THIRD TRIMESTER IUGR PREDICTORS AND ITS OBSTETRIC OUTCOME

BY

DR. SWAR SHAH

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SUMANDEEP VIDYAPEETH, PIPARIA, VADODARA

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UNDER THE GUIDANCE OF

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2015-2018

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Dear Dr. Swar,

The Sumandeep Vidyapeeth Institutional Ethics Committee (SV IEC) is in receipt of your above mentioned study document and as the research study classifies in the minimal risk category; as recommended by HRRP SBKS MI&RC. The SV IEC approves your study to be conducted in the presented form.

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Dr. Niraj Pandit
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STUDY COMPLETION CERTIFICATE

This is to certify that study entitled: "Third Trimester IUGR, predictors and its Obstetric Outcome" Research Project was done by **"Dr. Swar Shah"** (PG Student, Dept of Obstetrics & Gynecology, S.B.K.S MI & RC, Dhiraj Hospital, Piparia, Waghodia road, Vadodara-391760, Gujarat) and it was conducted to the satisfaction of the Sumandeep Vidyapeeth Institutional Ethics committee.

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I hereby declare that this dissertation/thesis entitled **"THIRD TRIMESTER IUGR, PREDICTORS AND ITS OBSTETRIC OUTCOME"** is a bonafide and genuine research work carried out by me under the guidance of Dr. Uday J. Patel, M.D.(Obstetrics and Gynaecology).

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Place: Piparia, Vadodara **Dr. Swar Shah**

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I hereby declare that Sumandeep Vidyapeeth, Piparia, Vadodara District, Gujarat have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purpose.

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Dr. Swar Shah

ABSTRACT

INTRODUCTION

Intrauterine growth restriction (IUGR) of the foetus is defined as the inability of a foetus to reach its genetically determined growth potential at a given gestational age that means the birth weight is below the 10th percentile or birth weight less than 2 standard deviations for that gestational age. IUGR is a major source of perinatal morbidity and mortality and this continues to pose a challenging problem for both the obstetrician and paediatrician.

MATERIALS AND METHOD

SOURCE OF DATA:

This prospective observational study was undertaken Dhiraj Hospital from 1stFebruary 2016 to 31st July 2017.

INCLUSION CRITERIA:

All singleton pregnant patients with vertex presentation (after 34 weeks of gestation) undergoing regular antenatal check-up (with accurate dates, which were substantiated by first trimester dating scan were enrolled) of which the cases were diagnosed of Late onset IUGR were taken and followed till delivery.

EXCLUSION CRITERIA:

Autoimmune disease

Eclampsia

Exposure to drugs, alcohol, nicotine abuse

Multiple pregnancy

Malpresentation

Constitutionally small babies

Congenital malformations

STUDY DESIGN:

The enrolment of women for this study was performed after having confirmed IUGR.

This was done by

- History
- Clinical examination
- Ultrasonography
- Clinical examination of newborn
- Pregnancy outcome record

RESULT:

The prevalence of Late onset IUGR diagnosed in our institution was 2.44%. Majority of women (54.84 %) were in the age group 25-29 years. All (100 %) of the women belonged to the lower middle and lower socioeconomic status. Majority (85.48 %) women had weight gain in pregnancy less than 8 kilograms. All IUGR patients had estimated foetal weight less than 10th percentile appropriate to that gestational age. The most prevalent risk factors were anaemia and gestational hypertension. In our study, out of 62 patients, 26 patients had AFI≤5, of which 13 foetuses had NICU admission and 30 patients from our study had abnormal CTG of which 15 foetuses had NICU admission. Out of 62 patients of our study, 12 patients had abnormal Ut A-

PI of which 4 foetuses had mortality. This suggests a strong association of Ut A-PI with neonatal mortality.25 patients in our study had MCA-PI abnormal and 12 out of these delivered by caesarean section. 64.52 % of patients underwent caesarean delivery. There was decrease in morbidity of newborn as the gestational age advances. 100 % babies had birth weight less than 2 kilograms. There was 6.45 % (4 foetuses) mortality in our study. There was no significant difference in maternal and neonatal morbidity in terms of mode of delivery (caesarean delivery/vaginal delivery). Birth asphyxia was found to be a major cause of NICU admission.

CONCLUSION:

Weight gain seems to be a very strong prognostic factor in terms of association with IUGR, so diagnosis of decrease in weight gain should be made at an earliest and efforts should be made towards adequate weight gain in pregnancy. Ut A-PI shows promising results in predicting severe foetal compromise. Our study suggests a strong co-relation of mortality with altered Ut A-PI. Late onset IUGR still remains a dilemma and it is difficult to predict, diagnose and even more difficult to manage.

KEY WORDS-

1. Late onset IUGR
2. Doppler studies
3. Perinatal outcome
4. Prevalence

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INTRODUCTION

Foetal growth is the result of maternal availability of nutrients, placental transfer and its own growth potential [(1)]. The "normal" neonate is the one whose birth weight is between the 10^{th} and 90^{th} percentile as per the gestational age, gender and race with no feature of malnutrition and growth retardation. Intrauterine growth restriction (IUGR) is associated with perinatal mortality and significant morbidity of surviving newborn. It is characterized by the failure of the foetus to reach its genetic growth potential [(2)]. The diagnosis of IUGR is currently performed on the basis of estimated foetal weight (EFW) below a given threshold, most commonly considered as 10^{th} percentile.

Normal foetal growth disturbance can cause abnormal weight, body mass or body proportion at birth. The two main foetal growth disorders are IUGR and macrosomia, both of which are associated with increased perinatal mortality rate and long-term morbidity [(3)].

Before antenatal USG for foetal growth was clinically available, absolute birth weight was classified as either macrosomia (>4000g) or low birth weight (<2500g), very low birthweight (<1500g) and extremely low birthweight (<1000g). The classification based on birthweight percentile has a significant prognostic advantage as it improves the detection of neonates with IUGR which are at increased risk of adverse health events throughout their life [(4)].

Neonates now are classified as very small for gestational age (below 3^{rd} percentile). Small for gestational age (below the 10^{th}percentile), appropriate for gestational age (10^{th}-90^{th}percentile) or large for gestational age (above 90^{th}percentile)

It is likely that the definition of IUGR lacks sensitivity, as in misses all the cases of growth retardation which aren't below 10thpercentile. Though, this definition takes into consideration a subset of pregnancies which are at higher risk in terms of perinatal outcome. Therefore, it is important that all neonates with a birth weight less than the 10thpercentile will be small for gestational age (SGA), but not an IUGR if there are no features of malnutrition, and a neonate with a birth weight greater than the 10thpercentile will be an IUGR in spite of being an appropriate for gestational age (AGA), if the infants have features of malnutrition at birth.

IUGR is associated with an increased risk of stillbirth, adverse perinatal outcomes and neuro developmental delay [(5) (6) (7)]. IUGR cases which develop before 32 weeks gestation can usually be managed conservatively because the complications of premature birth outweigh the potential benefit of delivery from a hypoxic and undernourished foetal environment [(2)].

Early onset IUGR cases can easily be detected using doppler ultrasound and delivery of such foetus is indicated to prevent stillbirth in the setting of deteriorating cardiac function.

In cases where IUGR develops after 34 weeks gestation, they are known as Late onset IUGR. The morbidity associated with preterm birth is much less significant in Late onset IUGR cases. However, if anyhow the condition goes undiagnosed, it can also result in adverse perinatal outcomes such as a compromised neonatal condition with long-term implications for neurodevelopment. It has been hypothesized that timely delivery of Late onset IUGR foetuses from an unhealthy in utero environment may avoid suboptimal perinatal outcomes [(8)].

Doppler ultrasound markers of placental insufficiency, especially the increase in umbilical artery pulsatility is typical of early onset IUGR. This is frequently absent in Late onset IUGR. There is physiological adaptation in case of Late onset IUGR which is associated with chronic hypoxia that occurs in the third trimester which may help explain the limitations of conventional doppler measures and ultrasound to detect Late onset IUGR. For example, animal studies indicate that the doppler changes seen in acute hypoxia may pseudo-normalize in chronic foetal hypoxia as foetal metabolic adaptation downregulates the foetal requirement for oxygen [(9)]. Moreover, there is hindrance in ultrasound-based foetal weight estimation in case of oligohydramnios, which is commonly associated with Late onset IUGR. This results in low detection rates for Late onset IUGR. The result is a high incidence of unnecessary iatrogenic Late preterm birth and unacceptably high rates of Late gestational stillbirth and perinatal brain injury [(2)].

IUGR should be a cause of concern because they not only indicate an imminent risk of malnutrition and morbidity in women of childbearing age but also signal of a high risk of malnutrition, morbidity and mortality for the newborn in the developing countries such as ours. Thus, we need to develop clinical measures and tools to detect Late onset IUGR.

AIM AND OBJECTIVES

AIM

The aim of this study is to pick up those foetuses that are getting compromised after 34 weeks of gestation mainly due to placental insufficiency and to deliver them before they become hypoxic so as to reduce neonatal morbidity and mortality

OBJECTIVES

1. To find out the prevalence of Late onset IUGR foetuses in our hospital.

2. To compare the predictors and to find out the best predictor for Late onset IUGR for our hospital.

REVIEW OF LITERATURE

By Definition, IUGR is

EFW <10th percentile (ACOG) based on BPD, HC, AC, FL

EFW < 3rd percentile (WHO)

EFW < 2SD below mean (< 2.5th percentile) (Europe)

EFW < 15th percentile (Others)

IUGR CLASSIFICATION

IUGR can be clinically classified as being either symmetric or asymmetric depending on the timing of the insult during pregnancy.

Symmetrical IUGR:

Early insult during pregnancy results in relative decrease in the number of cells and their size. For example, global insults such as from chemical exposure, viral infection or cellular maldevelopment with aneuploidy may cause a proportionate reduction of both head and body size [(10)].

Asymmetrical IUGR:

Late insult during pregnancy such as placental insufficiency from hypertension, resultant diminished glucose transfer and hepatic storage would primarily affect cell size and not number, and foetal abdominal circumference -which reflects liver size - would be reduced. Such somatic growth restriction is proposed to result from

preferential shunting of oxygen and nutrients to the brain. This allows normal brain and head growth, that is – "Brain Sparing" [10].

Table 1: Symmetrical Vs. Asymmetrical IUGR

	TYPE 1: SYMMETRICAL	**TYPE 2: ASYMMETRICAL**
INCIDENCE	25%	75%
CAUSES	Intrinsic genetic anomalies Extrinsic TORCH teratogens Severe malnutrition (?), Drugs, smoking, alcohol	Extrinsic utero placental insufficiency ie. , maternal disorders
TIMING OF INSULT	Before 28 weeks gestation	After 28 weeks gestation
CELL NUMBER	Decreased (hypoplastic)	Normal
CELL SIZE	Normal	Decreased (hypotrophic)
HEAD SIZE	Microcephaly	Usually Normal
BRAIN SIZE	Decreased	Usually Normal
LIVER-THYMUS SIZE	Decreased	Decreased
BRAIN/LIVER WEIGHT RATIO	Normal (3/1)	Increased (6/1)
PONDERAL INDEX (PI)	Normal	Decreased
CONGENITAL ANOMALIES	Frequent	Rare
ULTRASOUND BPD AC* HC/AC**RATIO	 Small Small Normal	Early-normal Late-small Small Early-increased Late-normal
POSTNATAL CATCH-UP GROWTH	Poor	Good

IUGR can phenotypically be classified as Early onset IUGR and Late onset IUGR that are distinct by the moment of onset, evolution doppler parameters modifications and postnatal outcome.

Table 2: Early onset Vs. Late onset IUGR

EARLY onset IUGR	LATE onset IUGR
PROBLEM: MANAGEMENT	PROBLEM: DIAGNOSIS
Degree of placental disease: high	Degree of placental disease: low
Frank hypoxia: Cardiovascular adaptation	Subtle Hypoxia: NO Cardiovascular adaptation
Tolerance to hypoxia: Natural history	Tolerance to hypoxia: NO Natural history
High mortality and morbidity	Low mortality but poorer outcome

Table 3: Risk factors of IUGR

Maternal, foetal and placental risk factors for IUGR
Maternal
Previous pregnancy with SGA or IUGR
Constitutionally small mother or low pre-pregnancy weight
Poor maternal weight gain and nutrition (< 1500 cal/day)
Low socioeconomic status
Smoking, alcohol, illicit drugs
Extremes of maternal age: < 16 years, > 35 years
Assisted reproductive technology
New partner for subsequent pregnancy
Teratogens: anticonvulsants, methotrexate, warfarin
Vascular disease: chronic hypertension, pre-gestational

diabetes, antiphospholipid antibody syndrome, collagen vascular disease (e.g., systemic lupus erythematosus, thrombophilia, renal disease, Crohn's disease, ulcerative colitis) Hypoxia—high altitude (> 10 000 ft) Anaemia including hemoglobinopathies
Foetal Congenital infections: cytomegalovirus, syphilis, rubella, varicella, toxoplasmosis, tuberculosis, HIV, congenital malaria Aneuploidies: triploidy, trisomy 13, 18, 21 Microdeletions: 4p- Imprinting: Russell-Silver syndrome Genetic syndromes or foetal anomalies Discordant growth in multiple gestation
Placental Uteroplacental vascular insufficiency Chorionic separation (partial abruption, hematoma) Extensive villous infarction Marginal or velamentous cord insertion (chorion regression) Major uterine malformations (unicornuate uterus) Confined placental mosaicism Advanced placental maturation

IUGR

The term "small for gestational age" has been synonymously used with IUGR many times so there has always been a needed to differentiate between IUGR and SGA. SGA can be described as foetuses with their EFW falling below the 10^{th} percentile corresponding to their appropriate gestational age, which is simply low foetal weight, while IUGR is a state when a foetus because of the deficit in placental supply of oxygen and nutrition fails to suffice its potential growth. The decrease in size can be due to genetic predisposition moreover than a growth restriction due to some pathology. Failing to discriminate between True IUGR and SGA can result in high false positive rate, because many SGA foetus are constitutionally small, not related to IUGR [(11)]. But foetus who are "Appropriate for Gestational Age" but are having EFW below 10th centile can be termed growth restricted [(12)].

IUGR can present in two ways, Early and Late Onset [(13)]. Early onset IUGR can present during the second trimester of pregnancy, and abnormal placental growth and development are usual associations. Foetal infections and/or genetic abnormality can be secondary associations [(14)]. Severity of Early onset IUGR is more than Late onset IUGR. Conventional Ultrasound is very easy procedure to identify Early onset IUGR. Also, a frequent feature of Early onset IUGR is "Increased Placental Vascular Resistance" due to abnormal placentation. So now Umbilical artery doppler and foetal biometry are more suitable for diagnosis [(15)].

The timing of Late onset IUGR is after 34 weeks of gestation, and it is the more common than Early onset form [(15)]. Placental dysfunction related to maternal malnutrition and substance abuse is more frequently seen with Late onset IUGR.

The placental supply fails to keep up with more demands of nutrients and oxygen in cases of Late onset IUGR. Neonates are majorly SGA in Early onset IUGR.

When serial growth measures of foetus are not present, foetal biometry can be wrongly reassuring. Moreover, Late onset IUGR is not all the time related with abnormalities of conventional doppler parameters [(16)]. Therefore, many of the Late onset IUGR cases go unnoticed by current protocols. Even though Late onset IUGR is the benign form of foetal growth restriction, any failure to identify and insufficiency in placenta occurring at the end of pregnancy can be of clinical attention, because even this is associated with heightened risk of complications in neonates and stillbirth.

ROLE OF PLACENTA

The major cause of Late onset IUGR is "Placental Insufficiency". Function of the placenta is to connect the developing foetus to the uterine wall and provide nutrients and oxygen via the uterine artery from the mother to foetus and also to removes waste products from the foetal blood. The intervillous space of placenta remains filled due to relatively high pressure from the uterine artery. This facilitates passing of oxygen and nutrients into foetal blood and into the foetal circulation via the umbilical vein [(17)]. (Figure 1a).

The blood flow of uterus increases as the gestation advances. In the foetus, the deoxygenated blood flows through the umbilical arteries to the placenta. Typically, during the late stages of gestation, physiological changes occur to optimize the exchange of gas and substrates from the mother to the foetus. Due to low blood resistance of placenta, it allows perfusion of maternal blood into the intervillous space and umbilical arteries [(17)]. Due to this, abnormal placentation and pathological changes will have a negative effect to foetal and maternal health.

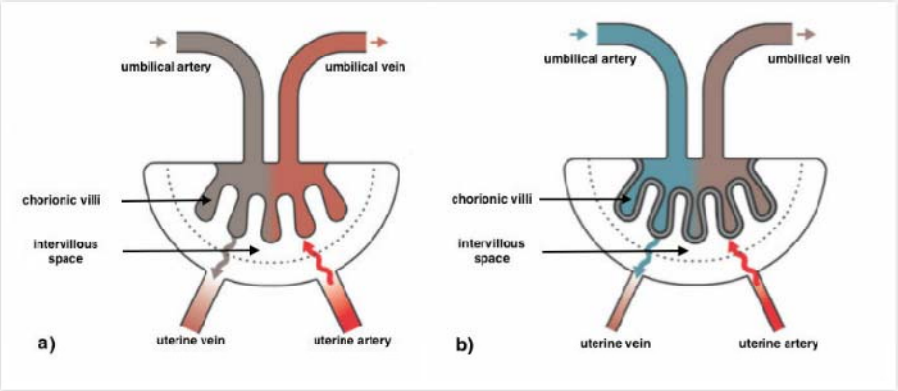


Figure 1: Placental circulation in normal and utero-placental vascular insufficient pregnancies. a) placental circulation in normal pregnancy. b) placental circulation when there is utero-placental vascular insufficiency.

Placental lesions are more commonly associated with majority of IUGR cases. This was established by Salafia et al. (1992), who investigated placental pathology in 128 IUGR cases and 179 gestational age matched placentas [(18)]. Common lesions of placenta are: Haemorrhagic Endovasculitis, Infarction of chronic villitis and placental vascular thrombosis.

In 55% of cases one or more placental lesions were present, which is more than non IUGR cases (32%). In IUGR pregnancy there are multiple lesions in placenta [(18)]. In co-ordination with Salafia et.al., Redline (2008) also demonstrated five patterns of placental injury related with IUGR complicated pregnancies including maternal and foetal vascular obstruction, perivillous deposition of fibrin and high grade villitis and chronic abruption [(19)]. The condition of the umbilical cord also affects foetal growth. It was also observed that growth restricted growth foetuses had lower placental weight as well as altered structure and function of the umbilical cord. These lesions together increase the vascular resistance of the placenta. (Figure 1b)

The clinical picture of Late onset IUGR is associated with mild forms of placental conditions [(20)]. Late onset IUGR is a mixture of foetal and maternal vascular compromise more than increased severe vascular lesions as reported in Early onset IUGR. Placental injuries have minimal effect in Late onset IUGR cases and on growth of foetus at beginning of gestation. But along with the growth the lesions become more complex and risky to foetal development. And soon after the placenta will fail to keep up with the increasing demand of oxygen and nutrients in last trimester, it will result in foetal hypoxia leading to slow growth. Because majority of lesions are related to Late onset IUGR are mild, it is commonly associated with absence of abnormal umbilical artery flow patterns [(16)]. Therefore, the diagnosis of Late onset IUGR relies on detection of physiological adaptations to dysfunction of placenta rather than direct assessment of placental flow resistance.

PHYSIOLOGICAL ADAPTATIONS OF LATE ONSET IUGR

There is a strong association of foetal hypoxemia with Late onset IUGR. Poudel et al. (2015) observed in foetal sheep carunclectomy model that foetal arterial oxygen saturations are approximately half their normal values in IUGR foetuses [(21)]. This relation has also been confirmed by cordocentesis in human IUGR pregnancies [(22)]. Currently identification of Late onset IUGR depends on the detection of hemodynamic adaptation to hypoxemia, which is the main feature.

Metabolism and growth is more majorly affected by hypoxia. To ensure viability and function both at cellular and organismal level, oxygen level is closely monitored. If there is drop in oxygen supply, adaptive responses coordinating with the demand and supply mismatch can lead to minimize the adverse effect caused by hypoxia.

Oxygen is required for metabolic activities such as RNA translation and cell growth at cellular level. Hypoxia can lead to starvation of energy, and stop protein synthesis through different pathways, which in total leads to disrupted growth and proliferation of cells [(23)]. This explains relation between hypoxemia and restricted growth in IUGR foetuses. Metabolic activity normally decides cellular respiration rate. But in limited oxygen levels cells are able to reduce the rate of respiration [(23)]. Delay in onset of tissue anoxia and limit in production of harmful reactive oxygen species can be due to reducing cellular respiration [(24)]. Hence it helps to prevent injury under oxygen deprivation [(24)]. Also, cells can reduce the metabolic activity and energy demand to counteract an increased resistance to the decreased oxygen supply [(25)]. This is called as "Oxygen conformance" [(25)]. For example, in 1986, Honachachaka showed that decreased oxygen delivery in the myocardium lead to decreased contractile function, and that lead to decreased oxygen demand [(26)].

Selective inhibition of metabolic activities allows the cells to preserve limited production of energy for only essential functions. Although the suppression of oxygen caused by chronic hypoxia is reversible and it did not cause any detectable cell injury, it will reduce the size of the cell [(23)]. Therefore, to maintain homeostasis and prevent damage to the tissue at the expense of other metabolic processes, downregulation of energy production and oxygen demand is required [(24)].

Due to this adaptation mechanism, decreased oxygen to IUGR foetal tissue would result into reduced oxygen demand over time at the expense of slowing down of growth. They are also able to adapt in utero to hypoxemia through various mechanisms. In 1974, Cohn et al., investigated the circulatory responses to acute hypoxemia in foetal lambs [(27)]. They changed the oxygen content of maternal ewe's

inspired air to create maternal hypoxia. To avoid hyperventilation of mother, a continuous decrease in foetal arterial oxygen saturation was achieved without any noteworthy changes in the maternal partial pressure of CO_2. Invasive measure of foetal cardiac output and flow distribution was done by nuclide – labelled microspheres. It was seen that in the group without acidaemia, cardiac output was slightly decreased and umbilical blood flow was maintained. So, the distribution of the cardiac output to the placenta was slightly increased. But along with this there was 2-3 times increase in the blood flow to the brain, heart and adrenal gland, and there was decrease in blood flow to pulmonary, renal and GUT circulations [(27)]. Same patterns were seen in other sheep models of placental dysfunctions [(28)].

This type of redistribution is only seen in blood flow across to the foetus and it is made possible by presence of connections between the systemic and pulmonary circulation. Portal vein and vena cava is connected by ductus venosus which allows the blood coming back from placenta to bypass the hepatic system. The foramen ovale allows flow of blood between the atriums and the ductus arteriosus connects the main pulmonary artery to the aorta. Ductus venosus is able to redirect a large proportion of the oxygenated umbilical venous return towards the liver and away from ductus venosus when there is reduced supply of oxygen from placenta [(29)]. Blood is shunted away from the lungs via the foramen ovale and ductus arteriosus. This is tolerated in the foetus because the lungs are not being used for gas exchange. Pulmonary vessels undergo increased vascular resistance when the foetal oxygen saturation is low [(27) (28) (30)]. This results into pulmonary venous return which is diminished and there is an increase in shunting of foramen ovale. This is explained as a reduction in impendence in the cerebral circulation functions in order to maintain the supply of oxygen and nutrients to the brain. This is known as “Brain Sparing effect” which a physiological

adaptation to hypoxia which is detected by abnormal doppler waveforms in MCA. It is both protective as well as pathological.

The neuroprotective effect is at the expense of foetal organs and body growth [(30)]. The foetal proteins that are predominantly produced by the liver is due to increased shunting at the ductus venosus which would affect the biosynthesis. This all leads to impaired foetal growth [(31)]. Disproportionate large head size of the foetus relative to their body size and a decrease in foetal subcutaneous fat is due to chronic redistribution of the oxygenated blood in foetal circulation [(32)].

Total foetal oxygen consumption could remain unchanged with up to 50% acute reduction in oxygen delivery and placental insufficiency directly affecting oxygen delivery to foetus. Increase in fractional oxygen extraction with more oxygen partial pressure difference in between the umbilical vein and umbilical artery is the result of oxygen delivery to the foetus [(30)]. Oxygen extraction fraction increase and foetal arterial oxygen can decrease before altering oxygen and it relates to the degree which is difficult to establish in human foetuses [(33)]. In process of setting of more profound oxygen desaturation of umbilical venous blood, the compensatory mechanism will eventually fail. Yaffe et al. created a model of placental dysfunction by chronically occluding blood flow in the uterine artery in foetal sheep. Measurement at the different levels of changes in foetal blood gas, heart rate and regional distribution was done and it was found that increased degree of foetal hypoxemia is related to progressive reduction of uterine blood flow. Blood flow was redistributed to brain, heart and the adrenal gland under a moderate level of hypoxemia. This feature was consistent with what Cohn et al. had shown [(27)]. Moreover, exposure to very severe foetal hypoxemia was associated with decrease in perfusion to all organs when uterine

blood flow was reduced to 25%. Although elevated oxygen extraction is not associated with elongated foetal oxygen delivery over several days. The conclusion was foetal oxygen consumption was positively related with oxygen delivery in severe chronic foetal hypoxia ($r=0.8$, $P<0.001$) [(34)]. Chronic foetal hypoxia would therefore lead to reduced foetal oxygen consumption in cases of prolonged Late onset IUGR causing cessation of growth and diminished activity. After that a reduction in metabolism of foetal brain and cerebral blood flow will decrease the "Brain Sparing Effect" [(33)].

It has been proved by studies that during sustained hypoxemia, foetal haemoglobin concentration rises, which can be related to the resolution of vasodilation of cerebellum through a rise in oxygen carrying capacity of the blood which has been provided to the brain.

A handful of studies have checked the relationship between cerebral oxygen delivery and in utero brain development, because there has been a lack of traditional methods which can measure foetal cerebral oxygen delivery in animals and humans. Animal models of placental insufficiency show that development of brain is affected. While number of neurons appear to be preserved, dendritic arborization appears to be diminished, and white matter myelination is delayed [(35)] [(36)]. Now there are some proofs that show catch up growth of brain structures in animals and humans in post-natal period [(36)] [(37)].

Impairment of brain growth and development resulting because of chronic adaptation to foetal hypoxia can lead to adverse neurodevelopmental outcomes for the child [(32)].

ADVERSE OUTCOMES IN IUGR

High morbidity and mortality is associated with IUGR pregnancies. Different studies on the perinatal outcome of IUGR pregnancies have shown that SGA and IUGR pregnancies are at an increased rate of stillbirth [38] [39]; increased rate of NICU admissions [40]; increased demand for emergency caesarean section [40] [41] and increased rates of respiratory distress and lower APGAR scores [40] [42].

There are also studies that have proven adverse long-term outcomes due to chronic foetal blood flow redistribution in addition to IUGR associated adverse perinatal outcomes. There has been a relation between cardiovascular disease and hypertension with adaptive redistribution of foetal blood flow in IUGR. Hecher et al. showed that IUGR foetus had systolic and diastolic cardiac dysfunction [43]. This discovery has been supported by a new study comprising of nine severe IUGR cases and nine AGA foetuses who died in perinatal period due to termination of pregnancy resulted from severe maternal illness or any non-cardiac malformation [44]. Biochemical markers and Echocardiographic results done before delivery or death showed signs of severe cardiac dysfunction in IUGR foetuses. The molecular changes of myocytes in these conditions are similar to those in dilated cardiomyopathy and diastolic heart failure. These damages were analogous to cardiac remodelling associated with sustained pressure and volume overload [44]. They are consistent with observational studies, which were reported with relation between LBW and increased foetal death risk from heart disease in adults [45].

