral artery done .Cerebroplacental ratio calculated from resistance index of middle cerebral and umbilical artery for all the patients.

Placental localization done.placental volume and placental bed vascularity measured.

All patients followed uptodelivery.The mode of delivery and the indication for LSCS noted.Birthweight of the baby noted.All perinatal morbidities like MSAF,low APGAR was noted.All babies followed uptodischarge.placental volume again measured after delivery.The placental volume measured by ultrasound was compared with that measured after delivery.The results compared with normal pregnancy.The

average placental volume in normal pregnancy is 582.82cm3.Average placental volume in IUGR pregnancy was 400.91cm3.

This shows significant difference in placental volume between these group.On statistical analysis,this showed significant difference.p=.001(highly significant:Levenes T-Test)

The placental volume done by usg before delivery was compared that of measured after delivery.The average placental volume after delivery in IUGR group was 403.65cm3.The average placental volume measured after delivery in normal pregnancy was 592.20cm3.These did not shows much difference that of usg measurement before delivery.

In normal pregnancies placental bed vascularity is on range of 30-32 than IUGR(18-20)

VI was 5-6 higher in normal pregnancy than IUGR.It clearly shows that placenta of IUGR has fewer blood vessels and decreased blood flow and placental insufficiency.

The average birthweight of babies in grade 1 placental volume 1.99kgs and in grade 2 placental volume is 1.82kgs.These 2 didn’t show much difference.The average birth weight in grade 3 placental volume is 1.25kgs.This shows significant difference in average birth weight.

When the placental volume was compared with that of baby,in grade 1 placental volume,there was 81.48% good outcome and 18.52% adverse outcome and in grade 2 placental volume,the good outcome is 48.57% and the bad outcome raised to 51.43% .In grade 3 placental volume 100%adverse outcome only.This shows the placental volume had good correlation with fetal outcome.

This study shows positive correlation between the severity of IUGR and placental volume,placental bed vascularity.It predicts adverse perinatal outcome of the fetus.Hence this can be taken as one of the methods of predicting adverse neonatal outcome in IUGR.

CONCLUSION

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

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

Ultrasonography plays a major role in early diagnosis of IUGR.

Doppler ultrasonogram helps in identifying fetuses already in hypoxia and academia 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 complication.

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.

The quantitative assessment of placental volume and PBVI is an adjunctive modality for differentiation between normal and IUGR.The measurement of placental volume and PBVIin 1st and ^{2n}d trimester of pregnancy enables identification of impaired trophoblast invasion and helps in predicting the development of IUGR and preeclampsia.

BIBLIOGRAPHY

1. Bakketeig LS, Eik-nes SH, Jacobson et al. Randomized control trial of ultrasonographic screening in pregnancy. Lancet. 1984;207-211.

2. Mckenna D, Tharmaratnam S, Mahsud S et al, A randomized trial using ultrasound to identify the high risk fetus in low risk population. ObtstGynaecol 2003;101 :623-632.

3. Lin CC, Moawad AH, Rosenow P, et al. Acid- base characteristics of fetuses with intrauterine growth retardation during labour and delivery. Am J obstetGynaecol 1980; 137: 553.

4. Marana LR. Intrapartum fetal morbidity and mortality in intra uterine growth retarded infants. J Am osteopath Assoc 1980;80:101.

5. Morrisen I, Olsen J. W eight specific stillbirths and associated causes of death: An analysis of 765 stillbirths. Am J obstetGynaecol 1985; 152: 975.

6. Chamberlein PF, Manning FA, Morrisen I, et al. Ultrasound evaluation of amniotic fluid: the relationship of marginal and decreased amniotic fluid volume to perinatal outcome. Am J obstetGynaecol 1984; 150;245.

7. Hill DF. Physical growth and development after intra uterine growth retardation Reprod Med 1978 Nov; 21 (5):335-42.

8. Robertson CMT, Etches PC, Kyle M. Eight year school performance and growth of preterm, SGA infants: a comparative study with subjects matched for birth weight or for gestational age. J.Pediat 1990;116:19-26.

9. Kok JH, Lya Den Ouden a Verloove – Vanhorick SP et al. outcome of very preterm small for gestational age infants: the first nine years of life. Br J obstetGynaecol 1998; 105: 152-168.

10. Odegard R, vatten LJ, Nilsen ST, ET AL. Pre eclampsia and fetal growth. Obtetgynaecol 2000; 96:950-955.

11. Long PA, Abell DA, Beischer NA. Fetal growth retardation and preeclampsia. Br J obstetgynaecol. 1980;87: 13-18.

12. Sibai BM. Chronic hypertension in pregnancy. Obstetgynaecol 2002; 100:369-377.

13. Yasuda M, Tkakuwa K, Togunaga A, et al. prospective studies of the association between anticardiolipin antibody and outcome of pregnancy. Obstetgynaecol. 1995; 86:555-559.

14. Howley HE, Walker M, Rodger MA.A systematic review of the association between factor v leiden mutation or prothrombin gene variant and intrauterine growth restriction. Am J obstetgynaecol. 2005; 192:694-708.

15. Kramer MS. Intra uterine growth and gestational duration determinants. Paediatriccs. 1987;80:502-511.

16. Cliver SP, Goldenberg RL, Cutter GR et al. The effects of cigarette smoking on neonatal anthropometric measurements. Ostetgynaecol. 199; 85:625-630.

17. Cnattingius S, Mills JL, Jonathan Y, et al. The paradoxic effects of smoking in pre eclamptic pregnancies : Smoking reduces the incidence but increases the rate of perinatal mortality, abruption placentae and intrauterine growth restriction. Am J obstetgynaecol. 1997; 177: 156-161.

18. Windham GC, fenster L, Hopskins B, et al. The association of moderate maternal and paternal alcohol consumption with birth weight and gestational age. Epidemiology. 1995; 6:591-597.

19. Maisonet M, Correa A, Misra D, et al. A review of literature on the effects of ambient air pollution on fetal growth. Environ Res. 2004; 95: 106-115.

20. Liu S, Krewski D, Shi Y, et al. Association between gaseous ambient air pollutants and adverse pregnancy outcomes in Vancouvr, Canada. Enviroment health perspect. 2003;111:1773-1778.

21. Bagadosingh RO, Lynch L, Deren O, et al. First trimester growth restriction and fetal aneuploidy: the effect of type of aneuploidy and gestational age. Am J obstetgynaecol. 197; 17:976-980.

22. Khoury MJ Erickson JD, Cordero JF. Congenital malformations and intra uterine growth retardation: a population study. Paediatrics. 1988; 82: 83-90.

23. Lieff S, Offenbaher S, Boggess KA, et al. Maternal periodontitis and prematurity. Part I: obstetric outcome of prematurity and growth restriction. Ann periodontal. 2001;164-174.

24. Guenwald P. Growth of the human fetus: II Abnormal growth in twins an infants of mothers with Diabetes, Hypertension, or Isoimmunisation. Am J obstetgynaecol. 1966;94:1120-1132.