There is redistribution of oxygenated blood flow towards the brain and heart in IUGR foetuses at the expense of healthy development of other foetal organs. A relation between LBW and hypertension in infants and adulthood hypertension has been

shown in many human studies, which has been thought to be related with renal function [46] [47]. Since the renal system has a vital role in regulation of blood pressure, it has been shown that relation of hypertension with IUGR could be due to impaired functions of kidney resulting because of reduction in number of nephrons as seen in both animals and stillbirth human foetuses [48] [49] [50].

IUGR foetuses can also be the cause of metabolic disturbances and can manifest in adult diseases such as diabetes mellitus and obesity [51]. There are also increasing evidences showing that intrauterine growth restricted foetus has adversely affects brain development [51].

TIMING OF DELIVERY IN LATE ONSET IUGR

Better techniques for detection of Late onset IUGR are emerging which might help in providing a window of opportunity of clinical intervention to optimize the perinatal and developmental outcomes of babies under effect of Late onset IUGR. Many studies have been done to assess the importance of potential treatment options such as "Low Dose Aspirin" and "Maternal Oxygenation" but they did not yield and convincing benefit in relation of birth weight or extending the gestational age in IUGR foetuses [52] [53].

So, estimating the optimal time for delivery remains the mainstay for management in IUGR. Early delivery from an unfavourable in utero condition could avoid some of the bad effects related to IUGR, but there are also risks associated with late preterm birth. Escobar et al. showed that infants born between 35 weeks to 36 weeks of gestation had significant amount of mortality and morbidity [54]. These infants had almost three times more rate of respiratory distress compared to infants born at or after 37 weeks of gestation. And also, late preterm infants were more likely to be

hospitalised again in comparison to term infants [54]. So, the relative risks of potential morbidity related to late preterm birth against the ones resulting from continued exposure to an unhealthy intrauterine environment should be considered and then decision should be taken. Till this date there has not been any convincing benefit in efforts to investigate the effect of modifying the timing of delivery. A large randomized controlled trial named Growth Restriction Intervention Trial (GRIT) (2004), was done to assess the survival and long term neurological outcomes of early elective delivery compared with delayed delivery in Early onset IUGR pregnancies. There was no difference seen in short term outcome with immediate delivery compared against more conservative management. There was no difference between the two groups in subsequent infant developmental assessments at two years, but the immediate delivery date which dated before 31 weeks of gestation, had a higher rate of severe disability [55]. It was concluded that the timing of delivery had a very less impact on long term neurodevelopment of foetus, so it was safer to wait especially before 36 weeks of gestation. But these findings have very less relevance to Late onset IUGR and they can be influenced by selection bias, with less severe cases, in which it would be safe to wait but it is more likely to be recruited. So, it was suggested that this selected group of GRIT study may not be the ultimate representative of the majority of IUGR cases [15].

Disproportionate Intrauterine Growth Intervention Trial at Term (DIGTAT) (2010) compared the short-term outcomes of induced labour to the outcomes of expectant monitoring for foetuses with suspected Late onset IUGR. It was a multicentre randomized trial, and in it 650 singleton pregnancies which were suspected to have IUGR beyond 36 weeks of gestation were recruited. Suspected IUGR was defined as foetal abdominal circumference below the 10^{th} Percentile. Induced labour group

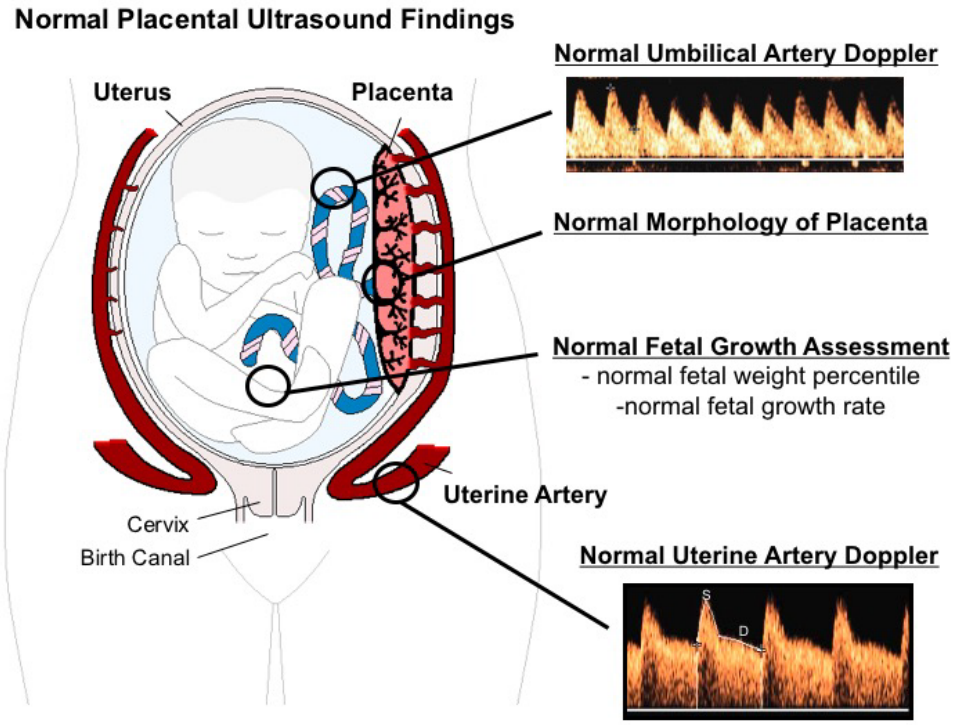
foetuses were delivered ten days earlier and weighed 130 grams less compared to those in expectant monitoring group. There was no marked difference in the adverse neonatal outcome (Including death, 5 min APGAR <7; Umbilical artery pH<7.05 Or Admission to ICU) in between the induction group and the expectant monitoring group (5.3% vs 6.1%, 95% CI of difference; -4.3% to 3.2%). The rate of caesarean was not increased by early induction [(11)]. In a subsequent neurodevelopmental follow up study, questionnaires designed to detect developmental delay and behavioural problems were completed by parents of children in the study. It was reported by the authors that developmental outcome was comparable between the two groups. However, they also showed that foetuses who had growth restriction and birth weight less than 3rd percentile 2 years of age had performed worse in their developmental tests. So, the conclusion was that severe growth restriction remains the most important predictor of abnormal developmental outcome at two years of age. Moreover, there was no difference between induction of labour or expected management in comparison of short or long-term outcomes in suspected IUGR pregnancies [(56)]. The limitations in this study had been considered while interpreting the study results. Even though foetus with both abnormal and normal UA doppler were included in the study (both having similar UA doppler parameters), the foetal monitoring failed to include the assessment of MCA doppler, which has been proven to be more accurate indicator of the presence of IUGR than doppler of umbilical artery in late pregnancy [(57) (41) (16)]. These limitations contribute to the 30% of the false positive cases of IUGR which were reported by the DIGITAT study.

This study nicely illustrates the challenges of accurate diagnosis of Late onset IUGR and that improved techniques for discriminating between SGA and Late onset IUGR are needed.

CONVENTIONAL SONOGRAPHY IN LATE ONSET IUGR

Current trend of pregnancy monitoring is largely relied on non-invasive obstetric ultrasound. Properties of acoustic physics are used in USG to localize and characterize different types of tissue. The frequency of these sound waves is higher than those audible to human ear. During the scan, an ultrasound transducer sends the impulses into the tissue and it receives the echoes that come back. Varying degrees of sound reflects the different tissues. Echoes containing spatial and contrast information of tissues are recorded and displayed as images [(58)]. This USG can provide intelligence about wellbeing of the foetus from varying aspects including the assessment of foetal growth rate and blood flow waveforms in major vessels of foetus.

The features of blood flow in foetal and maternal vessels and different foetal growth patterns are different in Early onset IUGR and Late onset IUGR are summarized in Figure 2



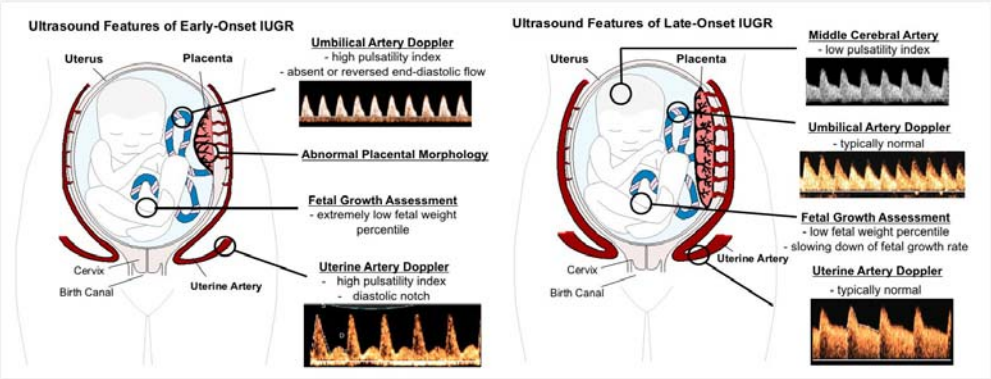


Figure 2: Ultrasound features of a) normal, b) Early onset IUGR and c) Late onset IUGR pregnancies. Normal, Early onset IUGR and Late onset IUGR pregnancies have different features of blood flow in foetal and maternal vessels and different patterns of foetal growth.

BIOMETRY

Ultrasound based assessment of foetal biometry has become a very routine practice in obstetrics during the last 40 years, and it has a crucial role in decision making process regarding the timing of delivery [(59)].

A vital aspect of pregnancy is accurate assessment of foetal growth and decision making on pregnancy management. Crown rump length is a measure of the foetus from the head to the buttocks and it is mainly used to know the gestation age [(60)].

Estimation of gestational age based on crown rump length (CRL) is more reliable than the calculations which are based on the first day of the last menstrual period according to various studies [(61)]. Rate of post term pregnancy gets reduced with the use of systemic ultrasound of pregnancy dating. It helps in reducing the unnecessary interventions and also improves identification of post term pregnancies, which are at risk of complications [(62)].

Several equations have been developed to calculate the EFW mathematically. Amongst all these, Hadlock's formula has been proved to give most accurate results and it is widely accepted and commonly used [(63)]. This formula was proven on 167 live born foetuses which were examined within one week of delivery. Oliver et al. had evaluated the accuracy of Hadlock equation in 709 women who had undergone ultrasound examination within 8 weeks of delivery [(64)] and showed that the Hadlock equation was only having -0.47% systemic error for scans done within 2 weeks of delivery. This gave a spotlight on a very small margin of error if the method of application of the formula was correct.

Even though the reliability of the formula has been proved by several studies [(65)] [(66)], there is decrease in the accuracy of the estimation as the interval between the testing and delivery increases [(64)]. In addition, the absolute error of the method tends to increase with higher birth weight regardless of the interval in between the ultrasound exam and delivery [(64)]. Moreover, the ultrasound based foetal weight estimation can also be disturbed by a low level of amniotic fluid and maternal obesity. Asymmetrically growth foetus such as IUGR foetuses who have "Brain Sparing Effect" can decrease the accuracy of Hadlock's Formula [(67)].

IUGR can be reliably identified by measures of foetal weight taken from serial ultrasounds in order to plot growth rate reliably [(53)]. But the estimation can be unreliable if the interval between the serial ultrasound scans are less than 2 weeks apart [(68)]. The most common limitation of this modality is that none of them directly measures the foetal volume or foetal weight. In order to identify IUGR weight estimation should be combined with other indicators of a compromised foetal conditions.

AMNIOTIC FLUID INDEX (AFI)

AFI is one of the deciding components of foetal biophysical profile and it can predict pregnancy outcome. Very low values of AFI suggest IUGR and renal anomalies of foetus whereas very high values point towards foetal gastrointestinal anomalies, maternal diabetes mellitus etc. AFI assessment by USG is an important tool in assessing the foetal health.

It is measured by four quadrant technique as described by Phelan et. al. in 1987 [(69)].

Normally AFI peaks at 32 to 34 weeks of gestation and thereafter there is a gradual reduction in amniotic fluid due to increase in concentrating capacity of foetal kidneys. However, sudden reduction in the amniotic fluid may suggest underlying placental insufficiency.

The values between 8-25 cm are considered normal AFI. The values between 5-8 cm are considered as borderline and less than 5 cm AFI is considered as oligohydramnios. In oligohydramnios, there is higher incidence of perinatal morbidity and mortality and this may suggest immediate delivery as the only way out.

AFI is the 5^{th} parameter of biophysical profile and the 2^{nd} parameter in modified manning's score.

Third trimester AFI correspond to foetal urine production and this if normal suggest good placental perfusion, foetal nutrition and oxygen transfer, hence measurement of AFI is important for foetal surveillance.

DOPPLER

SGA and IUGR are not the same, therefore along with foetal growth assessment, doppler ultrasound which helps in detection of haemodynamic adaptation of foetal hypoxia acts as a vital tool in the assessment of foetal wellbeing. Doppler ultrasonography uses the doppler principle, which states that the frequency of the echo reflected from the target is different from the incident frequency. To detect any movement of fluid such as blood, a series of pulses are sent over during a doppler ultrasound. If echoes received by the transducer are same from time to time, then it is a stationary tissue whereas echoes from the flowing blood have a marginal difference related to the time it takes for the signal to return to the transducer. This difference helps in deciding whether the blood is moving towards or away from the transducer. Echocardiography is application of this technique in evaluation of blood vessels or the heart itself. Thus, doppler plays an important role in diagnosis of IUGR. Recent screening methods with the use of doppler ultrasound indirectly examine placental insufficiency in cases of IUGR by identifying maternal adaptations to defective trophoblastic invasion process [(70)]. Preferential perfusion of vital organs such as the brain, heart and adrenal glands and spleen is due to foetal circulatory adaptations to acute and chronic hypoxia [(42)]. More accurately a doppler can also be useful to examine maternal uterine arteries and the foetal ductus venosus, but it has been more commonly used in foetal arterial system including the MCA and Umbilical artery. The parameters of doppler in these vessels are important markers of potential redistribution that blood flows in the setting of Late onset IUGR.

UTERINE ARTERY DOPPLER

The uteroplacental circulation by uterine artery can be assessed by doppler ultrasonography. It helps in predication of risk of Early onset IUGR and pre-eclampsia resulting from an abnormal placenta formation consisting abnormal trophoblast invasion of spinal arteries [(71)]. A high placental vascular impedance will give a notched uterine artery doppler waveform and low diastolic flow velocity in time of early gestation whereas in late gestation, placental vascular impedance should decrease and the notch should disappear.

An abnormality in uterine circulation is established by the persistence of uterine artery doppler notch in late second and third trimester [(72)]. The most commonly used doppler parameter is the pulsatility index (PI), which can be calculated by subtracting end diastolic blood flow from peak systolic blood flow and dividing it by mean flow. PI value shows the resistance of the blood flow resulted by the microvascular bed which is distal to the site of measurement. A high PI of uterine artery (Ut A-PI), which indicates high placental flow resistance, is associated with increased risk for pre-eclampsia and Early onset IUGR [(73)]. A meta-analysis done recently has found that abnormal uterine artery doppler indices are related with a three to four-fold increase in stillbirth risk [(39)]. There are two reviews on this with contradicting views on prediction of perinatal outcome of IUGR foetuses. Severi et al. deduced that in predicting any adverse perinatal outcome in Late onset IUGR pregnancy uterine artery doppler could prove helpful in providing additional information [(41)]. But on the contrary a systematic review of diagnostic studies states something opposite, that uterine artery doppler only has limited accuracy in predicting the IUGR or any other

adverse outcomes [74]. Therefore, there is need of more concrete evidence before we can use uterine artery doppler as a standalone monitoring tool for Late onset IUGR.

UMBILICAL ARTERY DOPPLER

Umbilical Artery (UA) doppler measures the vascular resistance in the placenta on the foetal side. In any normal conditions as shown in Figure 3a, if there is low resistance in the Umbilical artery than it allows the continuous advancing flow throughout the cardiac cycle [75]. Decreased, absent or even reversed end diastolic flow in UA is an indicator of a condition where the flow resistance is very high and it is related with abnormality of placental vasculature or dysfunction of the placenta [72]. An example of absent end diastolic flow in the UA of an IUGR foetuses is reflected in Figure 3b. UA doppler assessment is commonly agreed upon clinical standard for detecting early onset IUGR (RCOG, 2002). It has been proved that UA doppler adversely correlates with levels of glucose and amino acids in the blood of umbilical cord [76], so it is believed to be an effective measurement of placental function. Clinical studies on the subject of Early onset IUGR have shown that foetuses with absent or inverted end diastolic flow had a relative risk of 4.0 and 10.6 and on comparison to that of perinatal morbidity and mortality [77]. But UA PI might not be important in early detection of Late onset IUGR. In common relations with the ductus venosus doppler, it has been said that UA doppler only becomes abnormal in later stages of placental dysfunction. Rigano et al. has proved that by the time UA doppler detects any abnormality, umbilical vein flow is already diminished [78]. In an animal model of foetal distress, the diastolic UA flow only became inverted just before the foetal death in six or seven animals [79]. It has also been shown that in the cases of inversed end diastolic flow in UA, >70% of artery situated in placental tertiary villi were obliterated [80]. So, UA

doppler isn't a reliable modality to identify early and mild signs of Placental Dysfunction. McCowan et al., who worked on 186 SGA foetuses, gave a conclusion that UA doppler is a reliable indicator of the severity of IUGR, but not solely associated with neonatal outcome [(81)]. In this study, they proved that those SGA foetuses with an abnormal UA doppler which were born earlier than two weeks were smaller in all growth parameters than those with normal UA doppler but had similar Ponderal Index. When they adjusted for the effect of birth weight and gestational age at birth, UA doppler did not serve as a predictor of the chances of newborn admission to nursery and length of stay in the hospital [(81)]. But the interesting finding here was that, that with increase in the gestational age at the time of onset of IUGR, the chances of finding abnormal UA doppler levels decreased. In one such study dealing with the utility of various doppler parameters in the situations of Late onset IUGR, UA doppler results stayed within the normal range despite the formation of brain sparing physiology showed by other parameters in up to 20% of cases of SGA [(16)]. Thus, a major proportion of SGA foetuses with normal ranged UA doppler are in reality Late onset mild IUGR cases and they are at risk of developing adverse perinatal outcomes. This conclusion was supported by Figueras et al., (2009), who studied the neurobehavioral performance in 102 SGA foetuses with normal UA doppler and 100 AGA foetuses at the revised age of 40 ± 1 weeks. They concluded that performance score of SGA newborns was notably low which was suggestive of delayed neurologic maturation despite the normal UA doppler [(82)]. A study on the outcome of 2-year neurodevelopment of 112 full term SGA newborns with a normal UA waveform as compared with 111 AGA foetuses was done. After settling for important confounders such as gender, at birth gestational age, parental smoking, socioeconomic status, developmental results were assessed using the Bayley Scales of Infant Toddler

Development (Bayley III) test. All the Bayley III measures of cognitive, language, adaptive and motor skill scores were proven to be markedly poorer in SGA group with normal UA Doppler [83]. Thus, UA doppler cannot be considered as a standalone for placental insufficiency leading in adverse development of brain.

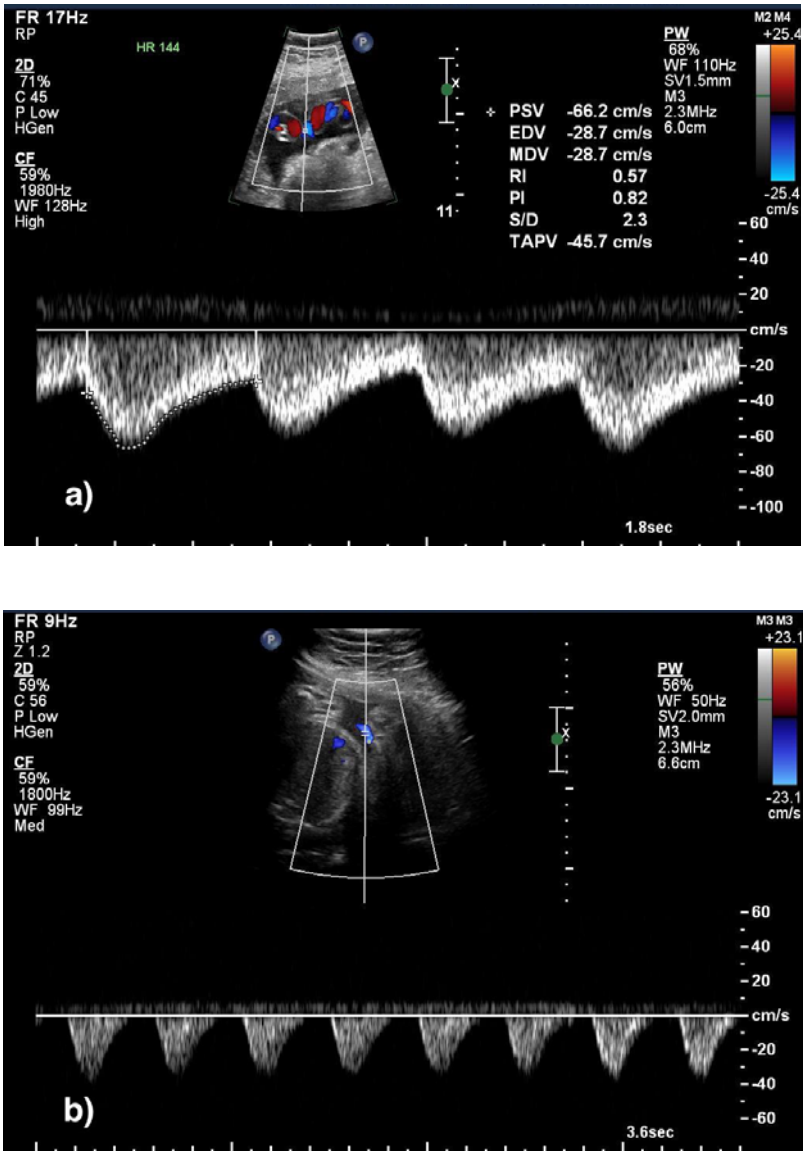


Figure 3: Example of normal and abnormal umbilical artery doppler. a) Umbilical artery doppler in a 37-weeks normal foetus with continuous flow throughout a cardiac cycle. b) Umbilical artery doppler in a 34-weeks IUGR foetus with absent end-diastolic flow.

MIDDLE CEREBRAL ARTERY DOPPLER

The Middle Cerebral Artery doppler is another reliable technique for identifying foetal adaptation of hypoxemia. The main tributaries in the Circle of Willis are right and left MCAs. Since they distribute over 80% of cerebral blood flow, and they are normally situated perpendicular to the anterior abdominal wall of the mother, they are amongst the preferred vessels to assess the foetal cerebral circulation [(84)].

The cerebral circulation has high resistance continuous forward flow throughout the cardiac cycle under normal conditions [(84)]. This has been depicted in Figure 4a. When foetal hypoxia is present, circulatory adaptation leads in redistribution of the blood flow in order to raise the perfusion to the vital organs including the brain [(27)]. Raised cerebral blood flow causes cerebral vasodilation, which is mediated many mechanisms along with the action of adenosine (Pearce, 2009). Inhuman IUGR pregnancies, this is related to raised diastolic blood flow and a decrease in PI [(85)] (Figure 4b).

It has been proposed that in SGA foetuses near term, MCA-PI can be a reliable indicator of adverse outcome independent of UA doppler findings [(57)] [(41)]. In Hershkovitz, et al.'s study, he had reported that amongst 47 SGA foetuses, (72%) had a normal UA doppler Results, but out of 34; nine had abnormal MCA in PI [(57)]. In the 13 foetuses consisting abnormal UA PI, seven out of them were also having abnormal MCA-PI. The ratio consisting head circumference/ Abdominal circumference (as a measure of asymmetrical growth) was negatively related with MCA-PI ($p<0.001$) [(57)]. So, abnormally low MCA-PI is associated with a disproportionately large head due to brain sparing effect. Moreover, this study also demonstrated association of brain sparing with increased incidence of caesarean section and NICU admissions. It was

also demonstrated that abnormal MCA-PI in SGA foetuses had increased chances of caesarean section due to foetal distress [41]. However, the relation of MCA-PI and risk of foetal distress was only reliable when abnormal uterine artery waveform was also considered. And even a systematic review questioned the proposal that MCA doppler independently should be used as a predictor of foetal compromise [86].

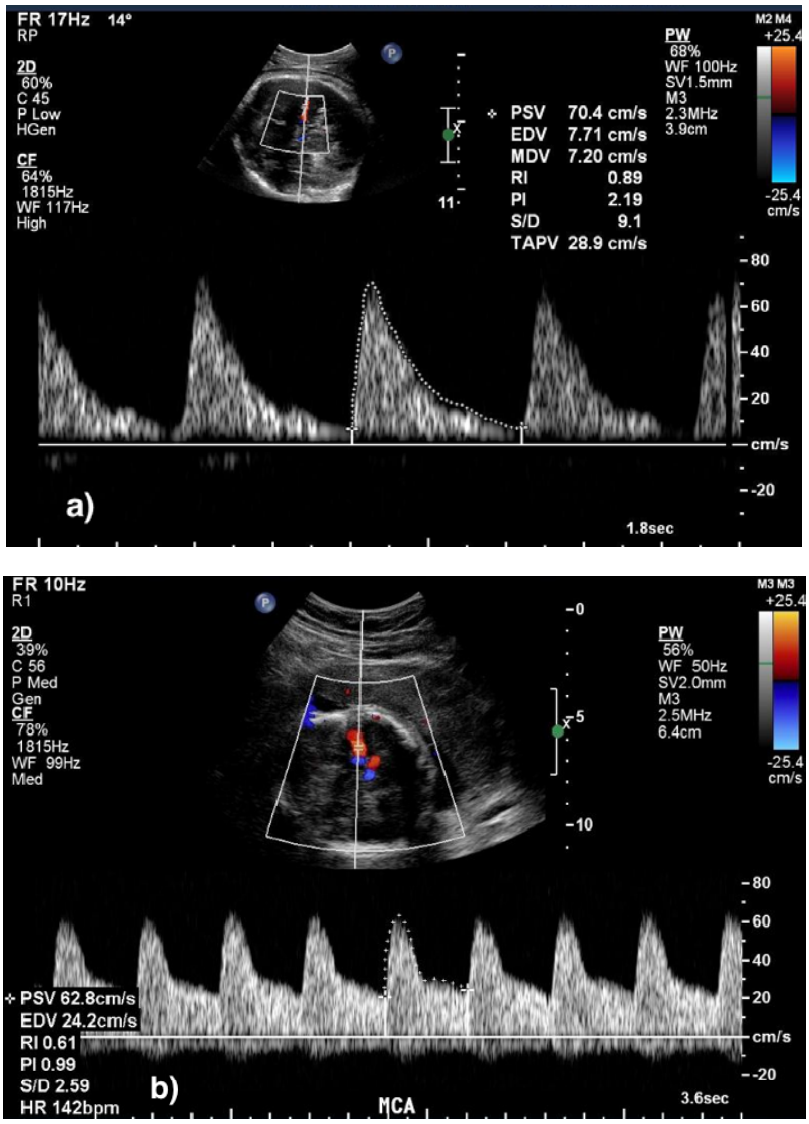


Figure 4: Example of middle cerebral artery doppler in a normal and an IUGR foetus. a) Middle cerebral artery doppler in a 37-weeks normal foetus. b) Middle cerebral artery doppler in a 36-weeks IUGR foetus with elevated diastolic flow, therefore, lower pulsatility index.

CEREBROPLACENTAL RATIO

Though there is controversy related to the use of abnormal UA doppler and MCA doppler as separate indicators of foetal compromise, a combination of these two known as Cerebroplacental Ratio (CPR) is emerging out as reliable predictor of adverse pregnancy outcomes [(87)]. The CPR is calculated by the division of MCA-PI by UA PI; and thus, it signifies the relation of placental status and response of foetus to it. CPR can be abnormally low if there is increase in placental flow resistance indicated by increased UA PI; decreases in cerebral flow resistance marked by decreased MCA-PI and finally if both the above coexist. Moreover, CPR could be abnormally low when UA and MCA-PI, both are nearly normal [(87)]. An abnormal CPR would signify either brain sparing or high resistance of the placenta or both together. Conventional thinking would justify that only SGA foetuses are having chances of placental dysfunction and foetal hypoxia, but it has been proved that abnormal CPR can prove to be an important indicator of foetal hypoxia and is independent of EFW (88,89,90). This is very important because a large portion of AGA foetuses are also subject to placental insufficiency and foetal hypoxia. In an AGA model, Prior et al. deduced that abnormal CPR was a better predictor of the need for emergency caesarean section than abnormal UA or MCA doppler alone [(89)]. In this study, amongst the 400 AGA foetuses at term, 36.4% of the foetuses were having CPR<10th centile (according to gestational age) and they had to undergo caesarean section because of foetal distress, while only 9.5% of those were having CPR between 10th and 90th centile and had to undergo caesarean section. Not a single foetus with CPR >90th centile required a caesarean section ($p < 0.001$) [(89)]. This study in favour with another study by Figueras's group (2015), which took 509 foetuses into consideration with Late onset SGA. In this study, amongst all these

foetuses with CPR <10th centile, 37.5% had adverse outcome (Neonatal acidosis, NICU Admission, 5 min APGAR <7, etc.); while 19.1% foetuses with CPR >10th centile had adverse outcome ($p<0.05$) [(90)]. Synonymously, the study by Khalil et al. (2014) of a large cohort study with >8,000 subjects having late gestation, also advocated the utility of CPR by showing that foetuses having abnormal CPR had an increased rate of NICU admission and complications with normal CPR ($P <0.004$). These studies weigh in the concept that CPR detects late gestation foetuses at increased risk of foetal distress and neonatal complications regardless of EFW.

A multicentred Prospective Observational Trial to Optimise Pediatric Health in IUGR(PORTO) consisting of 1200 SGA pregnancies having foetuses with EFW less than 10^{th} percentile were recruited for the study [(40)]. Amongst the 146 cases with CPR <1 (irrespective of gestation age), 64% were admitted to NICU with a mean length of stay around 31 days, as compared to foetuses with CPR>1.22% of which were admitted to a neonatal unit ($P<0.0001$). There was an eleven-fold increase seen in foetuses with abnormal CPR in the risk of adverse perinatal outcome ($P <0.0001$). At different cut-off values of CPR, the sensitivity (detection of true IUGR) and specificity (Detection of true non-IUGR) was assessed by this group. They debated that a categorical cut off of 1 for CPR was okay and manageable for clinical application. But when compared using CPR<5th centile for gestation age, CPR <1 regardless of the age of gestation had decreased sensitivity but raised specificity. The sensitivity of CPR in detecting perinatal outcome was proven better in comparison to UA PI and MCA-PI independently, even though both of these independent components were more specific than CPR [(16)]. More and more studies now weigh in the use of CPR as a predictor for pregnancy outcome in both severe and mild forms of

IUGR., and it should also be taken into consideration to guide the risk evaluations of IUGR pregnancies along with conventional UA doppler and EFW evaluations.

Table 4: Immediate Complications of Intrauterine Growth Restricted Newborn [91]

Morbidity	**Pathogenesis/Pathophysiology**	**Prevention/Treatment**
Intrauterine foetal death	Usually result of Placental insufficiency causing chronic hypoxia Foetal congenital malformation Maternal and foetal infection Sentinel events like Abruptio placentae, cord rupture or prolapse Placental infarcts and preeclampsia	Needs regular antepartum and intrapartum monitoring with planned delivery Plan delivery in case of severe/worsening foetal distress in tertiary care level centre
Neonatal Mortality	Antepartum, intrapartum and postpartum neonatal insults contributed by other neonatal morbidities	Tertiary level neonatal care
Perinatal/ Neonatal Asphyxia	Chronic foetal hypoxia superadded with acute foetal hypoxia Acute sentinel event like Abruptio placentae, cord rupture or prolapse Placental abnormalities leading to insufficiency Pre-eclampsia/eclampsia	Needs regular Antepartum and Intrapartum surveillance Regular foetal growth monitoring by USG and plotting on customized growth chart Early detection of IUGR/SGA Regular Biophysical profile (BPP) Delivery at appropriate time and place having appropriate neonatal facilities Delivery attended by person skilled in neonatal resuscitation

Hypothermia	Poor thermoregulation mechanism Increased surface area with large head Poor subcutaneous and body fat leading to less thermogenesis and lower insulation Less brown fat Deficiency of catecholamine in body Increased insensible water loss through skin Other associated neonatal morbidities like Hypoglycemia and Hypoxia	Warm delivery room with temperature around 26 to 28°C Using cling wrap, heated mattress and warm humidified gases in delivery room Protect heat loss by radiation, conduction, convection and evaporation. Maintain thermo-neutral temperature in nursery Early breastfeeding Rooming in with mother/ Warm Transport Early skin to skin contact in delivery room
Hypoglycemia	Poor glycogen stores of liver and muscles Poor other alternative energy source Decreased fat (adipose tissue) Decreased ability to oxidize free fatty acids and triglycerides for gluconeogenesis Poor gluconeogenesis and glycogenesis Low level counter-regulatory hormones like epinephrine and glucagon Secondary to other associated comorbidities including polycythaemia, hypoxia, hypothermia Heightened insulin receptors sensitivity	Monitoring Blood sugar for initial 48-72 h of post-natal life as per the protocol Early breast feeding within one hour of birth and if required formula supplementation Intravenous glucose when sugar is less than 25 mg/dl or symptomatic neonate

Hyperglycemia	Low insulin production secondary to immature pancreas Insulin resistance Too much exogenous glucose infusion Increased epinephrine and glucagon level	Sugar monitoring as per protocol Avoid high glucose concentration administration Treatment of symptomatic hyperglycaemia with infusion titration and insulin
Hypocalcemia	Decreased transfer of calcium in-utero secondary to hypophosphatemia induced by chronic hypoxia. Immaturity of parathyroid glands	Calcium supplementation Monitoring of calcium levels
Polycythaemia/ **Hyperviscosity/** **Leukoneutropenia**	Placental insufficiency causes chronic intra-uterine hypoxia that leads to high foetal erythropoietin Transfusion of blood from mother to foetus	Monitor haematocrit at 2, 12 and 24 h after birth Regular feeding Prevent excessive postnatal weight loss Fluid supplementation and partial exchange transfusion if symptomatic
Persistent pulmonary hypertension (PPHN)	Abnormal of pulmonary vasculature with thickened tunica media up-to intra-acinar arteries as result of chronic in-utero hypoxia Other associated morbidities like birth asphyxia, hypoglycemia, hypothermia, hypocalcemia, polycythaemia, hypoglycemia and sepsis	Avoid hypoxia and hyperoxia Normalization of metabolic milieu Cardiovascular support Selective and non-selective pulmonary vasodilator Mechanical ventilation if required
Pulmonary Haemorrhage	Abnormal pulmonary vasculature Other associated co-morbidities like hypothermia, polycythaemia,	Gentle ventilation Management of co-morbidities

	asphyxia and neonatal sepsis	Supportive care for pulmonary haemorrhage
Meconium Aspiration	Chronic in-utero hypoxia Intrapartum hypoxia secondary to any sentinel event	Regular monitoring during intrapartum for meconium passage No role of amnio-infusion for prevention of meconium aspiration syndrome (MAS) Resuscitation as per the NRP 2015 guidelines Establish regular respiration. No need role of tracheal suctioning for both vigorous/depressed newborns born with meconium stained liquor
Broncho-pulmonary dysplasia	Antenatal hits to foetal lung like chorioamnionitis, foetal infection and preeclampsia Abnormal pulmonary vasculature Post-natal insults to neonatal lungs like ventilation, hypoxia, hyperoxia, neonatal sepsis and Patent ductus arteriosus	Antibiotics to mother in case of chorioamnionitis Gentle ventilation Preventing hypoxia, hyperoxia, and neonatal sepsis
Feed intolerance/ **Necrotizing enterocolitis (NEC)**	Decreased intestinal perfusion secondary to redistribution of blood to vital organ in response to chronic hypoxia Focal intestinal ischemia Poor motility	Minimal enteral nutrition to be given Protocolised increase in daily feeds Cautious start of enteral feeding Use of probiotics and lactoferrin Use only breast milk (either owns mothers

		milk or donor milk) Supportive treatment in case of development of NEC
Renal Problems	Chronic in-utero hypoxia and perinatal asphyxia leads to renal tubular injury	Cardiovascular support Maintain adequate renal perfusion
Immunodeficiency	Chronic in-utero and post-natal malnutrition Congenital infection Reduced number of T and B lymphocytes Poor immunological maturity	Early, aggressive and optimal nutrition Promoting breast feeding Prevention of neonatal sepsis
Retinopathy of prematurity (ROP)	Intrauterine hypoxia Altered levels of growth factors Diminished antioxidant capacity Post-natal insults like hyperoxia, hypoxia, and sepsis	Targeted saturation (90-95%) ROP screening of susceptible Treatment if required
Ferritin	Low levels Defective transport through placenta Increased premature delivery	

MATERIALS AND METHOD

The present study is a prospective observational study and was conducted at Dhiraj Hospital, a tertiary care centre situated in the rural area of Vadodara from February 2016 to January 2017.

All pregnant women diagnosed of Late onset IUGR who fitted in the study criteria were selected for the study.

CRITERIA FOR SUBJECT SELECTION

INCLUSION CRITERIA

- All singleton pregnant patients with vertex presentation (after 34 weeks of gestation) undergoing regular antenatal check-up (with accurate dates, which were substantiated by first trimester dating scan were enrolled) of which the cases which were diagnosed of Late onset IUGR were taken and followed till delivery.

EXCLUSION CRITERIA

- Autoimmune disease.
- Eclampsia.
- Exposure to drugs, alcohol, nicotine abuse.
- Multiple pregnancy.
- Abnormal placentation.
- Malpresentation.
- Constitutionally small babies.
- Congenital malformations.

DIAGNOSIS

As there is no gold standard for diagnosis of Late onset IUGR, so we have taken into consideration the following aspects of foetal growth restriction:

1. EFW $<10^{th}$ percentile.
2. After 34 weeks of gestation.

Once diagnosis of Late onset IUGR was made, weekly follow-ups were done and following parameters were studied.

1. Estimated foetal weight(EFW) (by USG, using Hadlock's formula)
2. Amniotic Fluid Index (by USG-4 quadrant method)
3. Cardiotocography (CTG)
4. Umbilical Artery Pulsatility Index (UA-PI)
5. Uterine Artery Pulsatility Index (Ut A-PI)
6. Middle Cerebral Artery Pulsatility Index (MCA-PI)
7. Cerebroplacental Ratio (CPR)

PROTOCOL OF TERMINATION OF PREGNANCY

Abnormal CTG	Emergency LSCS
CPR reversal+ another co-morbid factor	Elective LSCS
CPR borderline+ another co-morbid factor	Elective LSCS
CPR reversal (<1)	Vaginal Delivery
CPR borderline (<1.08 to 1)	Vaginal Delivery

(Abnormal CTG=Multiple variable decelerations or Multiple late decelerations)

When vaginal delivery was indicated, if induction was done, continuous monitoring with CTG was done. In presence of abnormal CTG patterns, termination was done by Emergency LSCS.

Pregnancy outcome was then analysed as per the following criteria:

1. Gestational age at the time of delivery.
2. Induced or spontaneous onset of labour.
3. Mode of delivery with indication.
4. Foetus live/still born/intra uterine foetal demise.
5. Morbidity to the mother if any.
6. Birthweight of newborn.
7. Ponderal index of newborn.
8. APGAR Score at 1minute and 5minute.
9. NICU admissions if any.
10. Indication of NICU admission.
11. Mortality of the newborn.

The study was carried out in two phases.

In phase one, parameters for monitoring Late onset IUGR were studied and further management was decided.

In phase two, obstetric and neonatal outcome of Late onset IUGR cases were assessed.

RESULTS AND OBSERVATIONS

PREVALANCE

The total number of deliveries at Dhiraj Hospital, Piparia between 1st Febuary 2016 to 31st January 2017 were 2546

Prevalence = Total no of diagnose Late onset IUGR cases X 100

Total number of deliveries at Dhiraj Hospital

(February 2016 to January 2017)

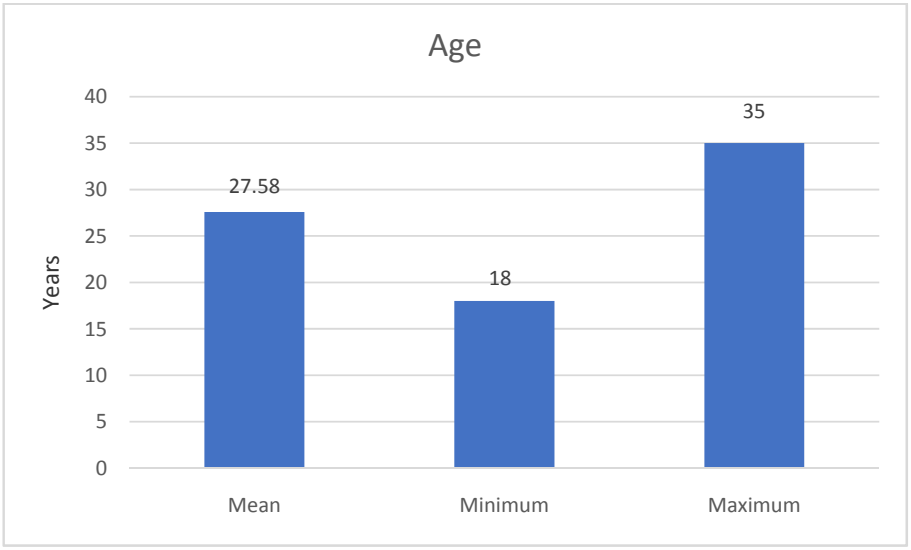
= (62X100) 2546

= 2.44%.

Table 5: Mean, Standard Deviation, Minimum, Maximum values of Age in study subjects. (n =62)

Age (years)	
Mean	27.58
Standard deviation	3.67
Minimum	18.00
Maximum	35.00

Figure 5: Mean, Minimum, Maximum values of Age in study subjects (n =62)

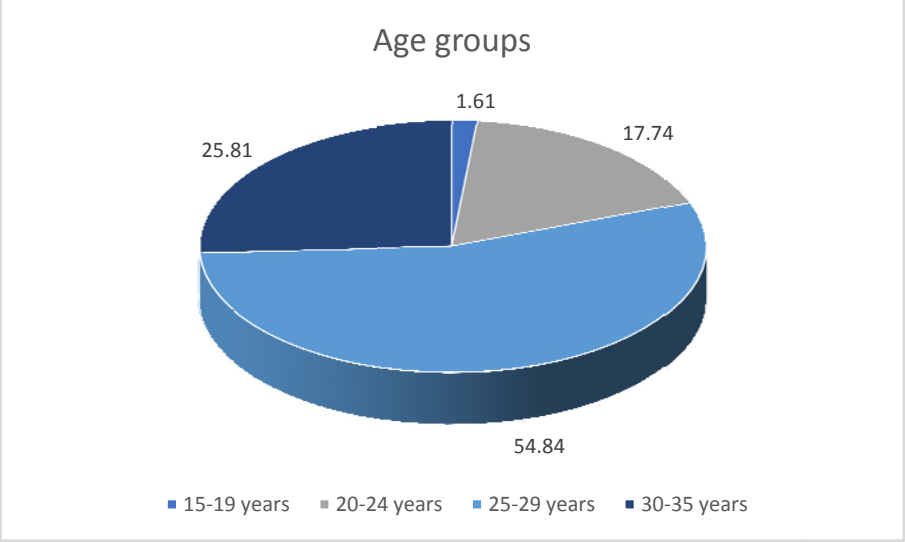


The above table and figure show mean, standard deviation, minimum and maximum values of age in study subjects (n =62). Mean ± SD of age of study subjects was 27.58 years ± 3.67 years. Minimum and maximum age of study subjects were 18.00 years and 35.00, respectively.

Table 6: Distribution of study subjects in different Age groups.

Age groups	n (%)
15-19 years	01 (1.61)
20-24 years	11 (17.74)
25-29 years	34 (54.84)
30-35 years	16 (25.81)
Total	**62 (100.00)**

Figure 6: Distribution of study subjects in different Age groups.

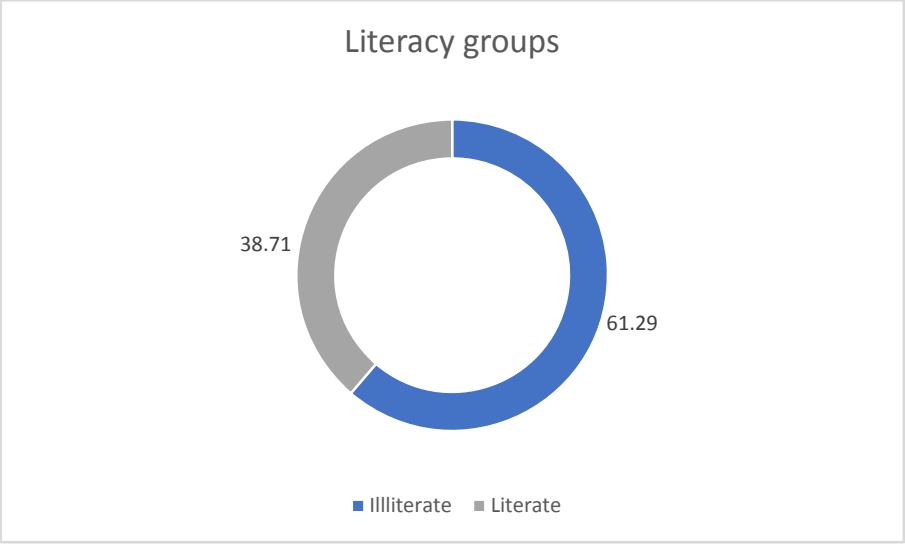


The above table and figure show distribution of study subjects in different age groups. There were 01 (1.61%), 11 (17.74%), 34 (54.84%) and 16 (25.81%) subjects in 15-19 years, 20-24 years, 25-29 years and 30-35 years age groups, respectively. Maximum study subjects (n = 34, 54.84%) were in 25-29 years age group.

Table 7: Distribution of study subjects according to Literacy.

Literacy groups	n (%)
Illiterate	38 (61.29)
Literate	24 (38.71)
Total	**62 (100.00)**

Figure 7: Distribution of study subjects according to Literacy.

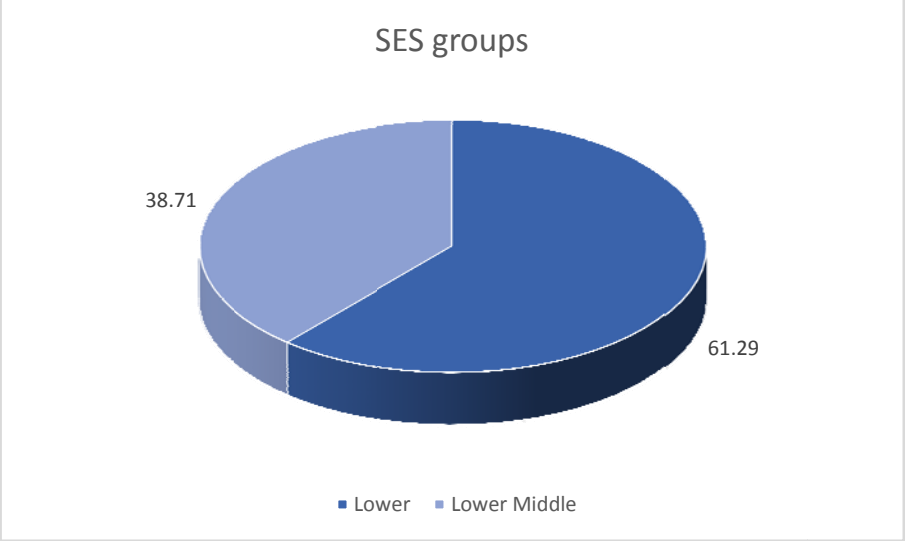


The above table and figure show distribution of study subjects according to literacy. Among the study subjects 38 (61.29%) were illiterate and 24 (38.71%) were literate. Maximum study subjects were illiterate.

Table 8: Distribution of study subjects according to Socioeconomic Status (SES).

SES groups	n (%)
Lower	38 (61.29)
Lower middle	24 (38.71)
Upper middle	00 (0.00)
Upper	00 (0.00)
Total	**62 (100.00)**

Figure 8: Distribution of study subjects according to Socioeconomic Status (SES).

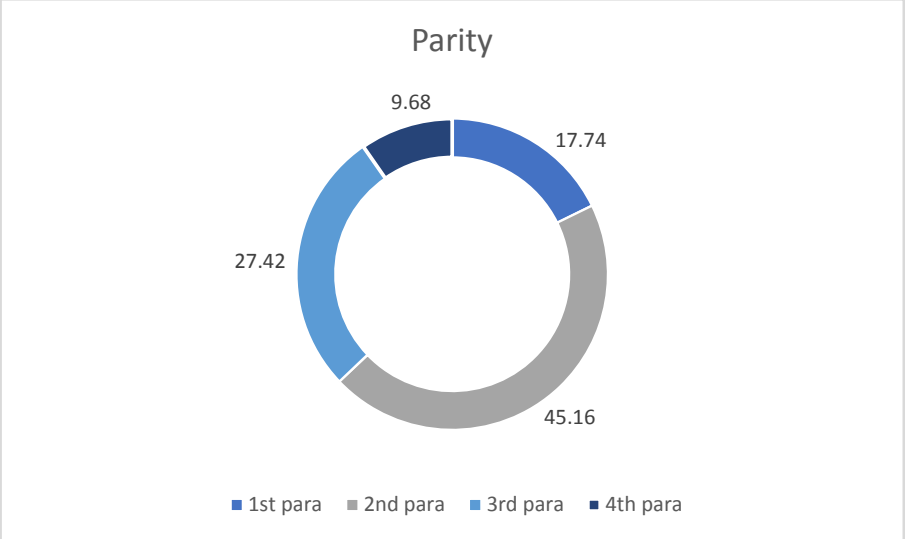


The above table and figure show distribution of study subjects according to socioeconomic status (SES). Thirty-eight (61.29%) study subjects belong to lower SES group and 24 (38.71%) belong to lower middle SES group. Maximum study subjects were from lower SES group. None of the study subjects were in Upper middle and Upper SES groups.

Table 9: Distribution of study subjects according to Parity.

Parity	n (%)
01	11 (17.74)
02	28 (45.16)
03	17 (27.42)
04	06 (9.68)
Total	**62 (100.00)**

Figure 9: Distribution of study subjects according to Parity.



The above table and figure show distribution of study subjects according to parity. There were 11(17.74%), 28 (45.16%), 17 (27.42%) and 06 (9.68%) subjects in 01, 02, 03 and 04 para groups. Maximum subjects (n =28, 45.16%) were of 2nd parity.

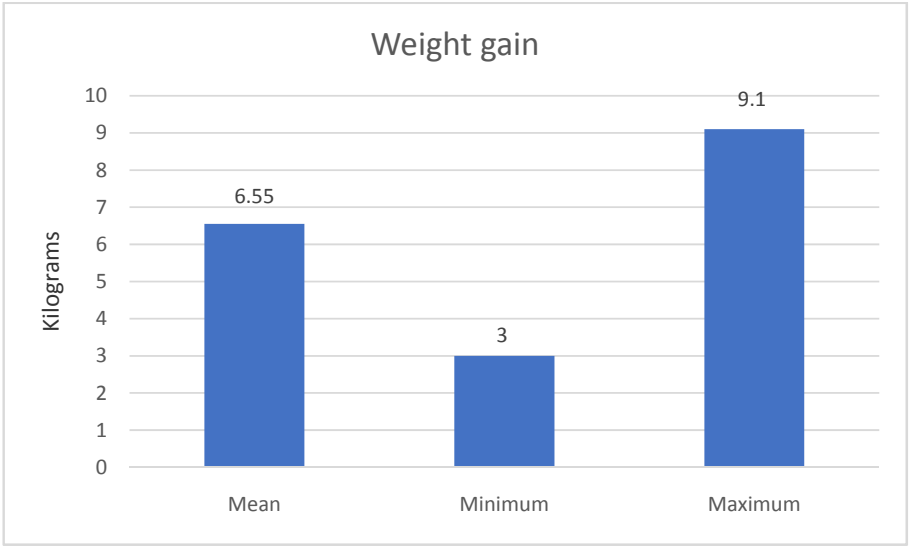
Table 10: Distribution of study subjects according to Risk Factors. (n =62).

Risk factors	n (%)
Anaemia	36 (58.06)
Gestational hypertension (G.Htn)	34 (54.84)
Sickling	23 (37.10)
Gestational diabetes (GDM)	12 (19.35)
Previous intra uterine growth retardation	10(16.13)
Jaundice	02 (3.23)

Table 11: Mean, Standard Deviation, Minimum, Maximum values of Weight Gain in study subjects. (n =62).

Weight gain (Kg)	
Mean	6.55
Standard deviation	1.51
Minimum	3.00
Maximum	9.10

Figure 10: Mean, Minimum, Maximum values of Weight Gain in study subjects.

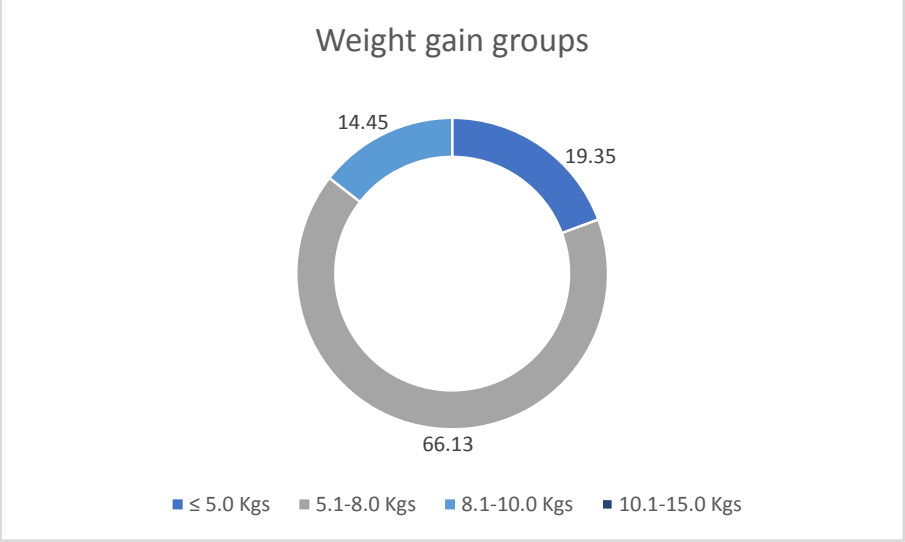


The above table and figure show mean, minimum and maximum values of weight gain in study subjects (n =62). Mean ± SD of weight gain of study subjects was 6.55 Kgs ± 1.51 Kgs. Minimum and maximum weight gain of study subjects were 3.00 Kgs and 9.10 Kgs respectively.