25. Krebs C, Macara LM, Leiser R, et al. Intra uterine growth restriction with absent end diastolic flow velocity in the umbilical artery is associated with Maldevelopment of the placental terminal villous tree. Am J obstetgynaecol. 1996; 175: 1534-1542.

26. TodrossT, Sciarrone A, Piccoli E, et al. Umbilical Doppler wave forms and placental villous angiogenesis in pregnancies complicated by intra uterine growth restriction. 1999; 93:499-503.

27. Cetin I. Placental transfer of amino acids in normal and growth restricted pregnancies. Eur J obstetgynaecolRepord Biol. 2003; 110 (Suppl): S50- S54.

28. Jansson T, Ylven K, Wennergren M, et al. Glucose transport and system A activity in syncytiotrophoblast microvillus and basal plasma membranes in intrauterine growth restriction. Placenta. 2002;23: 392-399.

29. Naeye RL. Disorders of the placenta, Fetus, and neonate: Diagnosis and clinical significance. St Louis : Mosby year book: 1992:132.

30. Raio L, Ghezzi F, Cromi A, et al. The thick heterogenous (jellylike) placenta: a strong predictor of adverse pregnancy outcome. Pregnatdiagn. 2004; 24: 182-188.

31. Salafia CM, Ernst LM, Pezzulo JC, et al. The very low birth weight maternal complications leading to preterm birth, placental lesions, and intra uterine growth. Am J obstetgynaecol. 19977; 12:106-110.

32. Benrischke K, Kaufmann P. Placenta shape aberrations. In: pathology of the human placenta. 4 th ed. Newyork springer, 1995;401-404.

33. Alherne D, Dunnill RA, et al, The volumetric growth of human placenta : a longitudinal ultrasonic study; Am J obstetgynaecol; 1997;127;657-661.

34. Griesson RT, Ogston SA, Patel NB, Christie AD, et al : Growth of total intra uterine intra amniotic and placental volume in normal singleton pregnancy measured by ultrasound: B J obstetgynaecol 1985; 92: 46-53.

35. Wallace JM, Aitken RP, Milne JS etal: Nutritionally mediated placental growth restriction in the growing adolescent/; consequences for the fetuses : 2004; 46; 765-771.

36. Thame M, Osmond C, Wilkis R, Benette FI and Forrester TE et al. Fetal growth is directly related to maternal anthropometry and placental volume; 98; 2: 279-283.

37. Clappe JF, III, Schmidt S, PAranjape A, et al. Maternal Insulin like growth factor I levels reflect placental mass and neonatal fat mass. Am J obstetgynaecol 2004;190:730-736.

38. Laviola L Perrni S, Belsanti G, et al. Intra uterine growth restriction in humans is associated with abnormalities in placental insulin like growth factor signaling. Endocrinology. 2005; 146: 1498-1505.

39. Lepercq J, Guerre- Millo M, Andre J, et al. Leptin: a potential marker or placental insufficiency. Gynaecolobstet invest. 2003; 55:151-155.

40. Cetin G, Alvino F et al, Glucose transport and system A activity in syncytiotrophoblast micro villous and basal plasma membranes in intra uterine growth restriction. Placenta; 2009,; 23:392-399.

41. Cetin J M, Foidart M, Miazzo RE et al. Differential effect of preterm birth and small gestational age on cognitive and motor development. 2004;76;F78-F81.

42. Marcus Rijken A, Wiliams E Moroski, SupornKiricharo et al. Effect of malaria on placental volume using 3 dimensional ultrasound obstetgynaecol 2012; 59:273-278.

43. Imdal R, Aame ER, Yakob, Mohammed Yawar et al, Arterial Doppler and anthropometry with IUGR and stillbirth. Am J obstetgynaecol, 2011; 19:268-273.

44. Hata T, Tanaka H, Noguchi J, Hata K et al. Two dimensional evaluation of placental morphology; Am J obstetgynaecol; 2011;123;167-172.

45. Hafner E, Philipp T, Schuchter K, Dillinger paller, Phillip K, Baure P et al. Second trimester measurement of placental volume by three dimensional ultrasound to predict small for gestational age infants. Am J obstetgynaecol. 2002;234;1469-1474.

46. Ferrazi E, Bulfamente G, Mezzopane R, Barbera A, Ghidini A, pardi G et al. Uterine Doppler velocimetry and placental hypoxic.ischemic lesion in pregnancies with fetal growth restriction. Am J obstetgynaecol. 19998;20;389-394.

47. Noguchi J, Tanaka H, Hata T et al. Placental vascular sonobiopsy using three dimensional ultrasound in normal and intra uterine growth restriction. Am J obstetgynaecol. 2009;111,1267-1274.

48. Jang, Dong Gyu, Yun sung, Lee et al. Perinatal outcome with maternal characteristics with absent end diastolic flow velocity. Am J obstetgynaecol 2011;23;197-178.

49. Hafner E, Philipp T, Schuchter K et al. Second trimester placental volume in three dimensional ultrasound to predict small for gestational age infants; ultrasound in obstetrics, Am J obstetgynaecol 1998;12;97-102.

50. Giuseppe, Rizzo, Alessandra Capponi, Ottaliacavichion, Marianne Vendole,DominciceArdini et al. First trimester uterine doppler and their three dimensional ultrasonogram placental volume calculation in predicting pre eclampsia. 200845:234-245.

51. Christiane Krebs, Lena M Marcerea, Rudolf Leisser et al. intra uterine growth retrictionwith absent end diastolic flow velocity in umbilical artery in association with maldevelopment of placental terminal villous tree. Am J obstetgynaecol. 1998;175:1534-1542.

52. Humberto Azpurua, Edmund F. Funai, Luisa M Coralluzi, Leo F Dolersty, Isaac E Sasson, MerunKlimen, Hervey J Klimen et al. Determination of placental weight using two dimensional sonography and volumetric mathematical modeling. Am J obstetgynaecol 2011. 176:1567-1576.

53. Humberto Azpurua, Edmund F. Funai, Luisa M Coralluzi, Leo F Dolersty, Isaac E Sasson, MerunKlimen, Harvey J Kilmen et al. Determination of placental weight using two dimensional sonography and volumetric mathematical modeling. Am J obstetgynaecol 2011.. 176:1567-1576.

54. Campbell, Deuhurst et al, Ultrasound measurement of fetal head to abdomen circumference ratio in assessment of growth retardation. 1971.Br J obstetgynaecol. 84:165-167.

55. Ott WJ. Sonographic diagnosis of intrauterine growth restriction. Clinobstetgynaecol. 1997:40:787-767.

56. Chervenac FA, Romero R, Berkowitz RL et al. The use of sonographic estimated fetal weight in the prediction of intrauterine growth restriction. Am J perinatol. 1984;1:298.

57. Arias F. Accuracy of the middle cerebral to umbilical artery resistance index ratio in the prediction of neonatal outcome in patients at high risk for fetal and neonatal complications. Am J obstetgynaecol. 1994;171;1541-1545.

58. Bagado –sing RO, Kovanchi E, Jeffers A, et al. the Doppler cerebro placental ratio and perinaeal outcome in intrauterine growth restriction. Am J obstet gynaecol.1999;180;750-756.

59. Taj Mohammed, Asmat Ara et al. Risk factors of intrauterine growth restriction. Am J obstetgynaecol. 2010;56;156-161.

60. Fikree et al. the differential neonatal mortality of intrauterine growth retardation syndrome. Am J obstetgynaecol 1990;163:151-157.

61. Thompson, Cliver SP, Neggres Y et al. the relationship between maternal characteristics and fetal and neonatal anthropometric measurements in women delivering at term. A summary. Acta obstetgynaecol 197;76:165-168.

62. Paterson RM, Gibbs, Woods et al. the prevalence of recurrent intrauterine growth restriction in pre eclampsia. Am J obstetgynaol 1986;47:23-134.

63. De paula CF, Auano R, Campos JA, Zugaib M et al. placental volume by three dimensional ultrasound in normal pregnancies from 12-40 weeks. J ultrasound med. 2008;27:1583-1590.

64. Muhammad, Asmat Area et al. Maternal factors associated with intrauterine growth restriction. 2010;22(4).

65. Jamel M Khan et al. Maternal factors associated with low birth weight. J Coll physicians surgpak 2003;13;256-258.

66. Ferraz et al . Maternal multi variable factors with intrauterine growth restriction. Am J obstetgynaecol. 2006;34:178-182.

67. Duldarenicz L, Kaluzewski B et al. Umbilical and middle cerebral artery blood velocimetry and relation ship with placental volume. 2000;6(2):266-270.

68. Investigate of three-dimensional power Doppler ultrasound used in placental perfusion assessment in pre-eclampsia pregnancies.Ma XQ, et al. Zhonghua Fu Chan KeZaZhi. 2009.

69. Alteration of vascularization in preeclamptic placentas measured by three-dimensional power Doppler ultrasound.Chen CY, et al. J Matern Fetal Neonatal Med. 2013.

70. Evaluation of the predictive value of placental vascularisation indices derived from 3-Dimensional power Doppler whole placental volume scanning for prediction of pre-eclampsia: A systematic review and meta-analysis.

ANNEXURE I

PROFORMA

DATE:

NAME :

AGE:

IP NO:

SOCIOECONOMIC CLASS:

RELIGION:

OCCUPATION :

ADDRESS & CONTACT NO:

DOA:

OBSTETRIC CODE:

D.O. DELIVERY:

DOD:

History of present illness :

Menstrual history:

Regular Irregular

LMP:

EDD:

Marital History:

Married Since :

Consanguinity:

H/o Infertility:

Obstetric History:

Previous Obstetric History:

Details of Outcome

Personal History:

Details of Outcome

Personal History:

Smoking –

Alcohol-

Diet –

Past Medical History:

Diabetes:

Chronic Hypertension:

Heart Disease:

Others:

Drug Intake:

Others :

Past Surgical History:

Present Pregnancy:

I Trimester:

Hyperemesis

Fever

Radiation Exposure

Medications

Pain Abdomen

II Trimester:

Date of quickening

Bleeding PV

GDM

Pre-eclampsia

III Trimester:

Bleeding Pv

GDM

Pre eclampsia

GENERAL EXAMINATION

Height :

Weight :

BMI:

Built: Thin : Average : Obese:

Pallor/ jaundice/clubbing/pedal edema/cyanosis/lymphadenopathy

Pulse : RR:

Blood Pressure:

Cardiovascular System:

Respiratory System:

Thyroid : Breast:

OBSTETRIC EXAMINATION

Fundal height:

FH:

Liquor adequacy:

PELVIC EXAMINATION:

Investigations:

Urine: Albumin

Sugar

Blood :

Hemoglobin :

Blood Sugar:

Urea:

S. Creatinine :

Blood groupingand typing :

HIV: VDRL: HBASg:

Ultra Sound:

BPD			
AC			
FL			
AFI			
EFW			
GA			
PLACENTA			

DOPPLER STUDY:

UMBILICAL ARTERY RI:

MIDDLE CEREBRAL ARTERY RI:

CPR:

PLACENTAL VOLUME:

PLACENTAL BED VASCUARITY:

DELIVERY:

VAGINAL : SPONTANEOUS:

INDUCED:

LSCS : ELECTIVE/EMERGENCY:

OUTCOME:

IUD/STILL BORN:

BIRTH WEIGHT: APGAR:

LIQUOR: CLEAR/MECONIUM:

PLACENTAL VOLUME:

ANNEXURE II

CONSENT FORM

I agree to participate in the study entitled **"COMPARATIVE STUDY OF USG ASSESMENT OF PLACENTAL VOLUME AND PLACENTAL BED VASCULARITY IN NORMAL PREGNANCY AND IUGR"** I confirm that I have been told about this study in my mother tongue and have had the opportunity to clarify my doubts.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reasons and without affecting my benifits.

I agree not to restrict the use of any data or results that arise from this study.

Name of the participant :

Sign / Thumb print :

Name of the investigator : Dr. A. KANIMOZHI

Sign of Investigator :

ANNEXURE III

ABBREVIATIONS

- LSCS- Lower Segment Caesarean Section.
- HSV – Herpes Simplex Virus
- HIV – Human Immunodeficiency Virus
- IUD – Intra Uterine Death
- BMI – Body Mass Index
- ACOG - American College of Obstetricians and Gynecologists
- LBW – Low birth weight
- PV – Placental Volume
- PBVI – Placental Bed Vascularity Index

ANNEXURE IV

ETHICAL COMMITTEE APPROVAL FORM

GOVERNMENT STANLEY MEDICAL COLLEGE& HOSPITAL,CHENNAI -01
INSTITUTIONAL ETHICS COMMITTEE

Title of the Work : COMPARATIVE STUDY OF USG ASSESMENT OF PLACENTAL VOLUME AND PLACENTAL BED VASCULARITY IN NORMAL PREGNANCY AND IUGR.

Principal Investigator : DR.A.KANIMOZHI,

Designation : MD OG,

Department : Department of O&G,
Govt. Stanley Medical College.