Table 12: Distribution of study subjects in different Weight Gain groups.

Weight gain groups	n (%)
≤ 5.0 Kgs	12 (19.35)
5.1-8.0 Kgs	41 (66.13)
8.1-10 Kgs	09 (14.52)
10.1-15 Kgs	00 (0.00)
Total	**62 (100.00)**

Figure 11: Distribution of study subjects in different Weight Gain groups.

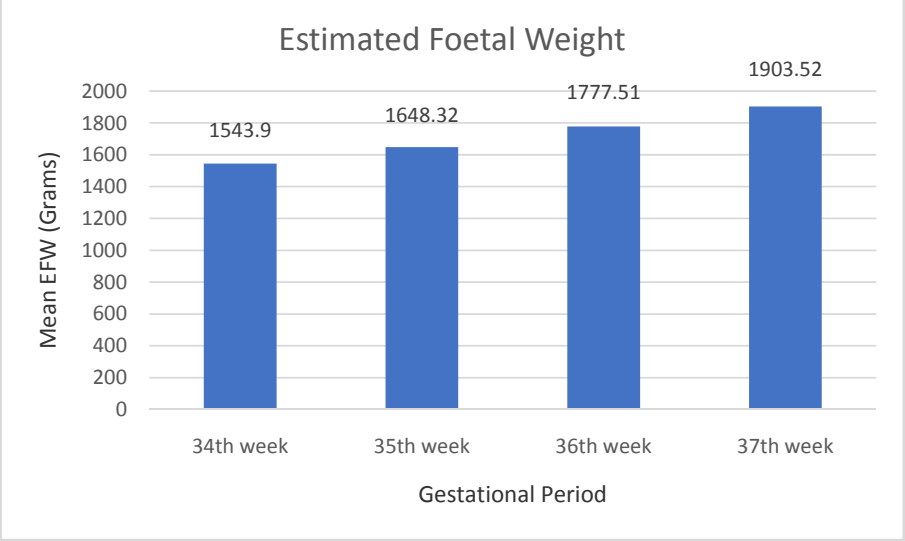


The above table and figure show distribution of study subjects in different weight gain groups. There were 12 (19.35%), 41 (66.13%) and 09 (14.52%) subjects in less than or equal to 5.0 Kgs, 5.1-8.0 Kgs and 8.1-10 Kgs weight gain groups. None of the subjects were in 10.1-15 Kgs weight gain group. Maximum study subjects (n = 41, 66.13%) were in 5.1-8.0 Kgs weight gain group.

Table 13: Mean, Standard Deviation, Minimum, Maximum values of Estimated Foetal Weight (EFW) in study subjects at different weeks of gestation.

Estimated Foetal Weight (EFW, in gm)	Gestation period			
	34th week	**35th week**	**36th week**	**37th week**
Mean	1543.90	1648.32	1777.51	1903.52
Standard deviation	59.01	67.54	65.82	68.35
Minimum	1468.00	1536.00	1678.00	1784.00
Maximum	1650.00	1766.00	1890.00	1990.00

Figure 12: Mean values of Estimated Foetal Weight (EFW) in study subjects at different weeks of gestation.



The above table and figure show mean, standard deviation, minimum and maximum values of Estimated Foetal Weight (EFW) in study subjects at different weeks of gestation.

At 34th week (n = 62, 100.00%), mean ± SD of EFW in study subjects was 1543.90 gms ± 59.01 gms. Minimum and maximum EFW in study subjects were 1468.00 gms and 1650.00 gms.

At 35th week (n = 59, 100.00%), mean ± SD of EFW in study subjects was 1648.32 gms ± 67.54 gms. Minimum and maximum EFW in study subjects were 1536.00 gms and 1766.00 gms.

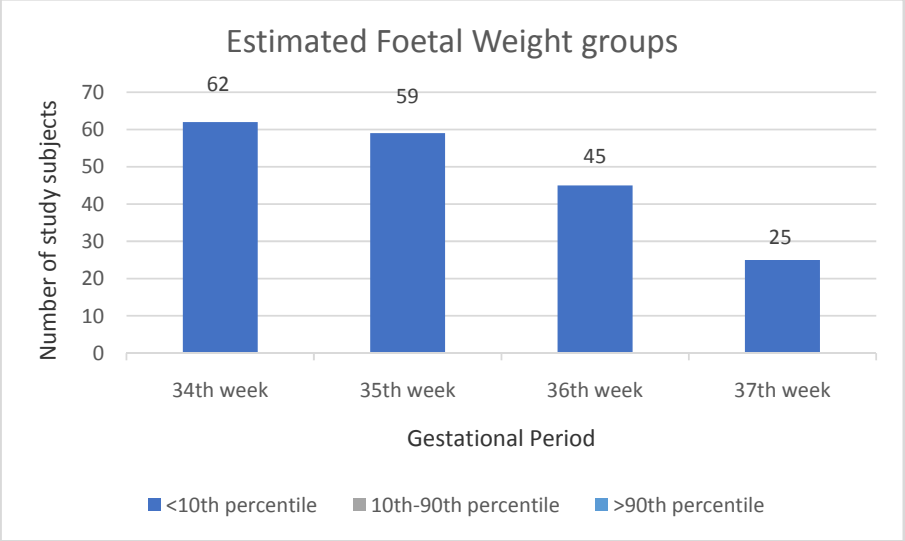
At 36th week (n = 45, 100.00%), mean ± SD of EFW in study subjects was 1777.51 gms ± 65.82 gms. Minimum and maximum EFW in study subjects were 1678.00 gms and 1890.00 gms.

At 37th week (n = 25, 100.00%), mean ± SD of EFW in study subjects was 1903.52 gms ± 68.35 gms. Minimum and maximum EFW in study subjects were 1784.00 gms and 1990.00 gms.

Table 14: Distribution of study subjects in different Estimated Foetal Weight (EFW) groups at different weeks of gestation.

Gestation period	Estimated Foetal Weight (EFW) groups			Total n (%)
	<10th percentile n (%)	10th-90th percentile n (%)	>90th percentile n (%)	
34th week	62 (100.00)	00 (0.00)	00 (0.00)	62 (100.00)
35th week	59 (100.00)	00 (0.00)	00 (0.00)	59 (100.00)
36th week	45 (100.00)	00 (0.00)	00 (0.00)	45 (100.00)
37th week	25 (100.00)	00 (0.00)	00 (0.00)	25 (100.00)

Figure 13: Distribution of study subjects in different Estimated Foetal Weight (EFW) groups at different weeks of gestation.



The above table and figure show distribution of study subjects in different Estimated Foetal Weight (EFW) groups at different weeks of gestation.

At 34th week (n = 62, 100.00%), all the subjects (n = 62, 100.00%) subjects were in less than 10th percentile category of EFW. None of the subjects were in 10th-90th percentile and more than 90th percentile category of EFW.

At 35th week (n = 59, 100.00%), all the subjects (n = 59, 100.00%) subjects were in less than 10th percentile category of EFW. None of the subjects were in 10th-90th percentile and more than 90th percentile category of EFW.

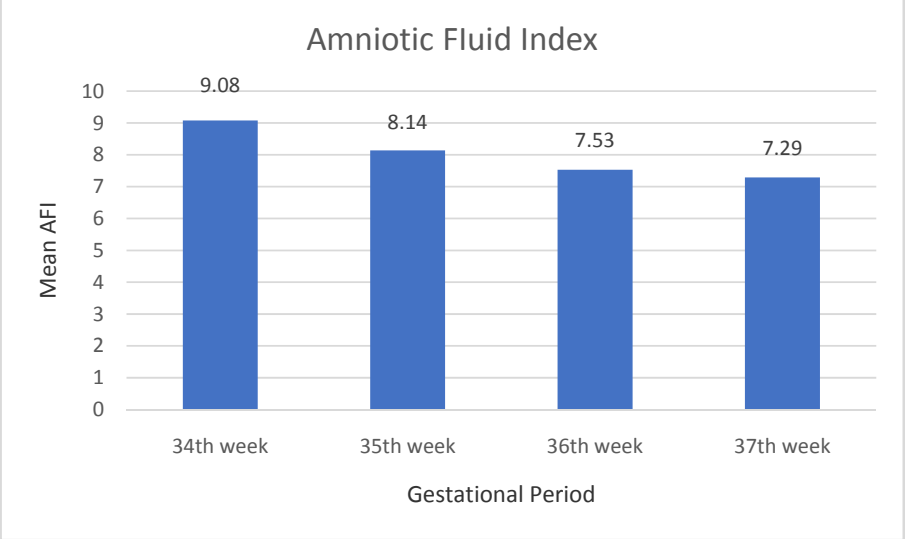
At 36th week (n = 45, 100.00%), all the subjects (n = 45, 100.00%) subjects were in less than 10th percentile category of EFW. None of the subjects were in 10th-90th percentile and more than 90th percentile category of EFW.

At 37th week (n = 25, 100.00%), all the subjects (n = 25, 100.00%) subjects were in less than 10th percentile category of EFW. None of the subjects were in 10th-90th percentile and more than 90th percentile category of EFW.

Table 15: Mean, Standard Deviation, Minimum, Maximum values of Amniotic Fluid Index (AFI) in study subjects at different weeks of gestation.

Amniotic Fluid Index (AFI)	Gestation period			
	34th week	**35th week**	**36th week**	**37th week**
Mean	9.80	8.14	7.53	7.29
Standard deviation	2.29	2.42	1.94	1.88
Minimum	3.60	3.60	4.20	2.80
Maximum	14.30	12.80	10.40	9.60

Figure 14: Mean values of Amniotic Fluid Index (AFI) in study subjects at different weeks of gestation.



The above table and figure show mean, standard deviation, minimum and maximum values of Amniotic Fluid Index (AFI) in study subjects at different weeks of gestation.

At 34th week (n = 62, 100.00%), mean ± SD of AFI in study subjects was 9.80 ± 2.29. Minimum and maximum AFI in study subjects were 3.60 and 14.30.

At 35th week (n = 59, 100.00%), mean ± SD of AFI in study subjects was 8.14 ± 2.42. Minimum and maximum AFI in study subjects were 3.60 and 12.80.

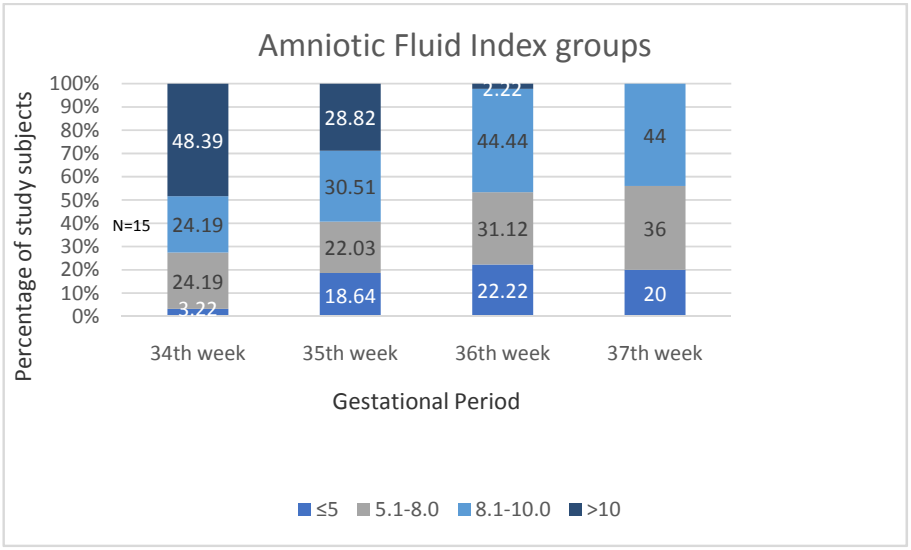
At 36th week (n = 45, 100.00%), mean ± SD of AFI in study subjects was 7.53 ± 1.94. Minimum and maximum AFI in study subjects were 4.20 and 10.40.

At 37th week (n = 25, 100.00%), mean ± SD of AFI in study subjects was 7.28 ± 1.88. Minimum and maximum AFI in study subjects were 2.80 and 9.60.

Table 16: Distribution of study subjects according to Amniotic Fluid Index (AFI) groups in study subjects at different weeks of gestation.

Gestation period	Amniotic Fluid Index (AFI) groups				Total n (%)
	≤5 n (%)	5.1-8.0 n (%)	8.1-10.0 n (%)	>10 n (%)	
34th week	02 (3.22)	15 (24.19)	15 (24.19)	30 (48.39)	62 (100.00)
35th week	11 (18.64)	13 (22.03)	18 (30.51)	17 (28.82)	59 (100.00)
36th week	10 (22.22)	14 (31.12)	20 (44.44)	01 (2.22)	45 (100.00)
37th week	05 (20.00)	09 (36.00)	11 (44.00)	00 (0.00)	25 (100.00)

Figure 15: Distribution of study subjects according to Amniotic Fluid Index (AFI) groups in study subjects at different weeks of gestation.



The above table and figure show distribution of study subjects in different Amniotic Fluid Index (AFI) groups at different weeks of gestation.

At 34th week (n = 62, 100.00%), 02 (3.22%), 15 (24.19%), 15 (24.19%) and 30 (48.39%) study subjects were in less than or equal to 5, 5.1-8.0, 8.1-10.0 and more than 10 AFI group, respectively. Maximum number of subjects (n = 30, 48.39%) were in more than 10 AFI group.

At 35th week (n = 59, 100.00%), 11 (18.64%), 13 (22.03%), 18 (30.51%) and 17 (28.82) study subjects were in less than or equal to 5, 5.1-8.0, 8.1-10.0 and more than 10 AFI group, respectively. Maximum number of subjects (n = 18, 30.51%) were in 8.1-10.0 AFI group.

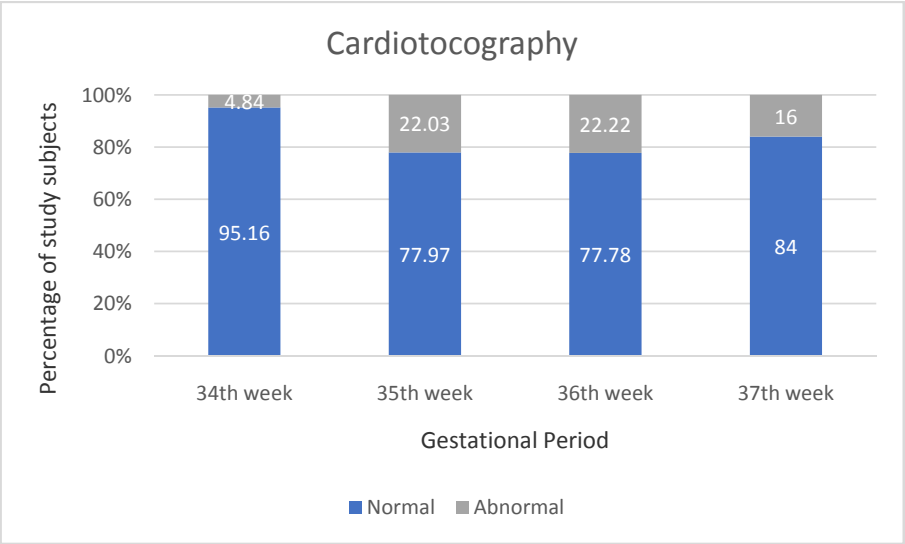
At 36th week (n = 45, 100.00%), 10 (22.22%), 14 (31.12%), 20 (44.44%) and 01 (2.22%) study subjects were in less than or equal to 5, 5.1-8.0, 8.1-10.0 and more than 10 AFI group, respectively. Maximum number of subjects (n = 20, 44.44%) were in 8.1-10.0 AFI group.

At 37th week (n = 25, 100.00%), 05 (20.00%), 09 (36.00%) and 11 (44.00%) study subjects were in less than or equal to 5, 5.1-8.0 and 8.1-10.0 respectively. None of the subjects were in more than 10 AFI group. Maximum number of subjects (n = 11, 44.00%) were in 8.1-10.0 AFI group.

Table 17: Distribution of study subjects according to Cardiotocography (CTG) in study subjects at different weeks of gestation.

Gestation period	Cardiotocography (CTG)		Total n (%)
	Normal n (%)	Abnormal n (%)	
34th week	59 (95.16)	03 (4.84)	62 (100.00)
35th week	46 (77.97)	13 (22.03)	59 (100.00)
36th week	35 (77.78)	10 (22.22)	45 (100.00)
37th week	21 (84.00)	04 (16.00)	25 (100.00)

Figure 16: Distribution of study subjects according to Cardiotocography (CTG) in study subjects at different weeks of gestation.



The above table and figure show distribution of study subjects according to Cardiotocography (CTG) at different weeks of gestation. Number of study subjects available at 34th week, 35th week, 36th week and 37th week were 62, 59, 45 and 25.

At 34th week (n = 62, 100.00%), 59 (95.16%) subjects were Normal CTG and 03 (4.84%) were Abnormal CTG.

At 35th week (n = 59, 100.00%), 46 (77.97%) subjects were Normal CTG and 13 (22.03%) were Abnormal CTG.

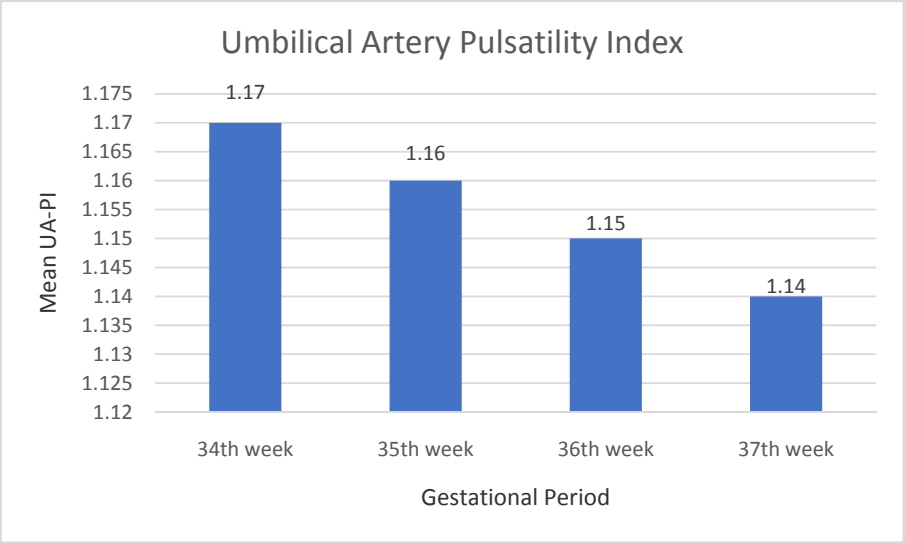
At 36th week (n = 45, 100.00%), 35 (77.78%) subjects were Normal CTG and 10 (22.22%) were Abnormal CTG.

At 37th week (n = 25, 100.00%), 21 (84.00%) subjects were Normal CTG and 04 (16.00%) were Abnormal CTG.

Table 18: Mean, Standard Deviation, Minimum, Maximum values of Umbilical Artery Pulsatility Index (UA-PI) in study subjects at different weeks of gestation.

Umbilical Artery Pulsatility Index (UA-PI)	Gestation period			
	34th week	**35th week**	**36th week**	**37th week**
Mean	1.17	1.16	1.15	1.14
Standard deviation	0.05	0.06	0.06	0.07
Minimum	1.01	0.98	0.96	0.92
Maximum	1.22	1.32	1.20	1.19

Figure 17: Mean values of Umbilical Artery Pulsatility Index (UA-PI) in study subjects at different weeks of gestation.



The above table and figure show mean, minimum and maximum values of Umbilical Artery Pulsatility Index (UA-PI) in study subjects at different weeks of gestation.

At 34th week (n = 62, 100.00%), mean ± SD of UA-PI in study subjects was 1.17 ± 0.05 Minimum and maximum UA-PI in study subjects were 1.01and 1.22.

At 35th week (n = 59, 100.00%), mean ± SD of UA-PI in study subjects was 1.16 ± 0.06. Minimum and maximum UA-PI in study subjects were 0.98and 1.32.

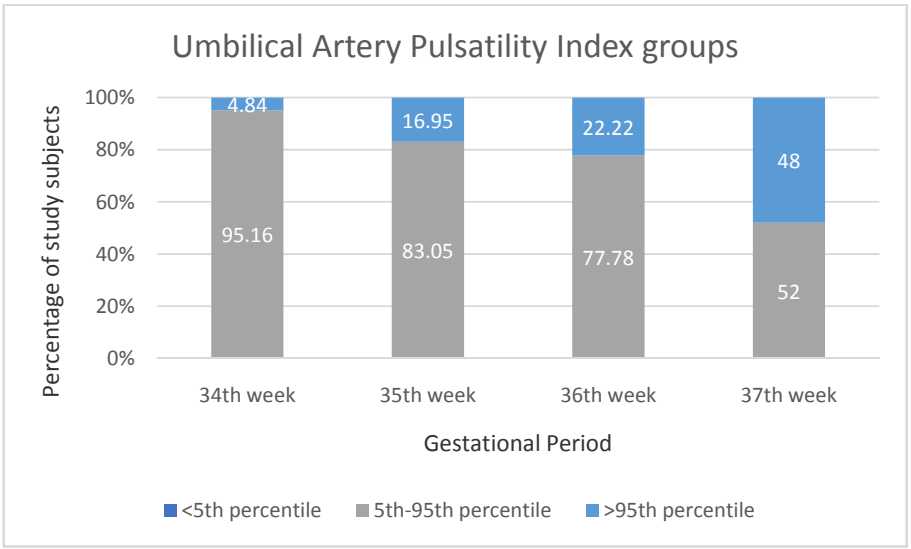
At 36th week (n = 45, 100.00%), mean ± SD of UA-PI in study subjects was 1.15 ± 0.06. Minimum and maximum UA-PI in study subjects were 0.96 and 1.20.

At 37th week (n = 25, 100.00%), mean ± SD of UA-PI in study subjects was 1.14 ± 0.07. Minimum and maximum UA-PI in study subjects were 0.92 and 1.19.

Table 19: Distribution of study subjects according to Umbilical Artery Pulsatility Index (UA-PI) groups in study subjects at different weeks of gestation.

Gestation period	Umbilical Artery Pulsatility Index (UA-PI) groups			Total n (%)
	<5th percentile n (%)	5th-95th percentile n (%)	>95th percentile n (%)	
34th week	00 (0.00)	59 (95.16)	03 (4.84)	62 (100.00)
35th week	00 (0.00)	49 (83.05)	10 (16.95)	59 (100.00)
36th week	00 (0.00)	35 (77.78)	10 (22.22)	45 (100.00)
37th week	00 (0.00)	13 (52.00)	12 (48.00)	25 (100.00)

Figure 18: Distribution of study subjects according to Umbilical Artery Pulsatility Index (UA-PI) groups in study subjects at different weeks of gestation.



The above table and figure show distribution of study subjects in different Umbilical Artery Pulsatility Index (UA-PI) groups at different weeks of gestation.

At 34th week (n = 62, 100.00%), 59 (95.16%) subjects were in 5th-95th percentile and 03 (4.84%) were in more than 95th percentile category of UA-PI. None of the subjects were in less than 5th percentile category of UA-PI.

At 35th week (n = 59, 100.00%), 49 (83.05%) subjects were in 5th-95th percentile and 10 (16.95%) were in more than 95th percentile category of UA-PI. None of the subjects were in less than 5th percentile category of UA-PI.

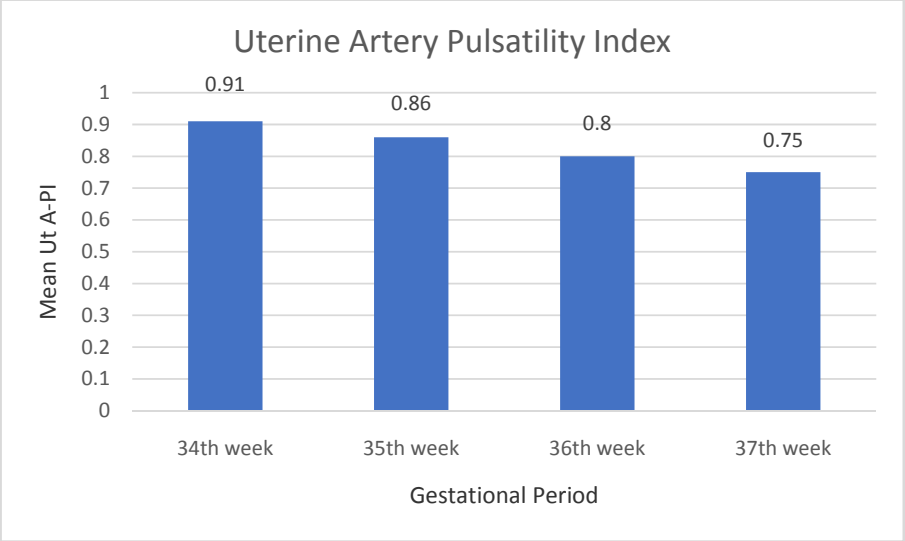
At 36th week (n = 45, 100.00%), 35 (77.78%) subjects were in 5th-95th percentile and 10 (22.22%) were in more than 95th percentile category of UA-PI. None of the subjects were in less than 5th percentile category of UA-PI.

At 37th week (n = 25, 100.00%), 13 (52.00%) subjects were in 5th-95th percentile and 12 (48.00%) were in more than 95th percentile category of UA-PI. None of the subjects were in less than 5th percentile category of UA-PI.

Table 20: Mean, Standard Deviation, Minimum, Maximum values of Uterine Artery Pulsatility Index (Ut A-PI) in study subjects at different weeks of gestation.

Uterine Artery Pulsatility Index (Ut A-PI)	**Gestation period**			
	34th week	**35th week**	**36th week**	**37th week**
Mean	0.91	0.86	0.80	0.75
Standard deviation	0.17	0.12	0.08	0.06
Minimum	0.73	0.71	0.70	0.67
Maximum	1.45	1.24	1.18	0.92

Figure 19: Mean values of Uterine Artery Pulsatility Index (Ut A-PI) in study subjects at different weeks of gestation.



The above table and figure show mean, standard deviation (SD), minimum and maximum values of Uterine Artery Pulsatility Index (Ut A-PI) in study subjects at different weeks of gestation.

At 34th week (n = 62, 100.00%), mean ± SD of Ut A-PI in study subjects was 0.91 ± 0.17. Minimum and maximum Ut A-PI in study subjects were 0.73and 1.45.