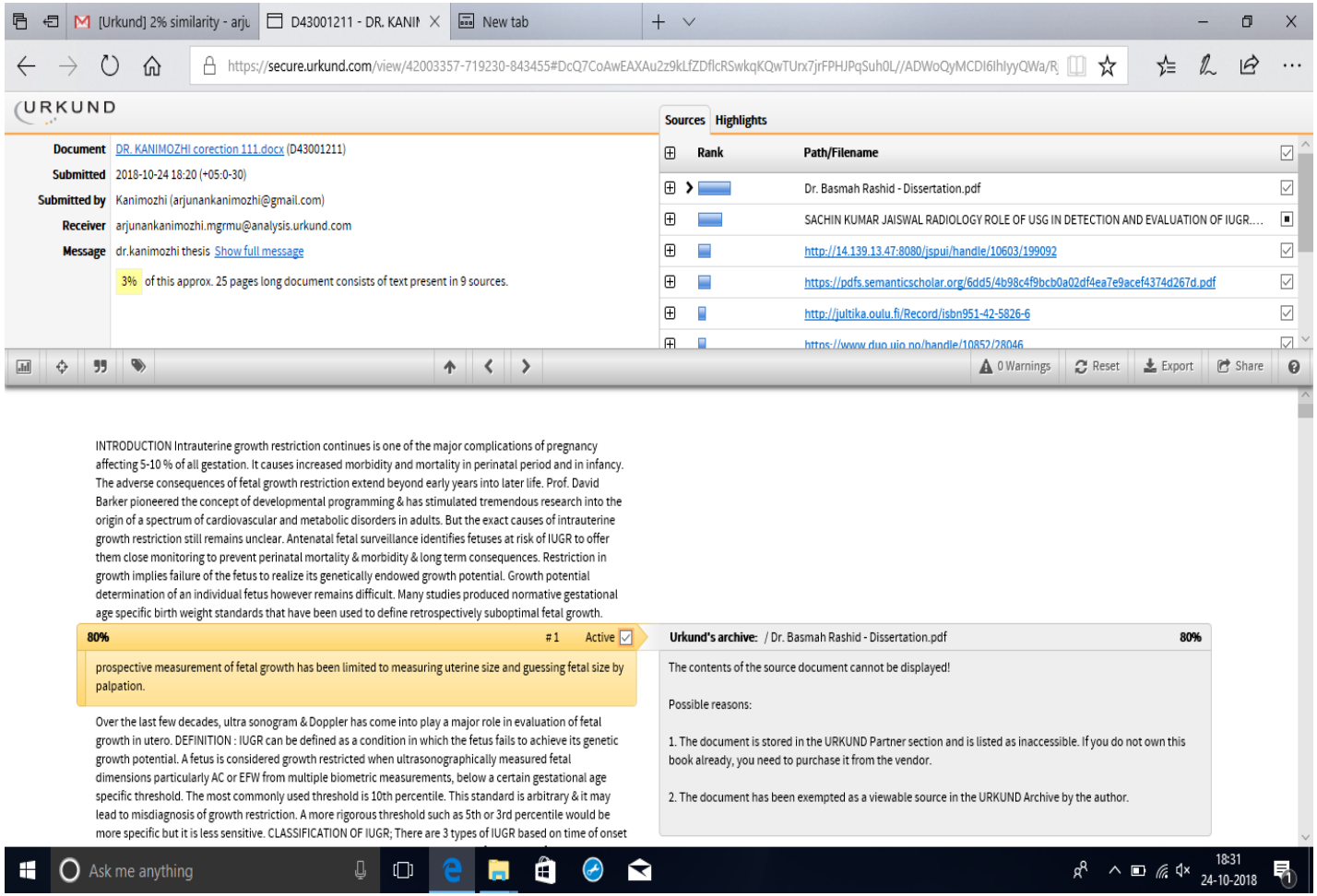
The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 21.12.2017 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY, 22/12/17
IEC, SMC, CHENNAI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INTRODUCTION Intrauterine growth restriction continues is one of the major complications of pregnancy affecting 5-10 % of all gestation. It causes increased morbidity and mortality in perinatal period and in infancy. The adverse consequences of fetal growth restriction extend beyond early years into later life. Prof. David Barker pioneered the concept of developmental programming & has stimulated tremendous research into the origin of a spectrum of cardiovascular and metabolic disorders in adults. But the exact causes of intrauterine growth restriction still remains unclear. Antenatal fetal surveillance identifies fetuses at risk of IUGR to offer them close monitoring to prevent perinatal mortality & morbidity & long term consequences. Restriction in growth implies failure of the fetus to realize its genetically endowed growth potential. Growth potential determination of an individual fetus however remains difficult. Many studies produced normative gestational age specific birth weight standards that have been used to define retrospectively suboptimal fetal growth.
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prospective measurement of fetal growth 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. 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
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24-10-2018

PLAGRISM V

MASTER CHART- NORMAL PREGNANCIES

Sl.No	Name	Age	IP No	Obst Code	G/A(LMP)	GA (USG)	PV (USG)	PV (DELIVERY)	PBVI	DELIVERY	BW	OUTCOME
1	ZEENTH	17	9748	Primi	33-34	34-35	532	542	31.24	VAGINAL	2.4	LOWAPG
2	SANGEETHA	19	9695	Primi	34-35	34-35	580	590	30.14	VAGINAL	2.6	GOOD
3	VALLI	21	9731	Primi	34-35	33-34	590	580	32.34	LSCS	2.7	GOOD
4	SHALINI	22	9723	Primi	35-36	36-37	565	570	33.1	LSCS	2.5	RESP DISTRESS
5	VINITHA	19	9738	G2A1	34-35	35-36	568	550	30.12	VAGINAL	2.4	GOOD
6	SATHYA	20	9648	Primi	35-36	35-36	540	535	31.21	VAGINAL	2.5	GOOD
7	JOTHI	22	9713	G2P1L1	34-35	35-36	560	575	29.21	LSCS	2.6	GOOD
8	KAYATHIZHI	27	9717	G2P1L1	35-36	36-37	572	585	29.34	VAGINAL	2.7	GOOD
9	SADHANA	21	9683	G3P1P1L1A1	35-36	34-35	565	580	31.34	LSCS	2.9	GOOD
10	SUBHALAKSHMI	25	9798	Primi	35-36	36-37	555	570	33.14	VAGINAL	2.6	GOOD
11	RAMYA	22	9794	G2A1	34-35	34-35	545	560	29.24	VAGINAL	2.5	RESP DISTRESS
12	RADHA	25	9708	G2P1L1	36-37	36-37	585	592	28.91	VAGINAL	2.7	GOOD
13	DURGA	21	9729	Primi	35-36	36-37	590	585	29.14	LSCS	2.6	GOOD
14	ESTHER RANI	24	9349	Primi	34-35	35-36	540	555	28.34	VAGINAL	2.9	GOOD
15	GOMATHI	23	9809	G2A1	34-35	34-35	552	565	31.14	LSCS	2.8	GOOD
16	BHARANI	23	9801	G2P1L1	35-36	36-37	562	582	30.32	VAGINAL	2.7	GOOD
17	NAVEEN	28	9810	Primi	34-35	35-36	515	525	31.24	LSCS	2.4	LOWAPG
18	NALINI	29	9724	G3PIL2	35-36	34-35	546	555	33.14	VAGINAL	2.7	GOOD
19	DIVI	31	9819	G3P2L2	36-37	35-36	546	585	30.64	VAGINAL	2.9	GOOD
20	GAYATHIRI	30	9807	Primi	35-36	36-37	550	572	32.74	LSCS	2.8	GOOD
21	GEETHA	29	9354	G3P1L1A1	34-35	34-35	536	545	31.46	VAGINAL	2.3	RESP DISTRESS
22	NAGENI	27	9804	G2P1L1	35-36	34-35	542	565	32.64	VAGINAL	2.7	GOOD
23	ASHWINI	28	9814	G2P1L1	35-36	36-37	570	580	30.04	LSCS	2.8	GOOD

24	JAYASUDHA	26	9803	Primi	36-37	35-36	575	590	30.68	VAGINAL	2.5	MSAF
25	GAWRI	30	9797	G3P1L1A1	35-36	34-35	580	595	30.72	VAGINAL	2.7	GOOD
26	FARODJA	31	9730	G3P2L0	34-35	33-34	530	555	31.84	VAGINAL	2.6	GOOD
27	REVATHY	29	9743	G2A1	35-36	35-36	545	560	31.82	LSCS	2.5	GOOD
28	NADHIYA	31	9791	Primi	35-36	36-37	550	560	28.74	VAGINAL	2.6	GOOD
29	SILAMBARASI	34	9793	G3P2L2	34-35	33-34	525	540	28.72	LSCS	2.8	GOOD
30	NISHATHINI	31	9843	Primi	34-35	35-36	568	580	28.64	LSCS	2.7	GOOD
31	DIANA	33	9827	G2A1	36-37	35-36	575	570	28.61	LSCS	2.8	GOOD
32	SHIFANA	29	9836	G2A1	34-35	34-35	536	545	28.2	VAGINAL	2.5	GOOD
33	MAHALAKSHMI	31	12080	G2A1	34-35	35-36	535	542	29.73	LSCS	2.7	GOOD
34	SUDHA	31	12255	G3P2L2	35-36	36-37	542	555	29.73	VAGINAL	2.9	GOOD
35	SUMADHI	30	12261	Primi	34-35	35-36	515	525	30.14	VAGINAL	2.2	RESP DISTRESS
36	MEGALA	32	16678	G3P1L1A1	35-36	35-36	550	560	30.16	LSCS	2.6	GOOD
37	POORKODI	29	12077	G2P1L1	36-37	36-37	568	580	31.18	LSCS	2.9	GOOD
38	RANJITHA	28	12104	G3P1L1A1	36-37	36-37	572	585	30.68	VAGINAL	2.8	GOOD
39	RENUKA	31	12218	G2PILO	34-35	35-36	525	540	31.72	LSCS	2.7	GOOD
40	PREMA	32	12253	Primi	36-37	34-35	584	575	30.62	LSCS	2.7	GOOD
41	DEEPA	30	11942	G2P1L1	35-36	34-35	570	580	30.74	VAGINAL	2.6	GOOD
42	SABIYA	31	11472	Primi	34-35	36-37	538	545	32.7	VAGINAL	2.5	GOOD
43	MANJLA	34	11304	G3P1L1A1	35-36	33-34	574	590	32.62	LSCS	2.9	GOOD
44	MITHULA	33	11460	G3A2	35-36	34-35	563	570	31.84	LSCS	2.7	GOOD
45	BADHOUR NISHA	31	11470	G2P1L1	34-35	35-36	555	560	31.82	VAGINAL	2.8	GOOD
46	SATHIYAPRIYA	30	11355	G2A1	36-37	35-36	585	600	30.12	VAGINAL	2.9	GOOD
47	DEVAKRIBA	32	11453	G2P1L1	34-35	33-34	592	612	29.14	VAGINAL	3	MSAF
48	SHABA	33	11131	Primi	34-35	35-36	530	545	29.12	LSCS	2.6	GOOD
49	BARANI	29	11411	G2PILO	35-36	34-35	525	540	30.13	VAGINAL	2.4	GOOD
50	SEETHA	31	11488	Primi	33-34	34-35	548	565	31.16	VAGINAL	2.6	MSAF

51	VIJAYALAKSHMI	19	11433	Primi	36-37	36-37	605	640	31.13	LSCS	3.1	GOOD
52	VINODHIYA	20	11465	Primi	35-36	36-37	595	580	32.12	LSCS	2.9	GOOD
53	PRIYALAKSHMI	21	11417	Primi	36-37	36-37	580	570	33.12	VAGINAL	2.8	GOOD
54	SOWMEYA	22	11374	Primi	37-38	36-37	595	580	30.14	LSCS	2.9	GOOD
55	VALARMATHY	23	11471	G2A1	36-37	35-36	602	615	30.12	VAGINAL	3	GOOD
56	RAJITHA	25	11438	G2P1L1	37-38	37-37	600	620	29.12	VAGINAL	3.1	GOOD
57	FAYAZ	26	11434	Primi	36-37	35-36	592	610	29.14	LSCS	3	GOOD
58	LAKSHMI	25	11454	G2P1L1	36-37	35-36	584	580	30.16	VAGINAL	2.9	GOOD
59	HEMALATHA	27	11446	G2P1L1	36-37	37-38	575	602	30.12	VAGINAL	3.3	GOOD
60	ELIZABATH	28	11036	G3P1L1A1	36-37	37-38	610	625	30.14	VAGINAL	3.5	MSAF
61	ZAINAB	31	11317	Primi	35-36	34-35	595	585	30.16	VAGINAL	2.9	MSAF
62	DIVYA	29	11357	G2P1L1	36-37	35-36	582	590	31.12	VAGINAL	3	GOOD
63	VAISHALI	29	11240	G3P1L1A1	35-36	36-37	590	594	31.24	LSCS	2.8	GOOD
64	SIRUGANI	31	11239	G3A2	36-37	35-36	588	555	32.12	VAGINAL	3.2	GOOD
65	SUDARI	27	12399	G3P1L1A1	36-37	37-38	586	590	30.18	VAGINAL	3.2	GOOD
66	RAMYA	28	12338	G3P2L2	37-38	36-37	590	600	29.24	VAGINAL	3.3	GOOD
67	GEETHA	31	12396	Primi	37-38	38-39	598	615	29.2	LSCS	2.9	GOOD
68	DEVI	30	12259	Primi	36-37	37-38	565	585	31.12		3.2	MSAF
69	ANBUKARASI	32	11497	G2P1L1	36-37	35-36	575	590	30.13	VAGINAL	2.9	GOOD
70	ANJANI	32	11325	Primi	37-38	38-39	590	610	29.13	LSCS	3.4	MSAF
71	JANAKI	35	12300	G3P1L1A1	36-37	38-39	575	600	29.14	VAGINAL	3.1	GOOD
72	GAYATHIRI	34	12331	G2PILO	37-38	36-37	610	625	29.52	LSCS	3.2	GOOD
73	NIRMALA	34	12262	G3A2	36-37	37-38	587	602	30.54	LSCS	3.1	GOOD
74	LEAVASI	33	12298	G3P2L1	37-38	36-37	585	590	29.29	VAGINAL	3.2	GOOD
75	RANJAN	18	12980	Primi	36-37	37-38	625	633	30.31	VAGINAL	3.3	GOOD
76	NILO	20	12188	Primi	38-39	37-38	625	640	29.3	LSCS	3.1	GOOD
77	USHA	19	12348	G2A1	38-39	39-40	635	650	29.13	VAGINAL	3.2	LOWAPG