At 35th week (n = 59, 100.00%), mean ± SD of Ut A-PI in study subjects was 0.86 ± 0.12. Minimum and maximum Ut A-PI in study subjects were 0.71and 1.24.

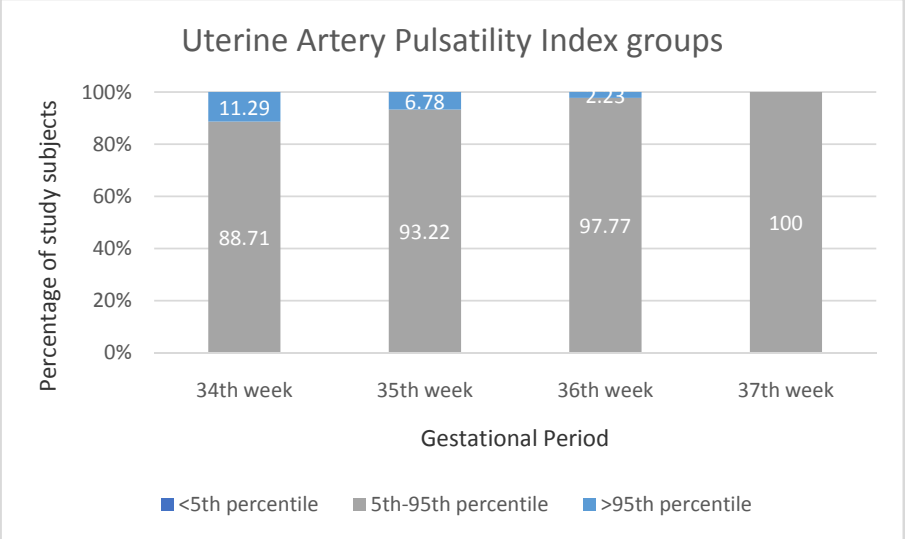
At 36th week (n = 45, 100.00%), mean ± SD of Ut A-PI in study subjects was 0.80 ± 0.08. Minimum and maximum Ut A-PI in study subjects were 0.70 and 1.18.

At 37th week (n = 25, 100.00%), mean ± SD of Ut A-PI in study subjects was 0.75 ± 0.06. Minimum and maximum Ut A-PI in study subjects were 0.67 and 0.92.

Table 21: Distribution of study subjects according to Uterine Artery Pulsatility Index (Ut A-PI) groups in study subjects at different weeks of gestation.

Gestation period	Uterine Artery Pulsatility Index (Ut A-PI) groups			Total n (%)
	<5th percentile n (%)	5th-95th percentile n (%)	>95th percentile n (%)	
34th week	00 (0.00)	55 (88.71)	07 (11.29)	62 (100.00)
35th week	00 (0.00)	55 (93.22)	04 (6.78)	59 (100.00)
36th week	00 (0.00)	44(97.77)	01 (2.23)	45 (100.00)
37th week	00 (0.00)	25 (100.00)	00 (0.00)	25 (100.00)

Figure 20: Distribution of study subjects according to Uterine Artery Pulsatility Index (Ut A-PI) groups in study subjects at different weeks of gestation.



The above table and figure show distribution of study subjects in different Uterine Artery Pulsatility Index (Ut A-PI) groups at different weeks of gestation.

At 34th week (n = 62, 100.00%), 55 (88.71%) subjects were in 5th-95th percentile and 07 (11.29%) were in more than 95th percentile category of Ut A-PI. None of the subjects were in less than 5th percentile category of Ut A-PI.

At 35th week (n = 59, 100.00%), 55 (93.22%) subjects were in 5th-95th percentile and 04 (6.78%) were in more than 95th percentile category of Ut A-PI. None of the subjects were in less than 5th percentile category of Ut A-PI.

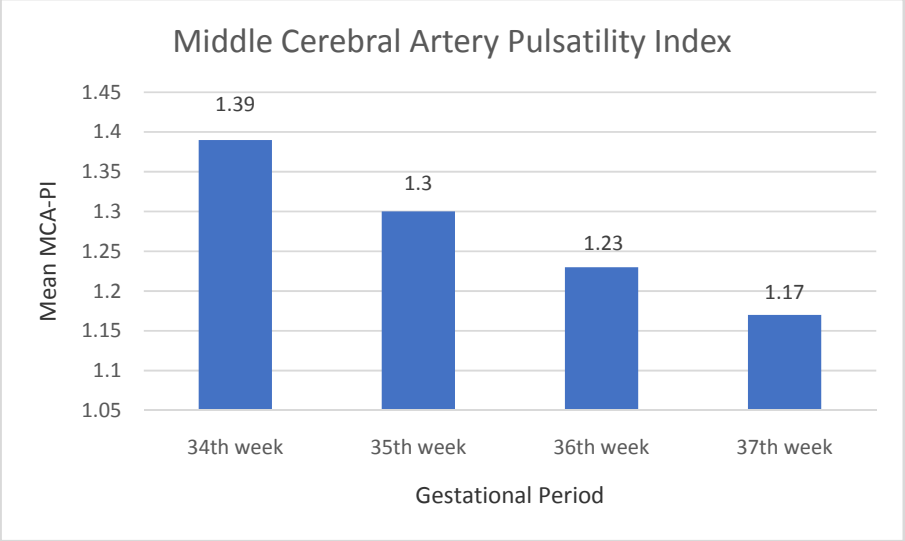
At 36th week (n = 45, 100.00%), 44 (97.77%) subjects were in 5th-95th percentile and 01 (2.23%) were in more than 95th percentile category of Ut A-PI. None of the subjects were in less than 5th percentile category of Ut A-PI.

At 37th week (n = 25, 100.00%), all the subjects (n = 25, 100.00%) subjects were in 5th-95th percentile category of Ut A-PI. None of the subjects were in less than 5th percentile and more than 95th percentile category of Ut A-PI.

Table 22: Mean, Standard Deviation, Minimum, Maximum values of Middle Cerebral Artery Pulsatility Index (MCA-PI) in study subjects at different weeks of gestation

Middle Cerebral Artery Pulsatility Index (MCA-PI)	Gestation period			
	34th week	**35th week**	**36th week**	**37th week**
Mean	1.39	1.30	1.23	1.17
Standard deviation	0.13	0.08	0.07	0.06
Minimum	1.02	1.18	1.08	1.02
Maximum	1.82	1.65	1.48	1.27

Figure 21: Mean values of Middle Cerebral Artery Pulsatility Index (MCA-PI) in study subjects at different weeks of gestation.



The above table and figure shows mean, standard deviation, minimum and maximum values of Middle Cerebral Artery Pulsatility Index (MCA-PI) in study subjects at different weeks of gestation.

At 34th week (n = 62, 100.00%), mean ± SD of MCA-PI in study subjects was 1.39 ± 0.13. Minimum and maximum MCA-PI in study subjects were 1.02 and 1.82.

At 35th week (n = 59, 100.00%), mean ± SD of MCA-PI in study subjects was 1.30 ± 0.08. Minimum and maximum MCA-PI in study subjects were 1.18 and 1.65.

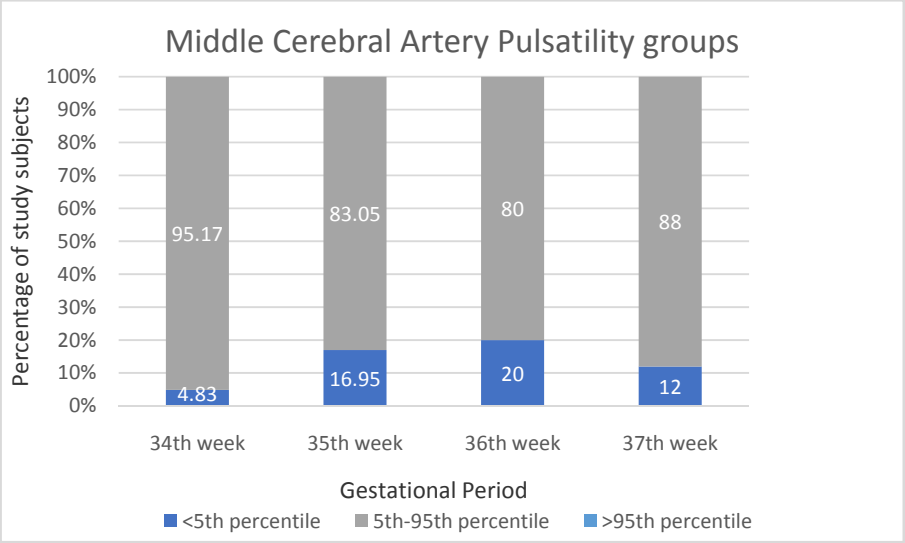
At 36th week (n = 45, 100.00%), mean ± SD of MCA-PI in study subjects was 1.23 ± 0.07. Minimum and maximum MCA-PI in study subjects were 1.08 and 1.48.

At 37th week (n = 25, 100.00%), mean ± SD of MCA-PI in study subjects was 1.17 ± 0.06. Minimum and maximum MCA-PI in study subjects were 1.03 and 1.27.

Table 23: Distribution of study subjects according to Middle Cerebral Artery Pulsatility Index (MCA-PI) groups in study subjects at different weeks of gestation.

Gestation period	Middle Cerebral Artery Pulsatility Index (MCA-PI) groups			Total n (%)
	<5th percentile n (%)	5th-95th percentile n (%)	>95th percentile n (%)	
34th week	03 (4.83)	59 (95.17)	00 (0.00)	62 (100.00)
35th week	10 (16.95)	49 (83.05)	00 (0.00)	59 (100.00)
36th week	9 (20)	36 (80)	00 (0.00)	45 (100.00)
37th week	03 (12.00)	22 (88.00)	00 (0.00)	25 (100.00)

Figure 22: Distribution of study subjects according to Middle Cerebral Artery Pulsatility Index (MCA-PI) groups in study subjects at different weeks of gestation.



The above table and figure show distribution of study subjects in different Middle Cerebral Artery Pulsatility Index (MCA-PI) groups at different weeks of gestation.

At 34th week (n = 62, 100.00%), 03 (4.83%) subjects were in less than 5th percentile and 59 (95.17%) were in 5th-95th percentile category of MCA-PI. None of the subjects were in more than 95th percentile category of MCA-PI.

At 35th week (n = 59, 100.00%), 10 (16.95%) subjects were in less than 5th percentile and 49 (83.05%) were in 5th-95th percentile category of MCA-PI. None of the subjects were in more than 95th percentile category of MCA-PI.

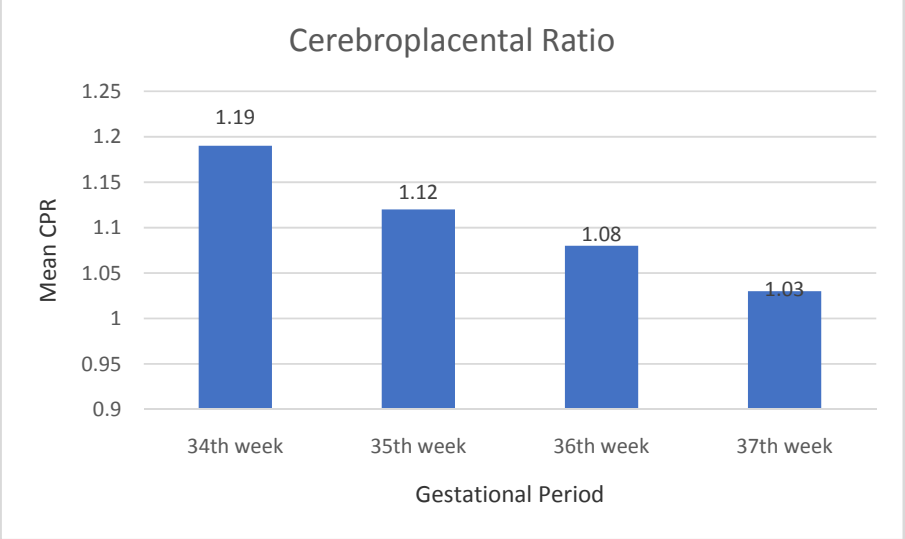
At 36th week (n = 45, 100.00%), 9 (20%) subjects were in less than 5th percentile and 36 (80%) were in 5th-95th percentile category of MCA-PI. None of the subjects were in more than 95th percentile category of MCA-PI.

At 37th week (n = 25, 100.00%), 03 (12%) subjects were in less than 5th percentile and 22 (84.00%) were in 5th-95th percentile category of MCA-PI. None of the subjects were in more than 95th percentile category of MCA-PI.

Table 24: Mean, Standard Deviation, Minimum, Maximum values of Cerebroplacental Ratio (CPR) in study subjects at different weeks of gestation.

Cerebroplacental Ratio (CPR)	Gestation period			
	34th week	**35th week**	**36th week**	**37th week**
Mean	1.19	1.12	1.08	1.03
Standard deviation	0.11	0.08	0.07	0.06
Minimum	0.95	0.98	0.98	0.95
Maximum	1.50	1.38	1.29	1.25

Figure 23: Mean values of Cerebroplacental Ratio (CPR) in study subjects at different weeks of gestation.



The above table and figure show mean, standard deviation, minimum and maximum values of Cerebroplacental Ratio (CPR) in study subjects at different weeks of gestation.

At 34th week (n = 62, 100.00%), mean ± SD of CPR in study subjects was 1.19 ± 0.11. Minimum and maximum CPR in study subjects were 0.95 and 1.50.

At 35th week (n = 59, 100.00%), mean ± SD of CPR in study subjects was 1.12 ± 0.08. Minimum and maximum CPR in study subjects were 0.98 and 1.38.

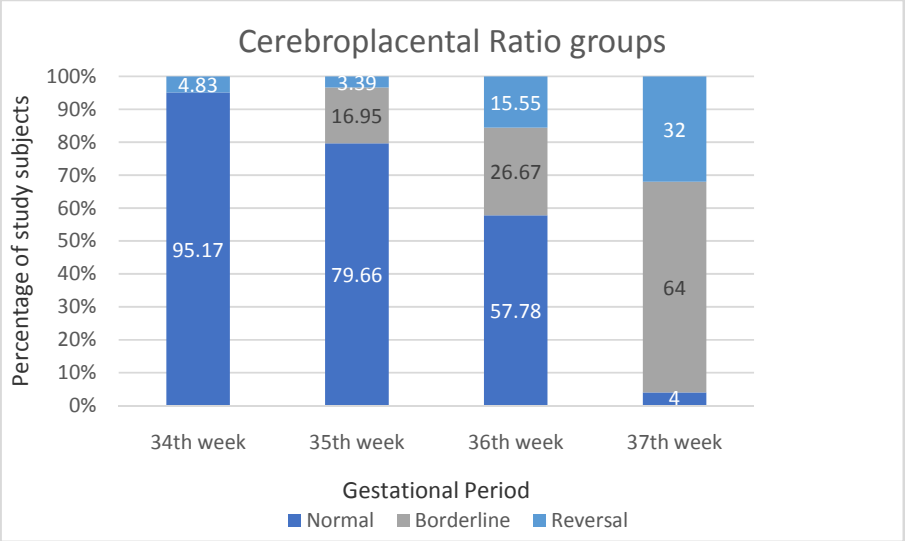
At 36th week (n = 45, 100.00%), mean ± SD of CPR in study subjects was 1.08 ± 0.07. Minimum and maximum CPR in study subjects were 0.98 and 1.29.

At 37th week (n = 25, 100.00%), mean ± SD of CPR in study subjects was 1.03 ± 0.06. Minimum and maximum CPR in study subjects were 0.95 and 1.25.

Table 25: Distribution of study subjects according to Cerebroplacental Ratio (CPR) groups in study subjects at different weeks of gestation.

Gestation period	Cerebroplacental Ratio (CPR) groups			Total n (%)
	Normal n (%)	Borderline n (%)	Reversal n (%)	
34th week	59 (95.17)	00 (00)	03 (4.83)	62 (100.00)
35th week	47 (79.66)	10 (16.95)	02 (3.39)	59 (100.00)
36th week	26 (57.78)	11 (26.67)	07 (15.55)	45 (100.00)
37th week	01 (4.00)	16 (64.00)	08 (32.00)	25 (100.00)

Figure 24: Distribution of study subjects according to Cerebroplacental Ratio (CPR) groups in study subjects at different weeks of gestation.



The above table and figure show distribution of study subjects in different Cerebroplacental Ratio (CPR) groups at different weeks of gestation.

At 34th week (n = 62, 100.00%), 59 (95.17%), 00 (0.00%) and 03 (4.83%) subjects were in normal, borderline and reversal CPR groups, respectively.

At 35th week (n = 59, 100.00%), 47 (79.66%), 10 (16.95%) and 02 (3.39%) subjects were in normal, borderline and reversal CPR groups, respectively.

At 36th week (n = 45, 100.00%), 26 (57.78%), 11 (26.67%) and 07 (15.55%) subjects were in normal, borderline and reversal CPR groups, respectively.

At 37th week (n = 25, 100.00%), 01 (4.00%), 16 (64.00%) and 07 (32.00%) subjects were in normal, borderline and reversal CPR groups, respectively.

Table 26: Association between Maternal Morbidity and Mode of Delivery among subjects who delivered at 34th, 35th, 36th and 37th weeks of gestation.

Gestation period	Mode of delivery	Maternal morbidity		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
34th week	Emergency LSCS	02 (66.67)	01 (33.33)	03 (100.00)	Test not applicable
	Elective LSCS	00 (0.00)	00 (0.00)	00 (0.00)	
	Vaginal	00 (0.00)	00 (0.00)	00 (0.00)	
35th week	Emergency LSCS	03 (23.08)	10 (76.92)	13 (100.00)	$\chi^2 = 0.294$, df = 1, P =0.588 (>0.05), (>0.05), Not significant
	Elective LSCS	00 (0.00)	01 (100.00)	01 (100.00)	
	Vaginal	00 (0.00)	00 (0.00)	00 (0.00)	
36th week	Emergency LSCS	03 (30.00)	07 (70.00)	10 (100.00)	$\chi^2 = 0.304$, df = 2, P =0.859 (>0.05), (>0.05), Not significant
	Elective LSCS	01 (33.33)	02 (66.67)	03 (100.00)	
	Vaginal	03 (42.86)	04 (57.14)	07 (100.00)	
37th week	Emergency LSCS	02 (28.57)	05 (71.43)	07 (100.00)	$\chi^2 = 1.071$, df = 2, P =0.585, (>0.05), Not significant
	Elective LSCS	00 (0.00)	03 (100.00)	03 (100.00)	
	Vaginal	03 (20.00)	12 (80.00)	15 (100.00)	

Fever and wound gap were considered in Maternal morbidity. Other causes of morbidity such as abdominal distention, burst abdomen, septicaemia were not observed in our study.

All the cases at 34th week were under emergency LSCS. Hence, no test of significance was applicable.

There was no significant association between maternal morbidity and mode of delivery among subjects who delivered at 35th week (χ^2 = 0.294, df = 2, P >0.05).

There was no significant association between maternal morbidity and mode of delivery among subjects who delivered at 36th week (χ^2 = 0.304, df = 2, P >0.05).

There was no significant association between maternal morbidity and mode of delivery among subjects who delivered at 37th week (χ^2 = 1.071, df = 2, P >0.05).

Table 27: Comparison of maternal morbidity between 34th, 35th, 36th and 37th weeks of gestation.

Gestation period	**Maternal morbidity**		**Total** n (%)
	Yes n (%)	**No** n (%)	
34th week	02 (66.67)	01 (33.33)	03 (100.00)
35th week	03 (21.43)	11 (78.57)	14 (100.00)
36th week	07 (35.00)	13 (65.00)	20 (100.00)
37th week	05 (20.00)	20 (80.00)	25 (100.00)
Chi-square test	χ^2 = 3.843, df = 3, P =0.279 (>0.05), Not significant		

There was no significant difference for maternal morbidity between 34th, 35th, 36th and 37th weeks (χ^2 = 3.843, df = 3, P >0.05).

Table 28: Association of various parameters with neonatal morbidity at 34th week of gestation (n = 03).

Parameters	Categories	Neonatal morbidity		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
Amniotic Fluid Index (AFI)	≤ 5	02 (100.00)	00 (0.00)	02 (100.00)	Not applicable
	>5	01 (100.00)	00 (0.00)	01 (100.00)	
Cardiotocography (CTG)	Abnormal	03 (100.00)	00 (0.00)	03 (100.00)	Not applicable
	Normal	00 (0.00)	00 (0.00)	00 (0.00)	
Umbilical Artery Pulsatility Index (UA-PI)	5th – 95th percentile	03 (100.00)	00 (0.00)	03 (100.00)	Not applicable
	>95th percentile	00 (0.00)	00 (0.00)	00 (0.00)	
Uterine Artery Pulsatility Index (Ut A-PI)	5th -95th percentile	00 (0.00)	00 (0.00)	00 (0.00)	Not applicable
	>95th percentile	03 (100.00)	00 (0.00)	03 (100.00)	
Middle Cerebral Artery Pulsatility Index (MCA-PI)	<5th percentile	03 (100.00)	00 (0.00)	03 (100.00)	Not applicable
	≥5th percentile	00 (0.00)	00 (0.00)	00 (0.00)	
Cerebroplacental Ratio (CPR)	<1.08 (Altered)	03 (100.00)	00 (0.00)	03 (100.00)	Not applicable
	≥1.08 (Normal)	00 (0.00)	00 (0.00)	00 (0.00)	
APGAR Score at 1 min	< 7 (Abnormal)	03 (100.00)	00 (00.00)	03 (100.00)	Not applicable
	≥7 (Normal)	00 (0.00)	00 (0.00)	00 (0.00)	
APGAR Score at 5 min	< 7 (Abnormal)	03 (100.00)	00 (00.00)	03 (100.00)	Not applicable
	≥7 (Normal)	00 (0.00)	00 (0.00)	00 (0.00)	

*Neonatal morbidity was present in total 03 (100.00%) cases.

There was no significant association of AFI, CTG, UA-PI, Ut A-PI, MCA-PI, CPR and APGAR with neonatal morbidity at 34th week.

Table 29: Association of various parameters with neonatal mortality at 34th week of gestation (n = 03).

Parameters	Categories	Neonatal mortality		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
Amniotic Fluid Index (AFI)	≤ 5	02 (100.00)	00 (0.00)	02 (100.00)	Not applicable
	>5	01 (100.00)	00 (0.00)	01 (100.00)	
Cardiotocography (CTG)	Abnormal	03 (100.00)	00 (0.00)	03 (100.00)	Not applicable
	Normal	00 (0.00)	00 (0.00)	00 (0.00)	
Umbilical Artery Pulsatility Index (UA-PI)	5th – 95th percentile	03 (100.00)	00 (0.00)	03 (100.00)	Not applicable
	>95th percentile	00 (0.00)	00 (0.00)	00 (0.00)	
Uterine Artery Pulsatility Index (Ut A-PI)	5th -95th percentile	00 (0.00)	00 (0.00)	00 (0.00)	Not applicable
	>95th percentile	03 (100.00)	00 (0.00)	03 (100.00)	
Middle Cerebral Artery Pulsatility Index (MCA-PI)	<5th percentile	03 (100.00)	00 (0.00)	03 (100.00)	Not applicable
	≥5th percentile	00 (0.00)	00 (0.00)	00 (0.00)	
Cerebroplacental Ratio (CPR)	<1.08 (Altered)	03 (100.00)	00 (0.00)	03 (100.00)	Not applicable
	≥1.08 (Normal)	00 (0.00)	00 (0.00)	00 (0.00)	
APGAR Score at 1 min	< 7 (Abnormal)	03 (100.00)	00 (00.00)	03(100.00)	Not applicable
	≥7 (Normal)	00 (0.00)	00 (0.00)	00 (0.00)	
APGAR Score at 5 min	< 7 (Abnormal)	03 (100.00)	00 (00.00)	03(100.00)	Not applicable
	≥7 (Normal)	00 (0.00)	00 (0.00)	00 (0.00)	

*Neonatal mortality was present in total 03 (100.00%) cases.

There was no significant association of AFI, CTG, UA-PI, Ut A-PI, MCA-PI, CPR and APGAR with neonatal mortality at 34th week.

Table 30: Association of various parameters with neonatal morbidity at 35th week of gestation (n = 14).

Parameters	Categories	Neonatal morbidity		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
Amniotic Fluid Index (AFI)	≤ 5	07 (63.64)	04 (36.36)	11 (100.00)	$\chi^2 = 3.818$, df =1, P = 0.051 (>0.05), NS
	>5	00 (0.00)	03 (100.00)	03 (100.00)	
Cardiotocography (CTG)	Abnormal	06 (46.15)	07 (53.85)	13 (100.00)	$\chi^2 = 1.077$, df =1, P = 0.299 (>0.05), NS
	Normal	01 (100.00)	00 (0.00)	01 (100.00)	
Umbilical Artery Pulsatility Index (UA-PI)	5th – 95th percentile	00 (0.00)	00 (0.00)	00 (0.00)	Not applicable
	>95th percentile	01 (25.00)	03 (75.00)	4 (100.00)	
Uterine Artery Pulsatility Index (Ut A-PI)	5th -95th percentile	00 (0.00)	00 (0.00)	00 (0.00)	Not applicable
	>95th percentile	01 (33.33)	02 (66.67)	03 (100.00)	
Middle Cerebral Artery Pulsatility Index (MCA-PI)	<5th percentile	05 (50.00)	05 (50.00)	10 (100.00)	$\chi^2 = 0.000$, df =1, P = 1.000 (>0.05), NS
	≥5th percentile	02 (50.00)	02 (50.00)	04 (100.00)	
Cerebroplacental Ratio (CPR)	<1.08 (Altered)	05 (41.67)	07 (58.33)	12 (100.00)	$\chi^2 = 2.333$, df =1, P = 0.127 (>0.05), NS
	≥1.08 (Normal)	02 (100.00)	00 (0.00)	02 (100.00)	
APGAR Score at 1 min	< 7 (Abnormal)	07 (77.78)	02 (22.22)	09 (100.00)	$\chi^2 = 7.778$, df =1, P = 0.005 (<0.01), HS
	≥7 (Normal)	00 (0.00)	05 (100.00)	05 (100.00)	
APGAR Score at 5 min	< 7 (Abnormal)	01 (100.00)	00 (00.00)	01 (100.00)	$\chi^2 = 1.077$, df =1, P = 0.299 (>0.05), NS
	≥7 (Normal)	06 (46.15)	07 (53.85)	13 (100.00)	

#NS = Not significant, HS = Highly significant

*Neonatal morbidity was present in total 07 (50.00%) cases.

There was no significant association of AFI, CTG, UA-PI, Ut A-PI, MCA-PI, CPR and APGAR scores at 5 min with neonatal morbidity at 35^{th} week.

Morbidity was significantly higher among neonates with abnormal APGAR scores compared with normal APGAR scores at 1 min ($\chi^2 = 7.778$, df =1, P <0.01).

Table 31: Association of various parameters with neonatal mortality at 35th week of gestation (n = 14).