78	RAMYA	23	12223	Primi	37-39	38-39	604	615	30.14	VAGINAL	3.1	GOOD
79	PREMA	25	12268	Primi	37-38	36-37	610	625	32.16	VAGINAL	3.3	GOOD
80	DHANA	32	12311	G3A2	37-38	38-39	598	615	30	VAGINAL	3.1	GOOD
81	ANITHA	23	12067	G2P1L1	37-38	37-38	625	640	31	LSCS	3.2	GOOD
82	VANI	37	12330	G3P1L1A1	38-39	38-39	640	625	30.13	VAGINAL	2.9	MSAF
83	MEGALA	28	12354	Primi	38-39	37-38	625	610	30.16	LSCS	3	MSAF
84	AFRIA	31	12411	Primi	37-38	37-38	590	610	30.18	VAGINAL	3.1	GOOD
85	AMUL RANGANAYAG	30	12406	G2P1L1	38-39	38-39	610	600	31.28	VAGINAL	2.9	GOOD
86	USHA	29	12632	G2A1	37-38	36-37	610	625	32.38	VAGINAL	3.1	LOWAPG
87	PADMINI	29	12385	Primi	37-38	37-38	588	602	33.48	LSCS	3.2	GOOD
88	KALAYARASI	32	12410	G2A1	37-38	36-37	595	615	30.32	VAGINAL	3.2	LOWAPG
89	RASIDHA	31	12375	G3P2L2	38-39	37-38	625	610	30.52	VAGINAL	3.1	LOWAPG
90	UMADEVI	32	12388	G2P2L2	38-39	37-38	625	640	30.54	VAGINAL	3.5	GOOD
91	RADHA	33	12408	G3P1L1A1	37-38	38-39	610	625	29.62	LSCS	3.1	GOOD
92	MANIPRIYA	32	12296	G3A2	38-39	37-38	620	635	30.64	VAGINAL	3.25	MSAF
93	MABUNISHA	35	12344	G2P1L1	37-38	36-37	600	625	31.72	VAGINAL	3.1	GOOD
94	PREAMILA	36	12356	G3P1L1A1	38-39	38-39	626	645	30.45	VAGINAL	3.25	GOOD
95	GIRIJA	36	12288	G2P1L1	39-40	38-39	680	695	30.42	LSCS	3.6	GOOD
96	KAMALI	34	12584	G2A1	38-39	38-39	676	695	30.4	VAGINAL	3.4	GOOD
97	REVATHY	31	12562	Primi	39-40	38-39	680	665	30.43	LSCS	3.6	GOOD
98	MANO RANJITHA	34	12572	Primi	38-39	38-39	685	680	31.2	LSCS	3.75	MSAF
99	NAYAGI	30	12494	Primi	39-40	38-39	680	665	31.35	LSCS	3.2	GOOD
100	MUBEEN	34	12483	G3A2	39-40	39-40	680	675	32.2	LSCS	3.3	GOOD

MASTER CHART- IUGR PREGNANCIES																	
Sl.No	Name	Age	IP NO	Obst Code	G/A(LMP)	GA (USG)	MCA (RI)	UA (RI)	CPR	PV (USG)	placental Maturity	PV (DELIVER	PBVI	DELIVERY	IND	BW	OUTCOME
1	PREMKUMARI	19	9524	Primi	33-34	25-26	0.6	0.7	<1	184(III)	(III)	174	22.22	LSCS	OLIGO	1.1	NND
2	VINODHINI	22	9637	G2A1	34-35	28-29	0.65	0.74	<1	195(III)	(III)	190	20.12	LSCS	FAIL IND	1.2	LOWAPG
3	RAJESHWARI	21	9554	G2PILI	34-35	27-28	0.64	0.76	<1	174(III)	(III)	168	16.4	VAGINAL		840gms	IUD
4	VIJAYALAKSHMI	21	9518	Primi	33-34	30-31	0.7	0.8	<1	190(III)	(III)	196	18.4	LSCS	OLIGO	1.6	LOWAPG
5	JAYAVEERA	19	9665	Primi	34-35	24-25	0.7	0.96	<1	160(III)	(III)	150	16.1	VAGINAL		850GMS	IUD
6	JAYALAKSHMI	19	9619	Primi	34-35	26-27	0.6	0.75	<1	185(III)	(III)	195	19.2	LSCS	FAIL IND	1.4	LOWAPG
7	NITHYA	23	9644	Primi	35-36	30-31	0.8	7	>1	325(II)	(II)	315	28.12	VAGINAL		1.7	GOOD
8	JANSIRANI	24	9646	G2A1	34-35	29-30	0.76	0.64	>1	340(III)	(III)	325	26.14	VAGINAL		1.9	GOOD
9	BHARANI	23	9657	G2PILI	34-35	32-31	0.7	0.8	<1	330(II)	(II)	325	26.13	LSCS	OLIGO	1.7	GOOD
10	PRASANA ROJA	26	9617	G2A1	34-35	32-31	0.7	0.68	>1	315 (II)	(II)	303	25.11	LSCS	BREECH	1.6	GOOD
11	SAMDHANI	25	9296	Primi	34-35	28-29	0.8	0.9	<1	324 (II)	(II)	335	20	LSCS	OLIGO	1.8	LOWAPG
12	RAJALAKSHMI	26	9518	G2PILO	34-35	31-32	0.7	0.82	<1	338(II)	(II)	330	21	LSCS	FAIL IND	1.4	LOWAPG
13	INDHUMATHI	24	9582	G2ILO	35-36	32-33	0.7	0.82	<1	345(II)	(II)	360	23.5	LSCS	FAIL IND	1.8	GOOD
14	YASODHA	25	9667	G2A1	36-37	31-32	0.9	0.8	>1	350 (II)	(II)	355	23.6	LSCS	BREECH	1.5	GOOD
15	SARANYA DEVI	21	9502	G3PILIAI	34-35	30-31	0.86	0.74	>1	364(I)	(I)	374	23.7	VAGINAL		1.5	GOOD
16	DEVI	22	9692	G3PILIAI	32-33	30-31	0.8	0.74	>1	346 (II)	(II)	340	20	LSCS	FAIL IND	1.9	LOWAPG
17	SHYAMALA	29	9661	Primi	34-35	32-33	0.8	0.72	>1	412(1)	(I)	398	28	LSCS	FAIL IND	2	LOWAPG
18	DHILAGA	26	8276	G3PIL2	35-36	32-31	0.9	0.82	>1	328 (II)	(II)	325	28.14	LSCS	OLIGO	2.1	GOOD
19	JAVAHARSARTHAN	28	9699	G4PILIA2	34-35	30-31	0.7	0.8	<1	315 (II)	(II)	324	29.3	LSCS	BREECH	1.9	LOWAPG
20	POORNIMA	31	9611	Primi	35-36	31-32	0.7	0.68	>1	320(II)	(II)	312	24.6	LSCS	OLIGO	1.8	GOOD
21	VAHITHA	23	9710	G2PIL1	34-35	32-31	0.8	0.9	<1	300 (II)	(II)	315	22.1	LSCS	BREECH	1.5	LOWAPG
22	SUMAYA	28	9695	Primi	34-35	32-33	0.7	0.68	>1	486 (I)	(I)	280	21.3	LSCS	FAIL IND	1.8	LOWAPG
23	BISWAJITH	21	9641	Primi	34-35	31-32	0.8	0.7	>1	315 (II)	(II)	495	24	LSCS	BREECH	2	GOOD
24	RADHA	31	9655	G5PIL1A2	35-36	31-32	0.8	0.7	>1	350 (II)	(II)	305	25.16	LSCS	FAIL IND	1.9	GOOD
25	GOMATHY	30	9630	Primi	35-36	35-36	0.8	0.74	>1	424(I)	(I)	412	26.12	LSCS	FAIL IND	2.1	GOOD
26	JAYANTHI	29	9512	G2PIL1	35-36	35-36	0.8	0.84	<1	268 (II)	(II)	250	26.34	LSCS	FAIL IND	2	GOOD
27	BHAVATHI	26	9577	Primi	34-35	34-35	0.8	0.76	>1	475 (I)	(I)	482	21	LSCS	OLIGO	1.8	LOWAPG
28	FARZANA	29	9676	Primi	34-35	34-35	0.7	0.84	<1	328 (II)	(II)	345	26.4	LSCS	FAIL IND	1.9	GOOD
29	RAMYA	32	9725	Primi	32-33	32-33	0.7	0.68	<1	384(I)	(I)	364	19	LSCS		1.4	NND

30	JEEVITHA	30	9588	G3P2LO	35-36	35-36	0.82	0.74	>1	480 (I)	(I)	500	23	VAGINAL	BREECH	2	GOOD
31	MANIBALA	34	9742	G2P1LO	33-34	33-34	0.84	0.76	>1	494(I)	(I)	520	23.14	LSCS	FAIL IND	2.1	GOOD
32	NAGALAKSHMI	33	9295	G4PILIA2	36-37	36-37	0.8	0.76	>1	502 (I)	(I)	535	24.26	LSCS	FAIL IND	2.3	GOOD
33	SHILPA	36	9447	Primi	33-34	33-34	0.76	0.84	<1	325 (II)	(II)	330	21	LSCS		2.1	LOWAPG
34	KALAIVANI	35	9714	G2A1	32-33	32-33	0.8	0.76	>1	326 (II)	(II)	330	22	VAGINAL	FAIL IND	1.8	LOWAPG
35	YAUVARANI	35	9679	Primi	34-35	34-35	0.7	0.96	<1	384(I)	(I)	334	17.4	LSCS	FAIL IND	1.4	NND
36	PENERDEVI	32	12209	G3PILIAI	33-34	33-34	0.8	0.76	>1	315 (II)	(II)	320	19.2	LSCS	FAIL IND	1.6	LOWAPG
37	NALINI	33	12245	G2A1	35-36	35-36	0.8	0.72	>1	512(I)	(I)	540	24.6	LSCS		2.1	GOOD
38	DEVI	33	12277	G2PILI	34-35	34-35	0.9	1.1	<1	270(II)	(II)	295	24.8	VAGINAL		2.2	GOOD
39	GAYATHIRI	35	11434	G3P2L2	34-35	34-35	0.8	0.96	>1	383 (I)	(I)	400	26	VAGINAL	OLIGO	2	GOOD
40	PRIYANKA	34	11900	G3PILIAI	34-35	34-35	0.9	0.86	>1	254 (II)	(II)	275	19.2	LSCS	FAIL IND	1.7	LOWAPG
41	JAYEEHIBE	32	11427	Primi	34-35	34-35	0.74	0.86	<1	325(II)	(II)	330	23	LSCS		2.2	GOOD
42	SARANYA DEVI	33	12219	G2A1	34-35	34-35	0.7	0.84	<1	320(II)	(II)	308	24.6	VAGINAL		2.1	GOOD
43	MALAR	36	12039	G2P1L1	34-35	34-35	0.7	0.72	<1	325 (II)	(II)	335	25.6	VAGINAL	CTG NR	1.7	GOOD
44	VIDHYA	35	12209	G3PILIAI	34-35	34-35	0.8	0.76	>1	366 (I)	(I)	350	25.2	LSCS	FAIL IND	1.9	GOOD
45	MAHASWARI	33	12245	G3A2	34-35	34-35	0.84	0.76	<1	386 (I)	(I)	395	25.24	LSCS	FAIL IND	2	GOOD
46	BHRATHI	34	12277	G2PILI	34-35	34-35	0.76	0.86	<1	284(II)	(II)	244	19.2	LSCS	FAIL IND	1.6	NND
47	MICHAEL	32	11432	Primi	35-36	35-36	0.7	0.86	<1	416 (I)	(I)	440	24.12	LSCS	BREECH	1.9	GOOD
48	SHANDHINI	30	12228	G2PILI	35-36	35-36	0.76	0.68	>1	505(I)	(I)	525	24.72	LSCS	BREECH	2.1	GOOD
49	PEIRAVAI	31	12189	G3P2LO	35-36	30-31	0.68	0.84	<1	288(II)	(II)	271	18.14	LSCS	CTG NR	1.7	LOWAPG
50	MALTHY	32	12165	G2PILI	34-35	32-33	0.82	0.76	>1	330 (II)	(II)	345	19.2	LSCS	CTG NR	1.8	LOWAPG
51	LAKSHMI	35	12173	Primi	35-36	32-33	0.96	0.73	>1	412(II)	(II)	418	28	VAGINAL		2.1	GOOD
52	KAMAL	36	12241	Primi	36-37	33-34	0.8	0.76	>1	492(I)	(I)	510	24	VAGINAL		2	GOOD
53	SASIKALA	21	12246	Primi	35-36	34-35	0.74	0.76	<1	186(III)	(III)	188	20.1	VAGINAL		1.2	NND
54	ISHWARYA	22	12260	Primi	36-37	30-31	0.8	0.96	<1	190 (III)	(III)	210	20.34	LSCS	CTG NR	1.3	MSAF
55	NAGALAKSHMI	20	11925	G2AI	36-37	33-34	0.8	0.76	>1	366 (II)	(II)	375	24.23	LSCS	FAIL IND	2.1	GOOD
56	VASANTHI	24	11105	G2PILI	35-36	33-34	0.72	0.86	<1	584(II)	(II)	590	25.2	LSCS	CTG NR	2.2	GOOD
57	ELIZBETH	27	12006	G3PILIAI	36-37	33-34	0.86	0.7	>1	590 (I)	(I)	605	26.34	VAGINAL		2	GOOD
58	JEEVA	29	12005	G3A2	36-37	34-35	0.96	0.72	>1	582 (I)	(I)	575	22.34	VAGINAL		2.3	GOOD
59	VEERAMMAL	28	12003	Primi	36-37	34-35	0.92	0.76	>1	586 (I)	(I)	575	23.34	LSCS	BREECH	1.9	GOOD
60	ARCHANA	24	11968	G2PILI	36-37	32-33	0.76	0.92	<1	544(I)	(I)	562	23.62	LSCS	CTG NR	2	GOOD
61	MARY	30	11974	G4PILIA2	36-37	33-34	0.8	0.76	>1	344 (II)	(II)	360	23.72	LSCS	FAIL IND	2.2	GOOD
62	ANBU DEVI	31	11727	Primi	37-38	33-34	0.78	0.96	<1	572 (I)	(I)	580	21.22	LSCS	FAIL IND	2.3	MSAF
63	JEEVITHA	22	11800	G2AI	35-36	31-32	0.9	0.8	<1	560(I)	(I)	590	23.14	VAGINAL		2	GOOD