Parameters	Categories	Neonatal mortality		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
Amniotic Fluid Index (AFI)	≤ 5	01 (9.09)	10 (91.91)	11 (100.00)	$\chi^2 = 0.294$, df =1, P = 0.588 (>0.05), NS
	>5	00 (0.00)	03 (100.00)	03 (100.00)	
Cardiotocography (CTG)	Abnormal	01 (7.69)	12 (92.31)	13 (100.00)	$\chi^2 = 0.083$, df =1, P = 0.773 (>0.05), NS
	Normal	00 (0.00)	01 (100.00)	01 (100.00)	
Umbilical Artery Pulsatility Index (UA-PI)	5th – 95th percentile	00 (0.00)	00 (0.00)	00 (0.00)	Not applicable
	>95th percentile	01 (7.14)	13 (92.86)	14 (100.00)	
Uterine Artery Pulsatility Index (Ut A-PI)	5th -95th percentile	00 (0.00)	00 (0.00)	00 (0.00)	Not applicable
	>95th percentile	01 (7.14)	13 (92.86)	14 (100.00)	
Middle Cerebral Artery Pulsatility Index (MCA-PI)	<5th percentile	00 (0.00)	10 (100.00)	10 (100.00)	$\chi^2 = 2.692$, df =1, P = 0.101 (>0.05), NS
	≥5th percentile	01 (25.00)	03 (75.00)	04 (100.00)	
Cerebroplacental Ratio (CPR)	<1.08 (Altered)	01 (8.33)	11 (91.67)	12 (100.00)	$\chi^2 = 0.179$, df =1, P = 0.672 (>0.05), NS
	≥1.08 (Normal)	00 (0.00)	02 (100.00)	02 (100.00)	
APGAR Score at 1 min	< 7 (Abnormal)	01 (11.11)	08 (88.89)	09 (100.00)	$\chi^2 = 0.598$, df =1, P = 0.439 (>0.05), NS
	≥7 (Normal)	00 (0.00)	05 (100.00)	05 (100.00)	
APGAR Score at 5 min	< 7 (Abnormal)	00 (0.00)	00 (0.00)	00 (0.00)	Not applicable
	≥7 (Normal)	01 (7.14)	13 (92.86)	14 (100.00)	

#NS = Not significant

*Neonatal mortality was present in total 01 (7.14%) case.

There was no significant association of AFI, CTG, UA-PI, Ut A-PI, MCA-PI, CPR and APGAR scores with neonatal mortality at 35th week.

Table 32: Association of various parameters with neonatal morbidity at 36th week of gestation (n = 20).

Parameters	Categories	Neonatal morbidity		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
Amniotic Fluid Index (AFI)	≤ 5	03 (33.33)	06 (66.67)	09 (100.00)	χ^2 = 0.900, df =1, P = 0.343 (>0.05), NS
	>5	06 (54.55)	05 (45.45)	11 (100.00)	
Cardiotocography (CTG)	Abnormal	04 (40.00)	06 (60.00)	10 (100.00)	χ^2 = 0.202, df =1, P = 0.653 (>0.05), NS
	Normal	05 (50.00)	05 (50.00)	10 (100.00)	
Umbilical Artery Pulsatility Index (UA-PI)	5th – 95th percentile	05 (35.71)	09 (64.29)	14 (100.00)	χ^2 = 1.626, df =1, P = 0.202 (>0.05), NS
	>95th percentile	04 (66.67)	02 (33.33)	06 (100.00)	
Uterine Artery Pulsatility Index (Ut A-PI)	5th -95th percentile	08 (42.11)	11 (57.89)	19 (100.00)	χ^2 = 1.287, df =1, P = 0.257 (>0.05), NS
	>95th percentile	01 (100.00)	0 (0.00)	01 (100.00)	
Middle Cerebral Artery Pulsatility Index (MCA-PI)	<5th percentile	03 (33.33)	06 (66.67)	09 (100.00)	χ^2 = 0.900, df =1, P = 0.343 (>0.05), NS
	≥5th percentile	06 (54.55)	05 (45.45)	11 (100.00)	
Cerebroplacental Ratio (CPR)	<1.08 (Altered)	09 (50.00)	09 (50.00)	18 (100.00)	χ^2 = 1.818, df =1, P = 0.178 (>0.05), NS
	≥1.08 (Normal)	00 (0.00)	02 (100.00)	02 (100.00)	
APGAR Score at 1 min	< 7 (Abnormal)	09 (60.00)	06 (40.00)	15 (100.00)	χ^2 = 5.455, df =1, P = 0.020 (<0.05), S
	≥7 (Normal)	00 (00.00)	05 (100.00)	5 (100.00)	

APGAR Score at 5 min	< 7 (Abnormal)	02 (100.00)	00 (0.00)	02 (100.00)	χ^2 = 2.716, df =1, P = 0.099 (>0.05), NS
	≥7 (Normal)	07 (38.89)	11 (61.11)	18 (100.00)	

[#]NS = Not significant, S = Significant

*Neonatal morbidity was present in total 09 (45.00%) cases.

There was no significant association of AFI, CTG, UA-PI, Ut A-PI, MCA-PI, CPR and APGAR Score at 5 min with neonatal morbidity at 36th week.

Morbidity was significantly higher among neonates with abnormal APGAR scores compared with normal APGAR scores at 1 min (χ^2 = 5.455, df =1, P <0.05).

Table 33: Association of various parameters with neonatal mortality at 36th week of gestation (n = 20).

Parameters	Categories	Neonatal mortality		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
Amniotic Fluid Index (AFI)	≤ 5	00 (0.00)	09 (100.00)	09 (100.00)	Not applicable
	>5	00 (0.00)	11 (100.00)	11 (100.00)	
Cardiotocography (CTG)	Abnormal	00 (0.00)	10 (100.00)	10 (100.00)	Not applicable
	Normal	00 (0.00)	10 (100.00)	10 (100.00)	
Umbilical Artery Pulsatility Index (UA-PI)	5th – 95th percentile	00 (0.00)	00 (0.00)	14 (100.00)	Not applicable
	>95th percentile	00 (0.00)	20 (100.00)	06 (100.00)	
Uterine Artery Pulsatility Index (Ut A-PI)	5th -95th percentile	00 (0.00)	00 (0.00)	19 (100.00)	Not applicable
	>95th percentile	00 (0.00)	20 (100.00)	01 (100.00)	
Middle Cerebral Artery Pulsatility Index (MCA-PI)	<5th percentile	00 (0.00)	10 (100.00)	09 (100.00)	Not applicable
	≥5th percentile	00 (0.00)	10 (100.00)	11 (100.00)	
Cerebroplacental Ratio (CPR)	<1.08 (Altered)	00 (0.00)	18 (100.00)	18 (100.00)	Not applicable
	≥1.08 (Normal)	00 (0.00)	02 (100.00)	02 (100.00)	
APGAR Score at 1 min	< 7 (Abnormal)	00 (0.00)	15 (100.00)	15 (100.00)	Not applicable
	≥7 (Normal)	00 (0.00)	05 (100.00)	05 (100.00)	
APGAR Score at 5 min	< 7 (Abnormal)	00 (0.00)	02 (100.00)	02 (100.00)	Not applicable
	≥7 (Normal)	00 (0.00)	18 (100.00)	18 (100.00)	

*Neonatal mortality was not observed 36th week.

Test was not applicable as neonatal mortality was not observed at 36th week. There was no significant association of AFI, CTG, UA-PI, Ut A-PI, MCA-PI, CPR and APGAR scores with neonatal mortality at 36th week.

Table 34: Association of various parameters with neonatal morbidity at 37th week of gestation (n = 25).

Parameters	Categories	Neonatal morbidity		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
Amniotic Fluid Index (AFI)	≤ 5	02 (40.00)	03 (60.00)	05 (100.00)	$\chi^2 = 0.000$, df =1, P = 1.000 (>0.05), NS
	>5	08 (40.00)	12 (60.00)	20 (100.00)	
Cardiotocography (CTG)	Abnormal	02 (50.00)	02 (50.00)	04 (100.00)	$\chi^2 = 0.198$, df =1, P = 0.656 (>0.05), NS
	Normal	08 (38.10)	13 (61.90)	21 (100.00)	
Umbilical Artery Pulsatility Index (UA-PI)	5th – 95th percentile	10 (40.00)	15 (60.00)	25 (100.00)	Not applicable
	>95th percentile	00 (00.00)	00 (00.00)	00 (00.00)	
Uterine Artery Pulsatility Index (Ut A-PI)	5th -95th percentile	10 (40.00)	15 (60.00)	25 (100.00)	Not applicable
	>95th percentile	00 (00.00)	00 (00.00)	00 (00.00)	
Middle Cerebral Artery Pulsatility Index (MCA-PI)	<5th percentile	02 (66.67)	01 (33.33)	03 (100.00)	$\chi^2 = 1.010$, df =1, P = 0.315 (>0.05), NS
	≥5th percentile	08 (36.36)	14 (63.64)	22 (100.00)	
Cerebroplacental Ratio (CPR)	<1.08 (Altered)	10 (41.67)	14 (58.33)	24 (100.00)	$\chi^2 = 0.694$, df =1, P = 0.405 (>0.05), NS
	≥1.08 (Normal)	00 (0.00)	01 (100.00)	01 (100.00)	
APGAR Score at 1 min	< 7 (Abnormal)	09 (60.00)	06 (40.00)	15 (100.00)	$\chi^2 = 6.250$, df =1, P = 0.012 (<0.05), S
	≥7 (Normal)	01 (10.00)	09 (90.00)	10 (100.00)	
APGAR Score at 5 min	< 7 (Abnormal)	03 (100.00)	00 (0.00)	03 (100.00)	$\chi^2 = 5.114$, df =1, P = 0.024 (<0.05), S
	≥7 (Normal)	07 (31.82)	15 (68.18)	22 (100.00)	

#NS = Not significant, S = Significant

*Neonatal morbidity was present in total 10 (40.00%) cases.

There was no significant association of AFI, CTG, UA-PI, Ut A-PI, MCA-PI, CPR with neonatal morbidity at 37^{th} week.

Morbidity was significantly higher among neonates with abnormal APGAR scores compared with normal APGAR scores at 1 min ($\chi^2 = 6.250$, df =1, $P < 0.05$) and 5 min ($\chi^2 = 5.114$, df =1, $P < 0.05$).

Table 35: Association of various parameters with neonatal mortality at 37th week of gestation (n = 25).

Parameters	Categories	Neonatal mortality		Total n (%)	Chi-square test
		Yes n (%)	No n (%)		
Amniotic Fluid Index (AFI)	≤ 5	00 (0.00)	05 (100.00)	05 (100.00)	Not applicable
	>5	00 (0.00)	20 (100.00)	20 (100.00)	
Cardiotocography (CTG)	Abnormal	00 (0.00)	04 (100.00)	04 (100.00)	Not applicable
	Normal	00 (0.00)	21 (100.00)	21 (100.00)	
Umbilical Artery Pulsatility Index (UA-PI)	5th – 95th percentile	00 (0.00)	25 (100.00)	25 (100.00)	Not applicable
	>95th percentile	00 (0.00)	00 (00.00)	00 (00.00)	
Uterine Artery Pulsatility Index (Ut A-PI)	5th -95th percentile	00 (0.00)	25 (100.00)	25 (100.00)	Not applicable
	>95th percentile	00 (0.00)	00 (00.00)	00 (00.00)	
Middle Cerebral Artery Pulsatility Index (MCA-PI)	<5th percentile	00 (0.00)	03 (100.00)	03 (100.00)	Not applicable
	≥5th percentile	00 (0.00)	22 (100.00)	22 (100.00)	
Cerebroplacental Ratio (CPR)	<1.08 (Altered)	00 (0.00)	24 (100.00)	24 (100.00)	Not applicable
	≥1.08 (Normal)	00 (0.00)	01 (100.00)	01 (100.00)	
APGAR Score at 1 min	< 7 (Abnormal)	00 (0.00)	15 (100.00)	15 (100.00)	Not applicable
	≥7 (Normal)	00 (0.00)	10 (100.00)	10 (100.00)	
APGAR Score at 5 min	< 7 (Abnormal)	00 (0.00)	03 (100.00)	03 (100.00)	Not applicable
	≥7 (Normal)	00 (0.00)	22 (100.00)	22 (100.00)	

#NS = Not significant

*Neonatal mortality was not observed 37th week.

Test was not applicable as neonatal mortality was not observed at 37th week. There was no significant association of AFI, CTG, UA-PI, Ut A-PI, MCA-PI, CPR and APGAR scores with neonatal mortality at 37th week.

Table 36: Comparison of birth weight between 34th, 35th, 36th and 37th weeks of gestation.

	Birth weight (grams)			
	34th week	35th week	36th week	37th week
Mean ± SD	1437.33 ± 15.53	1553.29 ± 43.46	1751.00 ± 77.66	1893.60 ± 71.93
Min-Max	1420.00-1450.00	1498.00-1640.00	1650.00-1890.00	1750.00-1990.00
Kruskal Wallis test	$\chi^2 = 48.391$, df = 3, P =0.000 (<0.001), Very high significant			
Mann-Whitney U test	37th week> 36th week > 35th week > 34th week			

Birth weight at 37th week was significantly higher than 36th week, followed by 35th and 34th week.

Table 37: Comparison of neonatal morbidity between 34th, 35th, 36th and 37th weeks of gestation.

Gestation period	**Neonatal morbidity**		**Total** n (%)
	Yes n (%)	**No** n (%)	
34th week	03 (100.00)	00 (0.00)	03 (100.00)
35th week	07 (50.00)	07 (50.00)	14 (100.00)
36th week	09 (45.00)	11 (55.00)	20 (100.00)
37th week	10 (40.00)	15 (60.00)	25 (100.00)
Chi-square test	$\chi^2 = 3.958$, df = 3, P =0.266 (>0.05), Not significant		

There was no significant difference for neonatal morbidity between 34th, 35th, 36th and 37th weeks ($\chi^2 = 3.958$, df = 3, P >0.05).

Table 38: Comparison of neonatal mortality between 34th, 35th, 36th and 37th weeks.

Gestation period	**Neonatal mortality**		**Total** n (%)
	Yes n (%)	**No** n (%)	
34th week	03 (100.00)	00 (0.00)	03 (100.00)
35th week	01 (7.14)	13 (92.86)	14 (100.00)
36th week	00 (0.00)	20 (100.00)	20 (100.00)
37th week	00 (0.00)	25 (100.00)	25 (100.00)
Chi-square test	$\chi^2 = 46.615$, df = 3, P =0.000 (<0.001), Very high significant diff.		

Neonatal mortality at 34th and 35th weeks were significantly higher than 36th and 37th weeks ($\chi^2 = 46.615$, df = 3, P <0.001).

Table 39: Comparison of Mode of Delivery and Neonatal morbidity.

Comparison Groups	Mode of delivery	Neonatal morbidity		Total n	Chi-square test
		Yes n	No n		
Caesarean Section vs Vaginal Delivery	Caesarean Section	20	20	40	χ^2 =0.471 , df = 1, P =0.4924 (>0.05) Not significant
	Vaginal Delivery	09	13	22	
Emergency Caesarean Section VS Spontaneous Vaginal Delivery	Emergency Caesarean Section	17	16	33	X^2=1.428 ,dF=1 P=0.2321 (>0.05) Not significant
	Spontaneous Vaginal Delivery	03	07	10	
Emergency Caesarean Section VS Induction f/b Vaginal Delivery	Emergency Caesarean Section	17	16	33	X=0.008 ,df=1 P=0.9284 (>0.05) Not significant
	Induction f/b Vaginal Delivery	06	06	12	
Elective Caesarean Section vs Spontaneous Vaginal Delivery	Elective Caesarean Section	03	04	07	X=0.298 , df = 1, P =0.5851 (>0.05) Not significant
	Spontaneous Vaginal Delivery	03	07	10	
Elective Caesarean Section vs Induction f/b Vaginal Delivery	Elective Caesarean Section	03	04	07	X=0.090 ,dF=1 P=0.7636 (>0.05) Not significant
	Induction f/b Vaginal Delivery	06	06	12	
Induction f/b Caesarean Section vs Induction f/b Vaginal Delivery	Induction f/b Caesarean Section	02	01	03	X=0.268 ,df=1 P=0.6048 (.0.05) Not significant
	Induction f/b Vaginal Delivery	06	06	12	

The above table suggests that there is no significant association between neonatal morbidity and the mode of delivery of the foetus by Caesarean Section or Induced or Vaginal Delivery.

DISCUSSION

AGE

Half of the cases in our study were seen in the age group of 25-29 years (54.84%) followed by 25.81% of 30-35 years age group. The minimum maternal age was 18 years and maximum was 35 years. Lin et al. in their study tried to find a possible correlation between the extreme reproductive age groups that is below 17 years and above 35 years and IUGR foetuses but did not observe any associations and concluded that maternal age has no effect on the incidence of IUGR [(92)]. There were more number of patients in this age group as majority of women in our society conceive during this age.

SOCIOECONOMIC CLASS

In our study we observed that 61.29 % of women belonged to low socioeconomic group and 38.71% belonged to low middle socio-economic status. Moreover, most of the patients we cater to are from tribal areas. Thus, effect of social deprivation on birth weight is interconnected with the effects of associated lifestyle factors such as poor nutrition. In a study of 7493 British women, Wilcox and his associates did a retrospective analysis and found that the most socially deprived mothers had the smallest infants [(93)]. Similarly, Dejin-Karlsson and colleagues prospectively studied a cohort of Swedish women and found that lack of psychosocial resources increased the risk of growth restricted infants [(94)]. More than 100 years ago, Williams (1903) said "the social condition of mother and comforts by which she is surrounded also exert a marked influence on the child's weight, heavier children being more common in the

upper walks of life. Thus, screening of high risk patients with poor socioeconomic status is essential.

WEIGHT GAIN

Majority (85.48%) of the women in our study had weight gain of less than 8 kg during their pregnancy. This indicates the high incidence of IUGR being in those with poor maternal weight gain in pregnancy. Strauss and associates did a study on low maternal weight gain and its association with IUGR. The study was done on 10696 women enrolled in national collaborative perinatal project (NCPP) and the child health and development study (CHDS) and found out that low weight gain in third trimester was associated with a relative risk of IUGR of 1.7 (1.3-2.3) in the NCPP cohort and 2.59 (1.7-3.8) in the CHDS cohort. Low weight gain was defined as less than 0.1 kg per week for 1st trimester and less than 0.3 kg per week for the second and the last trimester [(95)]. The importance of weight gain during pregnancy has been mentioned in the textbook of Williams. The importance of weight gain had been studied by Abrams and Selvin and they observed that lack of weight gain in second trimester in strongly correlated with decreased weight gain [(96)]. The maternal weight gain in pregnancy is highly significant for prevention of IUGR.

RISK FACTORS

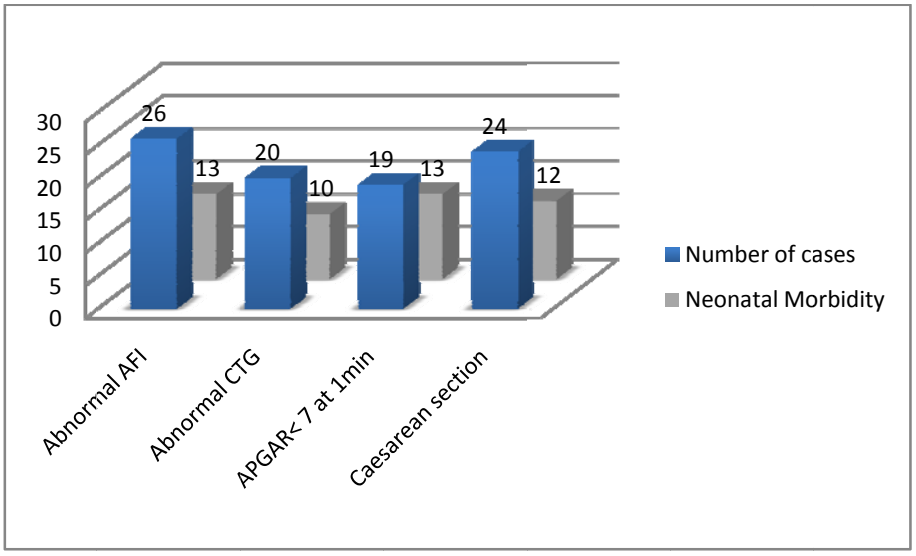
In our study, the most prevalent risk factors were anaemia (58.06 %) and gestational hypertension (54.84%). The third most prevalent factor of IUGR was sickling (37.10%). The patients we cater to are from the surrounding areas where sickle cell trait and disease are more prevalent. It was similar to Kozikui et al. and associates who did a meta-analysis and found that there was 50% increase in odds of SGA for

mothers with moderate to severe anaemia when pooling associations for haemoglobin cut offs of 9.0g% or 8.0 g% [97].

AMNIOTIC FLUID INDEX (AFI)

Out of 62 patients, 26 cases of our study had AFI≤5.

Figure 25: Comparison of study subjects of Abnormal AFI (AFI≤5) with Abnormal CTG, APGAR score at 1 minute and Caesarean Section in terms of Neonatal Morbidity (n=26).



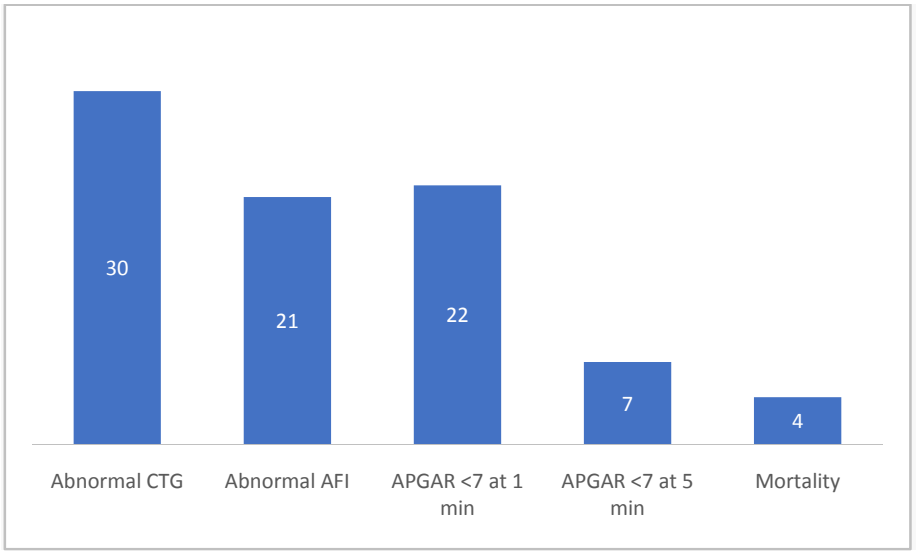
Out of 26 patients having AFI≤5, 13 foetuses had morbidity. Out of 26 patients having abnormal AFI, 20 patients had abnormal CTG of which 10 foetuses had morbidity. Out of 26 patients having abnormal AFI, 19 had APGAR less than 7 of which 13 foetuses had morbidity. Out of 26 patients having abnormal AFI, 24 patients underwent Caesarean Section of which 12 foetuses had morbidity. Our study is in accordance with Phelen et al. who defined AFI less than 5 as oligohydramnios and it correlated with increased rates of perinatal morbidity, caesarean delivery for foetal distress, meconium passage and low APGAR scores [69]. Simpson and creasy et al.

observed in their study that serial USG to assess amniotic fluid volume and interval foetal growth are important. Magaan et al. and associates also concluded in their study that SGA with oligohydramnios significantly increases the likelihood of a NICU admission [98]. Thus, in high risk pregnancies serial USG for AFI should be done so patients can be instructed on preventive measures like bed rest and empirical fluid intake.

CARDIOTOCOGRAPHY (CTG)

In our study, out of 62 patients, 30 patients had abnormal CTG

Figure 26: Comparison of study subjects of Abnormal CTG with Abnormal AFI (AFI≤5), APGAR score <7 at 1 minute, APGAR score <7 at 5 minutes and Mortality (n=30).



Out of these 30 foetuses which delivered,15 foetuses were admitted to NICU and 4 of them died in the NICU. Of these 30 patients with abnormal CTG, 21 patients had AFI≤5, 22 foetuses had APGAR at 1 minute less than 7 at birth and 5 foetuses had APGAR less than 7 at 5 minutes. Our study corelates with the study by Flynn et al.

and his colleagues in which they did a study on CTG in antepartum period involving 567 tracing of which 300 were non-reactive. In the study 22 cases were diagnosed of IUGR of which 14 had abnormal CTG and showed a significant association with stillbirths and neonatal deaths, admission to special care baby unit for conditions associated with intrauterine hypoxia, and low APGAR scores at 1 and 5 min [(99)].

UMBILICAL ARTERY PULSATILITY INDEX (UA-PI)

In our study, out of 62 patients, 34 patients had UA-PI more than 95th percentile (abnormal). No significant difference in neonatal morbidity was seen. Our study is in concordance with study by Mccowan et al. that UA-PI does not serve as a predictor for NICU stay [(100)].

UTERINE ARTERY PULSATILITY INDEX (Ut A-PI)

Similarly, 12 patients out of 62 had Ut A-PI more than 95th percentile. Out of 62 foetuses, 4 had mortality and of all these patients had Ut A-PI more than 95th percentile of that gestational age. Our study coincides with this study. A meta-analysis done by Allen et al. in 2016 indicated that high uterine pi was associated with increases (4 fold) chances of neonatal mortality.

MIDDLE CEREBRAL ARTERY PULSATILITY INDEX (MCA-PI)

In our study, neonatal morbidity and caesarean section seem to be directly correlated with MCA-PI less than 5th percentile but the number of study subjects is so less that it is difficult to come to a definite conclusion. Out of 62 patients, 25 patients had MCA-PI less than 5th percentile of which 13 (52%) foetuses had neonatal morbidity of which 12 were delivered by caesarean section. Severi et al. and associates found that abnormal MCA-PI in IUGR foetuses had increased chances of caesarean section [(41)].

CEREBROPLACENTAL RATIO (CPR)

Out of 62 patients, 57 patients had abnormal CPR (<1.08) of which 27(47.37%) foetuses were admitted to NICU. 5 patients had normal CPR of which 2(40%) foetuses were admitted to NICU. Our study coincides with the study by Figueras's group done in 2015 which consisted of 509 foetuses of which the patients who had abnormal CPR, 37.5% foetuses had adverse neonatal outcome [(90)].

OBSTETRIC AND NEONATAL OUTCOME

In our study of 62 patients, 40 (64.52%) patients underwent caesarean delivery. This coincides with the study done by Hasmasanu et al. In their study 66.9% of patients having IUGR underwent caesarean section [(101)].

There was decrease in morbidity as the gestational age advances but its statistical significance could not be established.

There was no statistical difference found in relation to morbidity of foetus in terms of mode of delivery and onset of labour (spontaneous/induced). In other words, caesarean section may not have an upper hand over vaginal delivery in terms of neonatal morbidity.

In our study, over all caesarean section rate was 64.52% (40/62). However, when the labour was induced, the caesarean section rate was 20% (Total number of labour induced-15, 12 delivered vaginally, 3 delivered by caesarean section).

Neonatal mortality was significantly higher in 34 and 35 weeks that 36 and 37 weeks of gestation with birth weight less than 1500 g. Our study matches with study by Bernstein et al. and associates who found statistically significant association of

intrauterine growth restriction with neonatal death (odds ratio, 2.77; 95% confidence interval, 2.31-3.33). They also found that intrauterine growth restriction within the range of 501 to 1500 g birth weight is associated with increased risks of neonatal death [(102)].