64	MAGESWARI	28	11937	Primi	37-38	34-35	0.96	0.72	<1	586 (I)	(I)	600	23.16	VAGINAL		2.4	GOOD
65	GIRIJA	26	11980	Primi	37-38	32-33	0.86	0.73	>1	550 (I)	(I)	575	21.12	VAGINAL		2.3	MSAF
66	KAVITHA	32	11975	G2PILI	36-37	32-33	0.8	0.76	<1	550(I)	(I)	540	22.34	LSCS	BREECH	2.1	GOOD
67	JESIPHIRE	33	11932	Primi	36-37	31-32	0.76	0.84	<1	320 (I)	(I)	335	20.62	LSCS	FAIL IND	2	MSAF
68	JENIFER	35	14114	Primi	35-36	31-32	0.8	0.72	<1	335 (II)	(II)	340	23.46	LSCS	FAIL IND	2.1	GOOD
69	SUBITHA	33	11882	G2PILI	36-37	31-32	0.9	0.72	>1	520(I)	(I)	524	21.64	LSCS	CTG NR	1.9	MSAF
70	JENIFER	32	11932	G3A2	37-38	32-33	0.74	0.96	<1	540 (I)	(I)	550	21.2	LSCS	BREECH	1.8	GOOD
71	JAUANJI	31	11970	Primi	36-37	30-31	0.74	0.82	<1	474(II)	(II)	432	20.18	LSCS	FAIL IND	1.6	LOWAPG
72	PRIYANKA	32	11981	G3P2L2	37-38	30-31	0.88	0.78	>1	440(I)	(I)	450	21.32	VAGINAL		2.2	LOWAPG
73	SUDHA	36	11954	G2PILI	36-37	31-32	0.76	0.92	<1	446(I)	(I)	450	20.42	LSCS	FAIL IND	1.7	MSAF
74	JENIFER	35	11799	G2PILO	36-37	30-31	0.92	0.86	>1	394(II)	(II)	398	18.42	LSCS	CTG NR	1.2	NND
75	JAYANJI	33	11932	Primi	37-38	30-31	0.78	0.88	<1	415(I)	(I)	410	21.14	LSCS	CTG NR	1.4	MSAF
76	PRIYANKA	32	11882	G2PILI	36-37	29-30	0.74	0.88	<1	400(I)	(I)	412	22.12	LSCS	CTG NR	1.6	LOWAPG
77	NANDHINI	18	11774	Primi	37-38	30-31	0.8	0.92	<1	202 (III)	(III)	195	19.74	VAGINAL		1.3	NND
78	ASMA	20	11513	Primi	37-38	31-32	0.96	1.2	<1	198 (III)	(III)	210	20.12	VAGINAL		1.25	NND
79	PVEETHI	24	11784	G2A1	36-37	31-32	0.82	0.76	>1	420 (I)	(I)	430	20.14	LSCS	CTG NR	1.5	MSAF
80	GORETHY	24	11778	Primi	36-37	33-34	0.84	0.76	>1	430(I)	(I)	415	21.12	LSCS	CTG NR	1.7	MSAF
81	SHAHIN	28	11726	G2PILI	37-38	34-35	0.82	0.76	>1	465(I)	(I)	475	24.43	LSCS	CTG NR	1.8	GOOD
82	SURYAKAK	27	11728	G2AI	37-38	32-33	0.68	0.78	<1	322(I)	(I)	330	20.12	VAGINAL		1.5	MSAF
83	JAYASRI	29	11727	G3PILIAI	37-38	31-32	0.72	0.86	<1	410 (I)	(I)	420	21.34	LSCS	CTG NR	1.4	LOWAPG
84	DHIVYA	31	11767	G2PILI	36-37	34-35	0.82	0.84	>1	550(I)	(I)	560	24.6	LSCS	FAIL IND	2.2	GOOD
85	ASTALAKSHMI	31	11765	G2PILO	36-37	30-31	0.72	0.76	<1	340 (I)	(I)	350	21.12	LSCS	CTG NR	1.6	MSAF
86	USHA	29	11572	Primi	37-38	33-34	0.82	0.84	>1	490 (I)	(I)	500	24.13	LSCS	CTG NR	1.8	GOOD
87	THANMOZHI	32	11318	G3PILIAI	37-38	34-35	0.78	0.76	<1	484(I)	(I)	472	24.72	LSCS	CTG NR	1.9	GOOD
88	VANISRI	31	11508	Primi	37-38	34-35	0.86	0.86	>1	501(I)	(I)	496	26.72	LSCS	CTG NR	2	LOWAPG
89	SUBHA	36	11791	G2PILI	37-38	32-33	0.84	0.75	>1	475(I)	(I)	485	25.12	LSCS	FAIL IND	1.7	LOWAPG
90	SEETHA	35	11416	G4PILIA2	36-37	31-32	0.74	0.78	<1	476(I)	(I)	464	25.62	LSCS	FAIL IND	1.8	GOOD
91	NARMADHALAKSH	34	11318	Primi	36-37	33-34	0.68	0.88	<1	492(I)	(I)	480	25.64	LSCS	FAIL IND	1.9	GOOD
92	BHAVANI	33	11656	G4PI2L2A1	38-39	34-35	0.74	0.82	>1	524(I)	(I)	522	28.12	LSCS	CTG NR	2.2	GOOD
93	KALPANA	32	11572	G2PILI	37-38	35-36	0.86	0.76	>1	565 (I)	(I)	579	22.12	LSCS	BREECH	2.4	GOOD
94	PENAYAGAI	29	11667	G2PILO	37-38	33-34	0.86	0.72	>1	440 (I)	(I)	450	20.12	LSCS	CTG NR	1.4	LOWAPG
95	SARANYA DEVI	32	11675	Primi	38-39	30-31	0.64	0.88	>1	200(III)	(III)	214	20.14	VAGINAL		1.3	NND
96	NANDHINI	35	11615	Primi	38-39	34-35	0.86	0.74	>1	630 (I)	(I)	640	2.16	LSCS	CTG NR	2.1	GOOD
97	THARA	34	11666	G3P2L2	39-40	35-36	0.76	0.84	>1	644(I)	(I)	620	26.28	LSCS	CTG NR	2.3	MSAF
98	RANI	27	11353	G3PILOA1	39-40	35-36	0.74	0.88	>1	675 (I)	(I)	675	27.28	VAGINAL		2	MSAF
99	KAVITHA	28	11492	G2A1	38-39	36-37	0.88	0.72	>1	680 (I)	(I)	690	28.46	VAGINAL		2.5	GOOD
100	UMA	30	11135	Primi	39-40	35-36	0.82	0.76	>1	540(I)	(I)	575	28.44	LSCS	CTG NR	2.4	GOOD