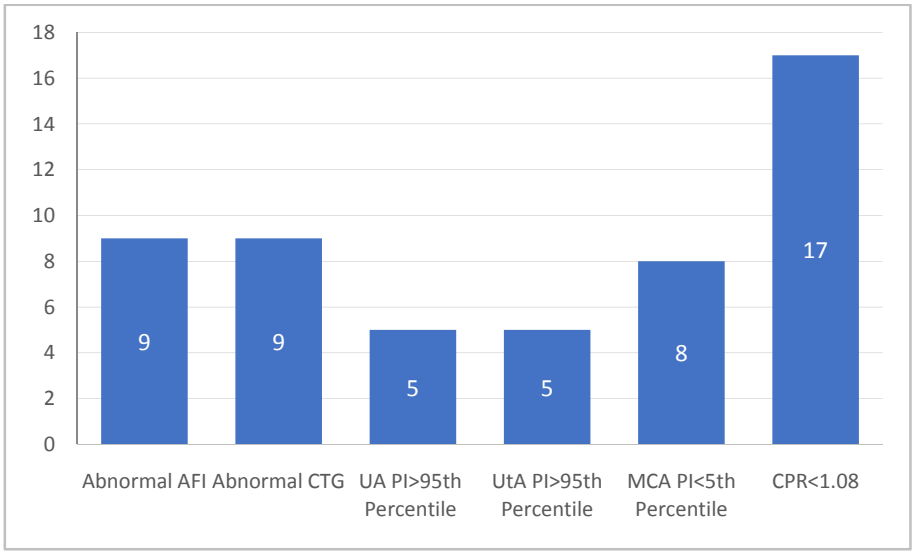
Mean APGAR score in our study at 1 minute was 6. This coincides with the study done by Hasmasanu et al. which included 142 subjects in which APGAR score at 1 min was 7 or more at 1 minute [(101)].

APGAR score at 1 and 5 minutes in vaginal delivery and caesarean section were 6 and 8 respectively. This indicates there was no difference in APGAR scores in vaginal delivery or caesarean section.

Birth asphyxia was found as a major cause of NICU admission in IUGR foetuses in our study. On comparing birth asphyxia with parameters such as AFI (≤5), CTG (Abnormal), UA-PI (>95th percentile), Ut A-PI (>95th percentile), MCA-PI (<5th percentile) and CPR (<1.08) it was found that all subjects having birth asphyxia had abnormal CPR (100%). Thus, birth asphyxia was highly prevalent in subjects having abnormal CPR. Similarly, the study concluded that birth asphyxia was prevalent in 71.4% cases with Ut A-PI >95th percentile, in 33.33% cases with AFI≤5, in 32% cases of MCA-PI<5th percentile, in 30% cases with Abnormal CTG and in 22.73% cases with UA-PI >95th percentile.

Out of 62 patients, 17 cases of our study had Birth asphyxia.

Table 27: Comparison of study subjects of Birth Asphyxia with Abnormal AFI (AFI≤5), Abnormal CTG, UA-PI >95th percentile, Ut A PI >95th percentile, MCA-PI <5th percentile and CPR <1.08 (n=17).



Out of these 17 cases of birth asphyxia, 9 cases had AFI≤5, 9 cases had Abnormal CTG, 5 cases had UA-PI>95th percentile, 5 cases had Ut A-PI>95th percentile, 8 cases had MCA-PI<5th percentile and all cases (17) had CPR<1.08.

SUMMARY

In this one and half year prospective study conducted from February 2016 to July 2017, 62 cases were diagnosed of "LATE ONSET IUGR" according to the previously mentioned criteria. The study was undertaken to find out the prevalence of Late onset IUGR and also to determine the best parameter as diagnostic and prognostic factor of Late onset IUGR. The following results were derived from the study:

- The prevalence of Late onset IUGR diagnosed in our institution was 2.44%.
- Majority of women (54.84 %) were in the age group 25-29 years.
- All (100 %) of the women belonged to the lower middle and lower socioeconomic status.
- Majority (85.48 %) women had weight gain in pregnancy less than 8 kilograms.
- All IUGR patients had estimated foetal weight less than 10^{th} percentile appropriate to that gestational age.
- The most prevalent risk factors were anaemia and gestational hypertension.
- Another important risk factor prevalent among our study patients was sickling.
- In our study, out of 62 patients, 26 patients had AFI≤5, 13 foetuses had NICU admission and 30 patients with abnormal CTG of which 15 foetuses had NICU admission.
- Out of 62 patients of our study, 12 patients had abnormal Ut A-PI of which 4 foetuses had mortality. This suggests a strong association of Ut A-PI with neonatal mortality.
- 25 patients in our study had MCA-PI abnormal and 12 out of these delivered by caesarean section.
- 64.52 % of patients underwent caesarean delivery.

- There was decrease in morbidity of newborn as the gestational age advances.
- 100 % babies had birthweight less than 2 kilograms.
- There was 6.45 % (4 foetuses) mortality in our study.
- There was no significant difference in maternal and neonatal morbidity in terms of mode of delivery (caesarean delivery/vaginal delivery).
- Birth asphyxia was found to be a major cause of NICU admission.

CONCLUSION

Weight gain seems to be a very strong prognostic factor in terms of association with IUGR, so diagnosis of decrease in weight gain should be made at an earliest. A deviation from the normal growth on the growth curve should make us think in the direction of foetus getting hampered.

PREDICTORS

In present study, we were not able to define the best predictor for diagnosis of Late onset IUGR. CTG and AFI, together seem to be a good prognostic factor for monitoring Late onset IUGR.

Ut A-PI shows promising results in predicting severe foetal compromise. Our study suggests a strong co-relation of mortality with altered Ut A-PI.

Abnormal CPR should also be considered for monitoring Late onset IUGR foetus in terms of neonatal morbidity.

HOW TO DELIVER?

There is no difference in neonatal morbidity in case of vaginal delivery or caesarean section, so once Late onset IUGR is diagnosed, utmost care should be taken in order to deliver the foetus before any sign of utero placental insufficiency are discovered. Moreover, preference should not be given to caesarean section unless signs of severe foetal compromise are seen.

THE FINAL VERDICT

Though recent advances and development in technology has made a big difference in our armamentarium to diagnose and manage Late onset IUGR, it still has so many lacunas and doubts. The simple test like AFI by USG and CTG are still very useful to monitor a case of Late onset IUGR.

Late onset IUGR still remains a dilemma and it is difficult to predict, diagnose and even more difficult to manage.

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ABBREVIATIONS

AC – Abdominal Circumference

ACOG – American Congress of Obstetricians and Gynecologists

AFI – Amniotic Fluid Index

AGA – Appropriate for Gestational Age

BPD – Biparietal Diameter

CPR – Cerebroplacental Ratio

CRL – Crown Rump Length

CTG – Cardiotocography

EFM – Electronic Foetal Monitoring

EFW – Estimated Foetal Weight

Elect. LSCS – Elective Lower Segment Caesarean Section

Emer. LSCS – Emergency Lower Segment Caesarean Section

FL – Femur length

FSB – Fresh Stillbirth

FTND – Full Term Normal Delivery

G.HTN – Gestational Hypertension

GDM – Gestational Diabetes Mellitus

gms – Grams

GUT – Genito-Urinary tract

HC – Head Circumference

Ht – Height

IL – Illiterate

IUD – Intra-Uterine Death

IUGR – Intra Uterine Growth Restriction

Kgs – Kilograms

L – Literate

LBW – Low Birth Weight

MCA – Middle Cerebral Artery

NICU – Neonatal Intensive Care Unit

Para - Parity

PI – Pulsatility Index

Prev. IUGR – Previous Intrauterine Growth Restriction

SD – Standard Deviation

SES – Socio-economic status

SGA – Small for Gestational Age

Sr. No. – Serial Number

UA – Umbilical Artery

USG – Ultrasonography

Ut A – Uterine Artery

Vg. Delivery – Vaginal Delivery

VLBW – Very Low Birth Weight

WHO – World Health Organization

Wt. – Weight

PROFORMA / FORMAT

S.B.K.S MEDICAL COLLEGE AND RSEARCH INSITUTE

DHIRAJ HOPITAL

SUMANDEEP VIDHYAPEETH UNIVERSITY

TITLE OF THE STUDY: THIRD TRIMESTER IUGR, PREDICTORS AND ITS OBSTETRIC OUTCOME

PROFORMA

- Name of patient
- Husband's name
- Age
- Address
- Ipd No.
- Education of patient
- Education of husband
- Registered/emergency case
- Socio economic status
- Occupation
- Date of admission
- Date of discharge
- Duration of pregnancy

PRESENTING COMPLAINS:

MENSTRUAL HISTORY

- L.M.P.
- E.D.D.
- Past Menstrual cycle

  Regular/Irregular

  Amount

  Painful/Painless
- Gestational age (weeks)

OBSTETRIC HISTORY

- Active Married life
- Gravida _____ Para_____ Live_____ Abortion_____

PARITY	DELIVERY	INDICATION	PLACE	SEX	AGE	COMPIACTION

PAST HISTORY:

- TB
- Diabetes
- Jaundice
- Asthma
- Any major medical or surgical illness
- Hypertensive disorders in pregnancy
- Hemorrhagic disorders

- Blood Transfusion
- On any kind of medication
- Autoimmune Disorders
- Drug allergy

FAMILY HISTORY:

Major illness (TB, Diabetes, Hypertension, Jaundice, Asthma, Multiple gestation, Epilepsy) or any other medical disorders.

PERSONAL HISTORY:

- Diet
- Appetite
- Sleep
- Bowel habits
- Bladder
- Addiction (if any)

GENERAL PHYSICAL EXAMINATION:

- Level of consciousness
- Cooperative / uncooperative
- Well oriented to time, place & person
- Built
- Nourishment
- Height (cm)
- Weight (kg)
- Temperature
- Pulse /min
- Blood Pressure mm/hg
- Respiratory Rate /min

- Pallor
- Icterus
- Cyanosis
- Clubbing/ Koilonychia
- Lymphadenopathy
- Pedal Oedema

SYSTEMIC EXAMINATION:

- Cardiovascular system
- Respiratory system

OBSTETRICS EXAMINATION:

PER ABDOMEN:

- **INSPECTION-**
    - Any scar on abdomen
    - Stria gravidarum
    - Linea nigra
    - Dilated veins
    - Umbilicus- shape, position
- **PALPATION-**
    - Fundal Height: cm
    - Abdominal girth: cm
    - EBW: Kg
    - Uterus: Contracted/ relaxed
    - Lie:
    - Presentation:
    - Engagement of presenting part
- **AUSCALTATION-**
    - Fetal heart sound:
    - Rate: beats/min
    - Rhythm: Regular/Irregular

PER SPECULUM

- **BLEEDING PER VAGINUM-**
    - Amount
    - Color of bleeding
- **LEAKING PER VAGINUM-**
    - Amount
    - Color of liquor
- **CERVICAL PATHOLOGY** (if any)
- **VAGINAL PATHOLOGY** (if any)

PER VAGINUM

- Cervical Dilatation
- Cervical Effacement
- Presenting part
- Membrane-Present/ Absent
- Station of head
- Pelvic assessment

INVESTIGATIONS:

- Hemoglobin: gm%
- RBS:
- Urine: R/M: Albumin:

  Sugar:
- Any other investigations if required.

ULTRASONOGRAPHY FINDINGS-

- IST TRIMESTER-

DATE:			
CRL			
EDD			

- IIST TRIMESTER-

DATE			
BPD			
FL			
AC			
EDD			
AGA			
EBW			
AFI			
PLACENTA			

- IIIST TRIMESTER-

DATE			
BPD			
FL			
AC			
EDD			
AGA			
EBW			
AFI			
PLACENTA			

- DOPPLER STUDIES:

	UA-PI	Ut A-PI	MCA-PI	CPR
34 weeks				
35 weeks				
36 weeks				
37 weeks				

- CTG- Reactive/Non-Reassuring/Abnormal (34/35/36/37 weeks)
- TYPE OF DELIVERY:

  SPONTANEOUS/INDUCED

  If induced; Indication of Induction:

  INSTRUMENTAL: YES/NO

  LSCS: EMERGENCY INDICATION-

  ELECTIVE INDICATION-
- Gestational age at the time of birth(weeks):
- **MOTHER**
  - Morbidity (If any):
  - Mortality (If any):
- **BABY**
  - Live birth/Still born/Intra uterine death
  - Sex of the baby: MALE/FEMALE
  - Weight: Kg.
  - Time of birth: a.m./p.m.
  - Date of birth: / /201
  - Ponderal Index
  - APGAR score
  - Admission to NICU: YES/NO

    If yes, indication:

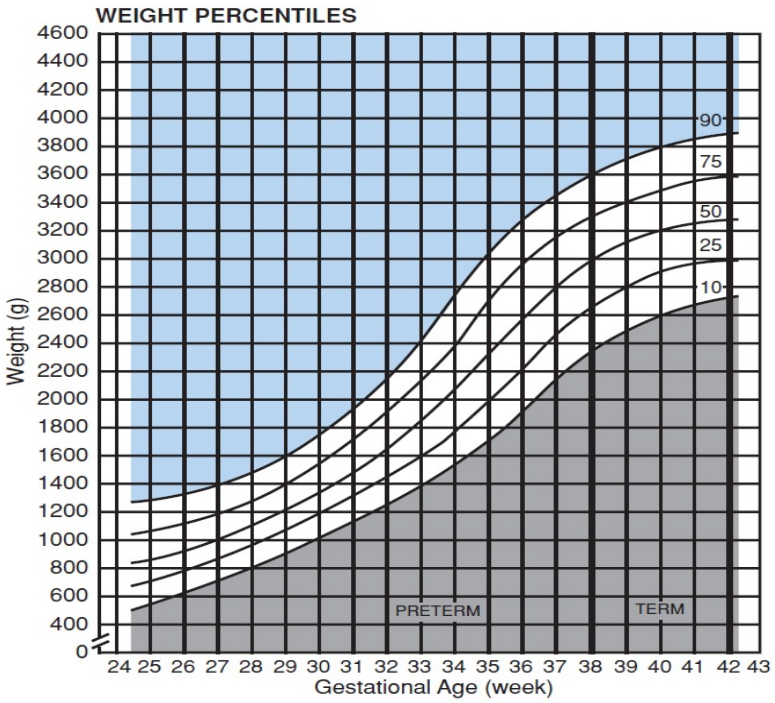

Figure 28: Weight Percentile Growth Chart

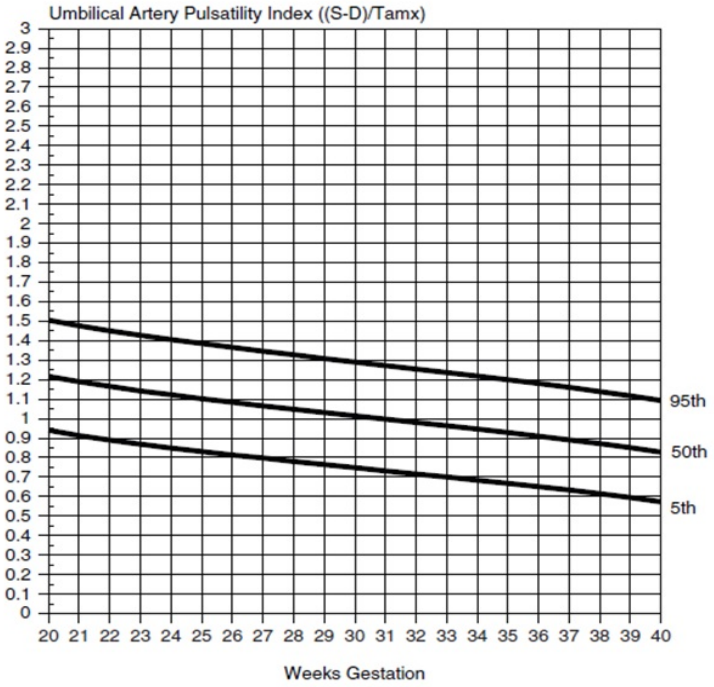

Figure 29: Umbilical Artery Pulsatility Index Graph

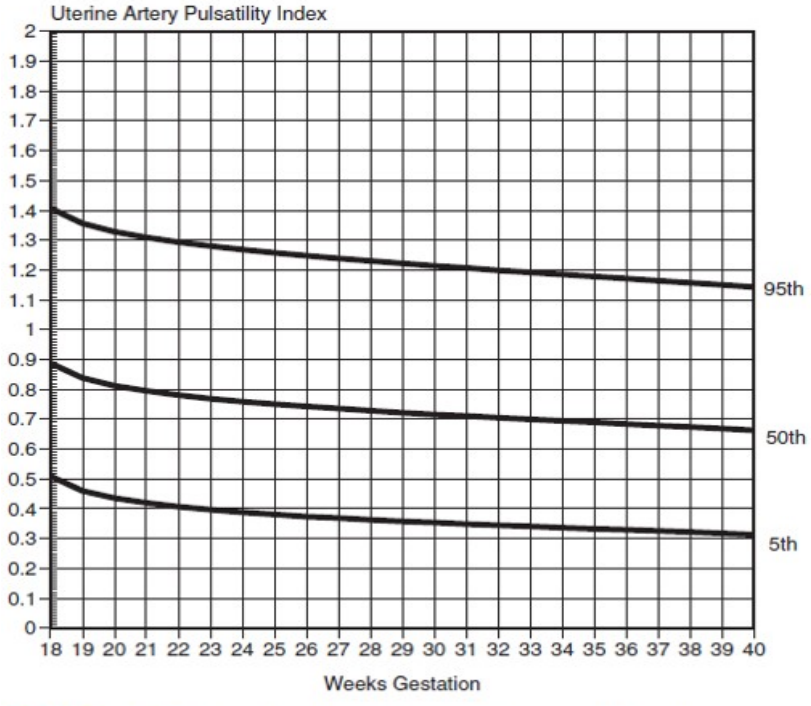

Figure 30: Uterine Artery Pulsatility Index Graph

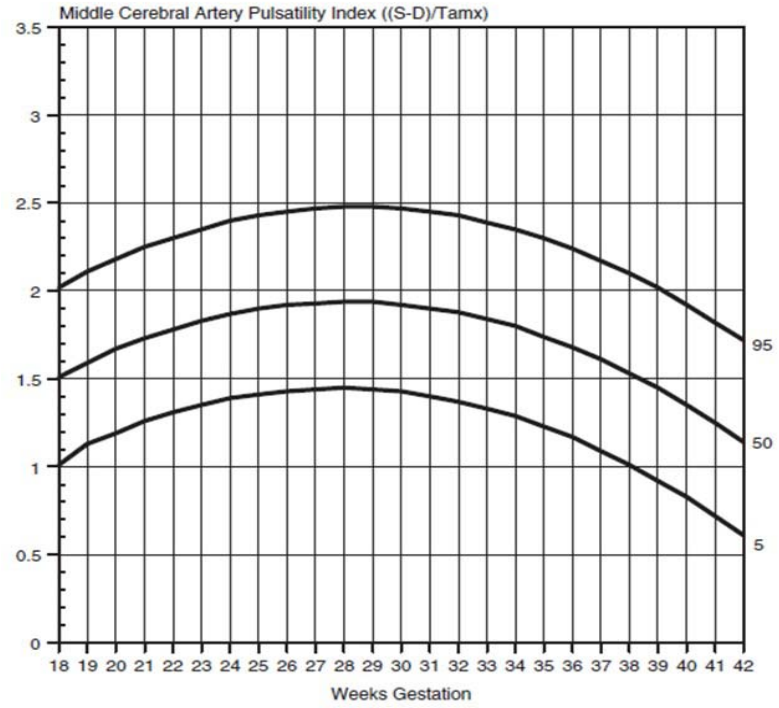

Figure 31: Middle Cerebral Artery Pulsatility Index Graph

PARTICIPANT INFORMATION SHEET

Title of the study:

THIRD TRIMESTER IUGR, PREDICTORS AND ITS OBSTETRIC OUTCOME

Introduction

In this study, the predictors and outcome of third trimester IUGR will be studied.

Study no: Date:

You are cordially invited for the study

1. What is the purpose of this study?

The purpose is to study the third trimester IUGR, predictors and its obstetric outcome in patients in patients of obstetrics in Dhiraj Hospital. The findings will help in confirmation of diagnosis of IUGR and will help in deciding the further management of pregnancy.

2. **Aim of Study:**

The aim of this study is to pick up those fetuses who are getting compromised after 34 weeks of gestation mainly due to placental insufficiency and to deliver them before they become hypoxic so as to reduce neonatal morbidity and mortality.

3.Why have I been chosen?

You have been chosen as you fit in the inclusion criteria of the study.

4. Do I have to take part?

It is totally voluntary to take part in this study.

5. How long will the study last?

This study will last for 1 year.

6. What will happen to me if I take part?

It is an observational study so nothing will happen to you**.**

7.What do I have to do?

You need to cooperate in our study till the end.

8. What is the drug being tested?

No drug is being tested. It is an observational study.

9. What are the benefits of the study?

This study will help in reducing the morbidity and mortality caused in cases of IUGR by its early prediction.

10. What are the side effects of the treatment received during the study?

There are no side effects as it is an observational study.

11. What if new information becomes available?

If any new information comes in between we will follow the new guidelines.

12. What happens when the study stops?

When the study stops, we will compile the data and statistically analyse the results

13. What if something goes wrong?

It is purely an observational study**.**

14.Will my taking part be kept confidential?

Yes, patients' information will be kept confidential.

15. What else should I know?

Not applicable.

17. What else can I know?

If you have anything in mind related to its advantages and disadvantages, you can ask about it without any hitch.

18.Who to call with questions?

Dr. SWAR SHAH

RESIDENT OBSTETRICS AND GYNECOLOGY,

DEPARTMENT OF OBSTETRICS AND GYNECOLOGY,

SBKS MI&RC, PIPARIYA

Tal. Waghodia, Dist. Vadodara

Mob: - 9099700250

Sumandeep Vidyapeeth University

Piparia, Ta. Waghodia, Dist. Vadodara. Pin 391760

Informed Consent Form (ICF) for Participants in Research Programmes involving studies on human beings:

Study title:

THIRD TRIMESTER IUGR, PREDICTORS AND ITS OBSTETRIC OUTCOME

Study Number: SVU/SBKS/ /2016-____

Participants Initials: _____

Participant's Name _____

Date of Birth / Age _____ (Years)

1. I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]
2. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]
3. I understand that the investigator of this study, others working on the investigator's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records, both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I agree to this access. However, I understand that my identity will not be revealed in any information related to third party or published. [ ]
4. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). [ ]

5. I agree to take part in the above study. [ ]

Signature (or thumb impression) of the participants /

Legally acceptable representative _____

Signatory's Name _____ Date _____

Signature of the investigator _____ Date _____

Study Investigator's Name _____

Signature of the impartial witness _____

Date _____

Name of the witness _____

cpN g¡“pf“y„ dprlsu‘ÓL$

Aæepk“y„ iuj®L$:
s©sue rÓdprkL$dp„ ApCeyÆApf ApNplu L$f_pfpAp¡ A_¡ s¡_p âkyrsip÷ `qfZpd.

`qfee
Ap Aæepkdp„ ApNplu L$f_pf A_¡ ÓuÅ rÓdprkL$ ApCeyÆApf_y„ `qfZpd Aæepk L$fhpdp„ Aphi¡.

Aæepk _„.
spfuM :
klcpNu_¡ Apd„ÓZ

(1) Ap Aæepk_p¡ l¡sy iy„ R>¡ ?
r^fS> lp¡[õ`V$g_p v$v$}Ap¡dp„ rÓdprkL$ ApC.ey.Æ.Apf. ApNplu L$f_pf A_¡ v$v$}Ap¡dp„ s¡_p
âk|rs k„b„^u `qfZpdp¡_p¡ Aæepk L$fhp. Ap spfZp¡ ApC.ey.Æ.Apf. _u `yrô$ L$fhpdp„ dv$v$
L$fi¡ A_¡ Ncp®hõ\p_p h^y ìehõ\p`__¡ _L$¼u L$fhpdp„ dv$v$ L$fi¡.

(2) Aæepk_y„ gÿe
Ap Aæepk_p¡ DØ¡i A¡hp c|Z _¡ _½$u L$fhp_y„ R>¡ L¡$ S>¡ d¡gu_u (Ap¡2 _u) A`|Z®sp_¡ L$pfZ¡
34 AWhpXuep_p Ncp®^pZ `R>u rhL$pk ê„$^pe Åe R>¡. A_¡ s¡ lpC`p¡[¼kk \sp„ `l¡gp s¡d_u
kyhphX$ L$fphhp dpV¡$ R>¡ S>¡\u r_ep¡_¡V$g fp¡N A_¡ d©Đeyv$f OVpXhpdp„ Aphu iL¡$.

(3) d_¡ ip dpV¡$ `k„v$ L$fhpdp„ Apìep¡ R>¡ ?
sd¡ Aæepk_p kdph¡i_p dp`v$„X$p¡dp„ auV$ \sp lp¡hp\u sd_¡ `k„v$ L$fhpdp„ Apìep R>¡.

(4) iy„ dpf¡ cpN g¡hp_u S>ê$f R>¡ ?
sd¡ õh¥[ÃR>L$ fus¡ Ap Aæepkdp„ cpN gC iL$p¡ R>p¡.

(5) Aæepk L¡Vgp kde ky^u Qpgi¡ ?
Ap Aæepk 1 hj® ky^u Qpgi¡.

(6) Å¡ ly„ cpN gD sp¡ iy„ \i¡ ?
Ap A¡L$ r_qfnZ dpV¡$_p¡ Aæepk R>¡, sd_¡ L$p„C _lu \pe.

(7) dpf¡ iy„ L$fhy„ `X$i¡ ?
sdpf¡ Aæepk Äep„ ky^u Qpg¡ Đep„ ky^u Ad_¡ klL$pf Ap`hp_u S>ê$f R>¡.

(8) v$hpAp¡ L¡$hu fus¡ `funZ L$fhpdp„ Aph¡ R>¡ ?
L$p¡C v$hp_y„ `funZ L$fhpdp„ Aphsy„ _\u, Ap A¡L$ r_qfnZ Aæepk R>¡.

(9) Ap Aæepk_p gpcp¡ iy„ R>¡?
Ap Aæepk\u âpf„rcL$ ApNplu Üpfp ApC.ey.Æ.Apf. _p fp¡N A_¡ d©Đeyv$f OV$pX$hpdp„ dv$v$
\i¡.

(10) Aæepk v$fçep_ dm¡g kpfhpf_u ApX$-Akfp¡ iy„ R>¡ ?

L$p¡C ApX$-Akfp¡ _\u L$pfZ L¡$ Ap A¡L$ r_qfnZ dpV¡$_p¡ Aæepk R>¡.

(11) Ap Aæepk v$fçep_ _hu dprlsu D`gå^ \pe sp¡ iy„ ?

Ap Aæepk v$fçep_ Å¡ _hu dprlsu Aph¡ sp¡ Ad¡ _hp r_v$£ip¡_y„ `pg_ L$fuiy„.

(12) Äepf¡ Aæepk b„^ \pe Ðepf¡ iy„ \pe R>¡. ?

Äepf¡ Aæepk b„^ \i¡ Ðepf¡, Ad¡ X¡Vp_y„ k„L$g_ L$fuiy„ A_¡ `qfZpdp¡_y„ ApLXpL$ue rhïg¡jZ

L$fuiy„.

(13) Å¡ L„CL Mp¡Vy„$ \pe sp¡ iy„ ?

Ap A¡L$ õ`ô$ `Z¡ r_qfnZ Aæepk R>¡.

(14) iy„ dpfp¡ cpN g¡hp_y„ Nyàs fpMhpdp„ Aphi¡ ?

lp, vv}Ap¡_u dprlsu_¡ Nyàs fpMhpdp„ Aphi¡.

(15) dpf¡ buSy>„ iy„ ÅZhy„ Å¡CA¡ ?

gpNy `X$sy„ _\u.

(16) ly„ buSy>„ iy„ ÅZu iLy„$?

Å¡ sd_¡ Aæepk_p gpcp¡ A_¡ N¡fgpcp¡ k„b„r^s d_dp„ L„$C`Z lp¡e sp¡ sd¡ s¡_¡ r_:k„L$p¡Q

`Z¡ `|R>u iL$p¡ R>p¡.

(17) âï_p¡ dpV¡$ L$p¡_¡ ap¡_ L$fhp¡.

X$pµ. õhf ipl

f¡kuX¡$ÞV$ X$p¸L$V$f Ap¡åõV¡$qV²Lk A“¡ Npe“¡L$p¡gp¡Æ,
Ap¡åõV¡$qV²$L$k A“¡ Npe“¡L$p¡gp¡Æ rhcpN,
A¡k.bu.L¡$.A¡k. A¡d.ApC. A¡ÞX$ Apf.ku., ‘u`muep,
sp. hpOp¡X$uep, rS>. hX$p¡v$fp. dp¡. : 9099700250

kyd“v$u‘ rhÛp‘uW$, ‘u‘muep, sp. hpOp¡X$uep, rS>. hX$p¡v$fp - 391 760

d_yóe `f_p Aæepkp¡ krls_p k„ip¡^“ L$pep£dp„ klcpNuAp¡ dpV¡$“y„ ÅZL$pfu k„drs ap¡d®

Aæepk“y„ iuj®L$:
ÓuÅ rÓdprkL$ ApCeyÆApf : A_ydp_p¡ A_¡ s¡_p âkyrsip÷ `qfZpd

spfuM :

Aæepk “„bf : A¡k.hu.ey./A¡k.bu.L¡$.A¡k./ 201

klcpNu“p ‘yfp “pd“p ‘l¡gp Anfp¡ : .....

klcpNu“y„ “pd ..... S>Þd spfuM/Jdf .....(..... hj®)

- lº„ `yrô$ L$fy„ Ry>„ L¡$ d¢ D`fp¡¼s Aæepk dpV¡$ spfuM :..... _u dprlsu‘ÓL$ hp„Qu A“¡ kdÆ“¡ Aæepkdp„ kpd¡g ’hp“u Mpsfu Ap‘y„ Ry>„ A“¡ d“¡ L$p¡C‘Z âñp¡ ‘yR>hp“u sL$ Ap‘hpdp„ Aphi¡. S>¡“u d“¡ Mbf R>¡.
- dpfu Aæepkdp„ cpNuv$pfu õh¥[ÃR>L$ R>¡ A“¡ lº„ dpfu cpNuv$pfu L$p¡C‘Z kde¡ L$p„C`Z L$pfZ Apàep hNf,
  dpfu sbubu kpfhpf“¡ Akf “p ‘lp¢Q¡ A’hp dpfp L$p“y“u Ar^L$pfp¡“¡ Akf “p ’pe s¡d Nd¡ Ðepf¡ ‘pR>u M¢Qu
  g¡hp õhs„Ó Ry>„.
- lº„ Ap Aæepk“p AæepkL$sp®, AæepkL$sp®“p klcpNu, A¡’uL$k L$rdV$u A“¡ f¡Áeyg¡V$fu Ap¡’p¡fuV$uk“¡ dpfu ‘fhp“Nu hNf dpfp õhpõÕe“p¡ f¡L$p¡X®$ L¡$ S>¡ Ap Aæepk L¡$ Ap crhóedp„ ’“pfp ‘qfnZ“p k„v$c®dp„ ’i¡. s¡“¡ Å¡hp“u k„drs Ap‘y Ry>„. Å¡ lº„ dpfu k„drs ‘pR>u M¢Qu gJ sp¡ ‘Z dpfu dprlsu AæepkL$sp® d¡mhu iL$i¡. R>sp„ lº„ kdSy> Ry>„ L¡$, dpfu Ap¡mMpZ L$p¡C‘Z õhê$‘dp„ ÓuÆ ìe[¼s“¡ A‘pi¡ “tl L¡$ Ål¡fdp„ âL$pris L$fhp _rl Aph¡.
- Ap Aæepk ‘f’u S>¡ ‘qfZpd L¡$ dprlsu dm¡ s¡“u ‘f lº„ ârsb„^ “rl dyLy„$. s¡“p¡ D‘ep¡N a¼s h¥opr“L$ l¡sy dpV¡$ L$fhpdp„ Aphi¡.
- lº„ D‘f“p Aæepkdp„ cpN g¡hp“u k„drs Ap‘y„ Ry>„.

klcpNu A\hp LpevpL$ue fus¡
õhuL$pe® ârsr“r^_u klu A’hp A„NyW$p“y„ r“ip“.....

kluL$sp®“y„ “pd : ..... spfuM .....
s`pk_uk_u klu ..... spfuM .....
Aæepk s`pk L$f_pf_y„ “pd : ..... spfuM .....
r“ó‘n kpnu“u klu ..... spfuM .....
r“ó‘n kpnu“y„ “pd : ..... spfuM .....

^mJ boZo dmbo H$m gyMZm nÌ
AÜ``Z H$m erf©H$
Vrgam {Ì{_{V` AmB©`wOrAma àr{S>ŠQ>g© Am¡a BgHo$ àgw{V n[aUm_

n[aM`
Bg AÜ``Z _|, ^{dî`dm{U`m| Am¡a Vrgao Ì¡_m{gH$ AmB©`wOrAma Ho$ n[aUm_ H$m AÜ``Z {H$`m OmEJm &

AÜ``Z Z§. :

{XZm§H$:

à{V^mJr H$mo Am_§ÌU

(1) Bg AÜ``Z H$m CÔoe Š`m h¡ ?

YraO hmopñnQ>b _| àgy{V Ho$ XXuAm| _| Vrgao {Ì{_{V` AmB©`yOrAma àr{S>ŠQ>g© Am¡a XXuAmo| _o BgH$m

àË`mamonU n[aUm_ H$m AÜ``Z H$aZm gh {ZîH$f© AmB©`wOrAma Ho$ {ZXmZ H$s nw{ï> _| _XX H$aoJm Am¡a J^m©dñWm

Ho$ AmJo Ho$ à~§YZ H$m {ZU©` boZo _| _XX H$aoJm &

(2) AÜ``Z H$m CÔoe

Bg AÜ``Z H$m CÔoe CZ ^«yUmo H$mo MyZZm h¡ Om¡ 34 gßVmh Ho$ J^© Ho$ ~mX g_Pm¡Vm H$a aho h¡ _w»` én go

~oah_m| go An`m©ßV H$maU Am¡a CÝh| hmænmopŠgH$$ hmoZo go nhbo {dV[aV H$aZm VmH$s ZdOmV H$s amoJÊVm Am¡a

¥Ë`wXa H$ hmo &

(3) _wPo Š`m| MwZm J`m h¡ ?

AÜ``Z _| em[_b {H$E JE _mnX§S>mo _| {\$Q> hmoZo Ho$ H$maU AmnH$mo MwZm J`m h¡ &

(4) Š`m _wPo ^mJ boZm h¡ ?

Bg AÜ``Z _| ^mJ boZm nyar Vah go ñd¡pÀN>H$ h¡ &

(5) AÜ``Z {H$VZo g_` VH$ MboJm ?

`h AÜ``Z 1 gmb VH$ MboJm &

(6) AJa _| AÜ``Z _| ^mJ boVm hÿ§ Vmo _oao gmW Š`m hmoJm ?

`h EH$ AdbmoH$Z g§~§Yr AÜ``Z h¡, Bg{bE Amn Ho$ gmW Hw$N> ^r Zhr hmoJm &

(7) _wPo Š`m H$aZm h¡ ?

AmnH$mo A§V VH$ AÜ``Z _| gh`moJ XoZo H$s Oê$aV h¡ &

(8) {H$g XdmB©`m§ H$m n[ajU {H$`m Om ahm h¡ ?

`h EH$ AdbmoH$Z g§~§Yr AÜ``Z h¡, H$moB© Xdm H$m n[ajU Zhr {H$`m Om ahm h¡ &

(9) AÜ``Z Ho$ Š`m bm^ h¡ ?

`h AÜ``Z AmB©`yOrAma Ho$ ewéAmVr nydm©Zy_mZ Ho$ AmYma na amoJr-g§»`m Am¡a _¥Ë`wXa H$mo H$_ H$aZo _| _XX

H$aoJm &

(10) AÜ``Z Ho$ Xm¡amZ àmßV CnMma Ho$ Xþîà^md H$`m h¡ ?

H$moB© Xþîà^md Zhr h¡ Š`m|{H$ `h EH$ AdbmoH$Z g§~§Yr AÜ``Z h¡ &

(11) `{X ZB© OmZH$mar CnbãY hmo Vmo Š`m hmoJm ?

AJa {~M _| H$moB© ^r ZB© OmZH$mar AmVr h¡ Vmo h_ ZB©© {Xem{ZX}emo H$m nmbZ H$a|Jo &

(12) O~ AÜ``Z ~§Y hmo OmVm h¡ Vmo Š`m hmoVm h¡ ?

O~ AÜ``Z ~§Y hmo OmVm h¡ Vmo, h_ S>oQ>m g§H${bV H$a|Jo Am¡a n[aUm_m| H$m gm§p»`H$r` ê$n _| {dûdbofU H$a|Jo &

(13) AJa Hw$N> JbV hmo OmE Vmo Š`m hmoJm ?

`h EH$ AdbmoH$Z AÜ``Z h¡ &

(14) Š`m _oam ^mJ boZm JmonZr` alm OmEJm ?

hm±, amoJr`m| H$s OmZH$mar JmonZr` alr OmEJr &

(15) _wPo Am¡a Š`m nVm hmoZm MmhrE ?

bmJy Zhr &

(16) _¡ Am¡a Š`m OmZ gH$Vm hÿ§ ?

`{X Amn An$Zo \$m`Xo Am¡a ZwH$gmZ go g§~§{YV Hw$N> ^r OmZZm MmhVo h¡ Vmo Amn BgHo$ ~mao _| {H$gr ^r àH$ma Ho$ g§H$moM Ho$ {~Zm nyN> gH$Vo h¡ &

(17) àûZ {H$ggo nyN> gH$Vo h¡ ?

S>m° ñda emh

Amdmgr` AmoãñQ´>oQ>rH$ Am¡a Jm`ZoH$mobmoOr,
Amo~ñQ>o´Q>rH$ Am¡a Jm`ZoH$mobmoOr {d^mJ,
Eg~rHo$Eg Eg AmB© AoÝS> Amagr, nrnar`m,
VmbwH$m : dmKmo{S>`m, {O. dS>moXam.
_mo~mB©b : 9099700250

gw_Z{Xn {dÚmnrR> `w{Zd{g©Q>r, nrnar`m, Vm. dmKmo{S>`m, {O. dS>moXam - 391 760

B§gmZmo na AÜ``Z go OwS>o AZwg§YmZ H$m`©H«$_mo _| à{V^m{J`mo Ho$ {bE gy{MV gh_{V nÌ (AmB©grE\$)

AÜ``Z erf©H$$:
Vrgam {Ì{_{V` AmB©`wOrAma àr{S>ŠQ>g© Am¡a BgHo$ àgw{V n[aUm_

{XZm§H$:

AÜ``Z g§»`m : Egdr`y / EgdrHo$Eg /201 .....

à{V^mJr`m| Ho$ Zm_ Ho$ nhbo Aja ..... OÝ_ {V{W/Am`w .....(..... df©)

(1) _¢ nw{ï> H$aVm hÿ§ Ho$ _oZo Cnamoº$ AÜ``Z Ho$ {bE {XZm§H$ ..... Ho$ gyMZm nÌ H$mo nT> Am¡a g_P {b`m h¡ &
Am¡a _wPo gdmb nwN>Zo H$m Adga {_bm h¡ &

(2) _¢ g_PVm hÿ§ H$s AÜ``Z _| _oar ^mJrXmar ñd¡pÀN>H$ h¡ Am¡a `h {H$ _o§ {H$gr ^r g_` {~Zm H$moB© H$maU {XE Bg
Aä`mg go _wº$ Ho$ {bE ñdV§Ì hÿ§ & {H$gr ^r H$maU go _oar {M{H$Ëgm XoI^mb `m H$mZyZr A{YH$mam| Ho$ {~Zm à^m{dV
hmo ahm h¡ &

(3) _¢ g_PVm hÿ§ Ho$ Bg AÜ``Z Ho$ AÝdofH$, AÝdofH$ H$s Amoa go H$m_ H$aZo dmbo AÝ`, E{WŠg H$s{_Q>r Am¡a {Z`m_H$
n«m{YH$mar`m| H$mo _oao ñdmñW` [aH$moS>© H$mo XoIZo H$s _oar AZw_{V H$s Amdí`H$Vm Zhr hmoJr &

(4) _¢ Bg AÜ``Z go CÎmnÞ hmoZo dmbo {H$gr ^r S>oQ>m `m n[aUm_m| Ho$ Cn`moJ H$mo à{V~§{YV H$aZo Ho$ [bE gh_V
Zht hÿ§, bo{H$Z Bg Vah H$m Cn`moJ H¡$db d¡km{ZH$ CÔoemo Ho$ {bE h¡ &

(5) _¢ Cnamoº$ AÜ``Z _| ^mJ boZo Ho$ {bE gh_V hÿ§ &

à{V^mJr`m| Ho$ hñVmja (`m A§JyR>o H$s N>mn) .....

H$mZyZr én go ñdrH$m`© à{V{Z{Y hñVmjaH$Vm© H$m Zm_ :
..... {XZm§H$ .....
Om§MH$Vm© Ho$ hñVmja .....
{XZm§H$ .....
AÜ``Z AÝdofH$ H$m Zm_ .....
XZm§H$ .....
{Zînj Jdmh Ho$ hñVmja .....
{XZm§H$ .....

gmjr H$m Zm_ .....
{XZm§H$ .....

Sr. No.	Age	Literacy	SE Status	Para	Risk Factors	Anaaemia grade	Weight Gain (kg)	Fetal weight (gm) by biometry				Amniotic Fluid Index(AFI) by four quadrant method				Cardiotocography (Normal(R)/ Abnormal(AB))				Umbilical artery(UA)				Uterine artery(UtA)			
																				PI	PI	PI	PI	PI	PI	PI	PI
								34 wks	35 wks	36 wks	37 wks	34 wks	35 wks	36 wks	37 wks	34 wks	35 wks	36 wks	37 wks	34 wks	35 wks	36 wks	37 wks	34 wks	35 wks	36 wks	37 wks
1	35	IL	Lower	4	Anaemia+Sickle Cell Trait+G.Htn	Moderate	6	1484	1569			5.2	3.8			R	AB			1.21	1.32			1.24	1.24		
2	30	L	Lower Middle	3	Anaemia+Sickle Cell Trait+G.Htn	Moderate	7	1490	1592	1678		6.5	6.1	4.4		R	R	R		1.18	1.18	1.19		1.02	0.93	0.88	
3	24	L	Lower Middle	1	GDM	No	7.5	1558	1664	1787	1880	10	9.4	8.8	7.4	R	R	R	R	1.18	1.17	1.18	1.18	0.89	0.84	0.79	0.77
4	26	IL	Lower	2	G.Htn+GDM	No	7	1650	1754	1890		11.4	10	9.4		R	R	R		1.18	1.17	1.16		0.86	0.83	0.79	
5	34	IL	Lower	4	Anaemia+Sickle cell disease+G.Htn	Severe	5.4	1484	1572			9.4	9			R	AB			1.18	1.2			1.21	1.2		
6	25	L	Lower Middle	2	Anaemia+Sickle Cell Trait+G.Htn	Moderate	8	1600	1720	1836	1944	8	6.2	5.1	2.8	R	R	R	AB	1.01	0.98	0.96	0.92	0.78	0.76	0.74	0.71
7	27	L	Lower Middle	2	Anaemia+Sickle Cell disease+G.Htn	Severe	6.2	1486	1590	1692	1800	11	10.2	9.7	8.5	R	R	R	AB	1.12	1.1	1.09	1.08	0.98	0.89	0.84	0.78
8	28	L	Lower Middle	2	Prev IUGR	No	8.6	1650	1752	1863	1990	11	7	6	4.3	R	R	R	AB	1.17	1.16	1.15	1.14	0.74	0.72	0.7	0.67
9	29	IL	Lower	4	Anaemia+Sickle Cell Trait+G.Htn	Moderate	6.7	1600	1732	1856		10	9	7.8		R	R	R		1.21	1.19	1.2		0.79	0.76	0.74	
10	35	L	Lower Middle	1	Anaemia+Sickle cell disease	Severe	5.8	1482	1559			8	4.5			R	AB			1.1	1.09			0.99	0.96		
11	24	IL	Lower	2	G.Htn	No	7.3	1486	1598	1684	1800	10	8	6	5.4	R	R	R	R	1.21	1.19	1.18	1.17	0.84	0.82	0.8	0.76
12	18	L	Lower Middle	1	Anaemia+Sickle cell disease+GDM+G.Htn	Severe	4	1481				5				AB				1.12				1.34			
13	28	IL	Lower	3	Anaemia+Sickle Cell Trait+G.Htn	Mild	5	1548	1644	1750		11	10.5	8		R	R	AB		1.1	1.08	1.01		0.84	0.82	0.8	
14	26	L	Lower Middle	2	Anaemia+Sickle Cell Trait	Mild	7.6	1650	1766	1840	1964	12	10.6	9	8.4	R	R	R	R	1.05	1.03	1.01	0.98	0.76	0.75	0.73	0.7
15	25	IL	Lower	2	Anaemia+Sickle Cell Trait	Moderate	5.6	1468	1598	1700		10	8	7.4		R	R	AB		1.2	1.13	1.13		0.87	0.86	0.8	
16	26	IL	Lower	3	Anaemia+Sickle Cell Trait+G.Htn	Mild	5.2	1489	1590	1712		10	6	4.3		R	R	AB		1.19	1.18	1.17		1.21	1.18	1.18	
17	27	IL	Lower	3	Anaemia+Sickle Cell Trait+G.Htn	Moderate	6	1488	1590	1700		11	10	9.4		R	R	AB		1.21	1.19	1.17		0.92	0.9	0.87	
18	25	L	Lower Middle	2	Anaemia+Sickle Cell Trait+Prev IUGR	Moderate	7.8	1576	1680	1796	1888	8	7.4	7	4.2	R	R	R	R	1.19	1.17	1.16	1.15	0.84	0.82	0.8	0.78
19	23	L	Lower Middle	1	Prev IUGR	No	8.4	1620	1740	1854	1948	11	10.2	9.4	9	R	R	R	R	1.17	1.15	1.15	1.14	0.78	0.75	0.74	0.71
20	24	IL	Lower	2	GDM	No	7.7	1498	1598	1700		8	7.4	4.3		R	R	AB		1.19	1.18	1.18		0.89	0.88	0.83	
21	25	IL	Lower	3	G.Htn	No	6.7	1610	1704	1800		12	10	8.3		R	R	R		1.21	1.19	1.19		0.78	0.77	0.75	
22	26	IL	Lower	3	GDM	No	7.2	1556	1648	1754		8	6.8	4.3		R	R	AB		1.01	1	0.99		0.88	0.84	0.82	
23	27	IL	Lower	3	GDM	No	9	1618	1722	1840	1956	9	8.6	7.8	7.5	R	R	R	R	1.16	1.15	1.14	1.12	0.8	0.76	0.75	0.74
24	26	L	Lower Middle	2	GDM	No	7.4	1490	1588	1694	1798	11	9.4	7.4	4.5	R	R	R	R	1.18	1.17	1.14	1.12	0.92	0.88	0.87	0.81
25	22	IL	Lower	1	Anaemia	Mild	4.5	1552	1660			7.8	3.6			R	AB			1.22	1.2			0.98	0.92		
26	28	IL	Lower	2	Jaundice	No	8.2	1610	1724	1848	1960	13	10	8.3	8	R	R	R	R	1.19	1.17	1.15	1.15	0.75	0.74	0.73	0.7
27	32	IL	Lower	3	Anaemia+Sickle cell disease+G.Htn+GDM	Moderate	3	1512				3.6				AB				1.16				1.43			
28	30	L	Lower Middle	3	Anaemia+Sickle Cell Trait+G.Htn	Moderate	5.4	1483	1576	1682		10.4	8.6	4.3		R	R	R		1.08	1.02	1.01		0.86	0.84	0.83	
29	29	IL	Lower	3	Anaemia	Mild	4.3	1500	1588			8	3.6			R	AB			1.12	1.09			1.08	1.02		
30	28	L	Lower Middle	2	G.Htn	No	7.5	1578	1682	1796	1900	12	10.4	8.8	8	R	R	R	R	1.2	1.18	1.17	1.16	0.84	0.82	0.81	0.79
31	23	IL	Lower	1	Anaemia+Sickle Cell Trait+G.Htn	Moderate	6.8	1490	1598	1700	1812	11	9.4	8	7.8	R	R	R	R	1.19	1.17	1.16	1.15	0.89	0.87	0.86	0.82
32	27	L	Lower Middle	3	Jaundice	No	7	1556	1648	1762		10	8.6	7.9		R	R	AB		1.2	1.18	1.1		0.85	0.84	0.82	
33	35	IL	Lower	4	Anaemia+Sickle Cell Trait+G.Htn	Moderate	6	1483	1570			8.9	4.6			R	R			1.18	1.17			1.02	1.01		
34	32	L	Lower Middle	2	Prev IUGR	No	8.2	1590	1700	1798	1902	13	11	10.4	9.6	R	R	R	R	1.1	1.07	1.03	1.01	0.78	0.76	0.75	0.73
35	22	L	Lower Middle	1	Anaemia+Prev IUGR	Moderate	8.4	1628	1750	1846	1962	14	12.8	9.7	8.8	R	R	R	R	1.21	1.2	1.19	1.19	0.73	0.71	0.7	0.69
36	25	IL	Lower	1	GDM	No	7.6	1554	1664	1758	1870	10	8.4	6.4	4.1	R	R	R	AB	1.21	1.19	1.18	1.18	0.83	0.82	0.8	0.79
37	31	IL	Lower	3	Anaemia+G.Htn	Mild	4.6	1538	1650			7.4	4.1			R	AB			1.19	1.17			0.99	0.96		
38	27	L	Lower Middle	2	G.Htn	No	6.6	1492	1582	1688	1784	11	10.2	9.8	7.8	R	R	R	R	1.21	1.2	1.19	1.19	0.9	0.89	0.87	0.86
39	29	IL	Lower	2	Anaemia+G.Htn	Mild	5.6	1487	1590			7.4	3.6			R	AB			1.22	1.2			1.23	1.18		
40	27	IL	Lower	3	Anaemia+Sickle Cell Trait	Moderate	7	1588	1696	1800	1928	11	10.4	9.6	8.4	R	R	R	R	1.21	1.2	1.19	1.19	0.84	0.82	0.81	0.79
41	30	L	Lower Middle	2	Anaemia+Sickle Cell Trait+G.Htn	Moderate	6.4	1485	1562	1684		10.4	8.6	4.8		R	R	AB		1.18	1.15	1.14		0.88	0.87	0.85	
42	22	IL	Lower	1	Prev IUGR	No	9	1622	1730	1850	1978	11.6	10.4	8.6	7.8	R	R	R	R	1.19	1.18	1.17	1.17	0.76	0.75	0.73	0.72
43	32	IL	Lower	4	G.Htn	No	4.2	1493	1536			6.8	4.4			R	AB			1.16	1.14			0.92	0.9		
44	34	IL	Lower	4	G.Htn	No	5	1481	1570			6.3	4			R	AB			1.19	1.18			1.14	1.1		
45	32	IL	Lower	3	Anaemia+G.Htn	Mild	4.4	1488	1556			7.2	4.7			R	AB			1.18	1.18			0.94	0.92		
46	27	IL	Lower	2	Anaemia+Sickle Cell Trait+G.Htn	Moderate	6	1584	1692	1800		8.7	7	4.3		R	R	AB		1.19	1.17	1.16		0.92	0.88	0.87	
47	27	L	Lower Middle	2	Prev IUGR	No	8.9	1614	1710	1824	1940	10.8	8.9	7.8	7.4	R	R	R	R	1.1	1.19	1.18	1.17	0.76	0.74	0.73	0.72
48	25	IL	Lower	2	Anaemia+GDM	Moderate	7.8	1598	1706	1802	1932	14.3	10.2	8.8	8.2	R	R	R	R	1.21	1.19	1.17	1.18	0.74	0.73	0.71	0.7
49	28	L	Lower Middle	3	Anaemia	Mild	4.1	1492	1596			6.4	4			R	AB			1.19	1.17			0.89	0.82		
50	28	IL	Lower	2	Prev IUGR	No	8	1576	1688	1794		10.7	9.5	8		R	R	R		1.2	1.17	1.2		0.87	0.84	0.82	
51	29	IL	Lower	2	Anaemia+G.Htn	Mild	5	1498	1578			8.4	6			R	AB			1.21	1.19			0.9	0.88		
52	25	IL	Lower	2	G.Htn	No	7.9	1578	1690	1800		11	10.6	8.8		R	R	R		1.19	1.17	1.17		0.88	0.87	0.85	
53	24	L	Lower Middle	2	Prev IUGR	No	8	1608	1714	1842	1956	11	10.3	8.7	8.2	R	R	R	R	1.21	1.2	1.19	1.19	0.76	0.74	0.72	0.71
54	24	L	Lower Middle	2	GDM+G.Htn	No	7.7	1586	1698	1816	1958	11.8	10.4	9.4	8.8	R	R	R	R	1.2	1.19	1.17	1.16	0.74	0.72	0.7	0.69
55	27	IL	Lower	3	Anaemia+G.Htn	Moderate	6.2	1650	1766	1880		8.9	7.4	4.2		R	R	AB		1.2	1.19	1.17		0.73	0.72	0.7	
56	28	IL	Lower	3	GDM+G.Htn	No	7	1500	1616	1742		14.3	10.8	8.4		R	R	R		1.21	1.2	1.19		0.82	0.8	0.79	
57	35	IL	Lower	1	Anaemia+Sickle cell disease+G.Htn	Severe	3	1482				8.2				AB				1.07				1.45			
58	23	L	Lower Middle	1	Anaemia+G.Htn	Mild	6.7	1497	1599	1698	1784	12.4	10.6	9	8.4	R	R	R	R	1.2	1.19	1.18	1.17	0.98	0.97	0.95	0.92
59	29	IL	Lower	2	Anaemia+Sickle Cell Trait	Mild	6	1480	1584	1690		8.7	7.2	4.3		R	R	R		1.19	1.17	1.17		0.95	0.92	0.91	
60	30	IL	Lower	2	Anaemia+G.Htn	Mild	5.8	1600	1712	1824		10.8	9.4	7.6		R	R	R		1.22	1.21	1.2		0.79	0.76	0.75	
61	29	L	Lower Middle	2	Prev IUGR	No	9.1	1622	1728	1838	1954	13.4	10.3	9.4	9	R	R	R	R	1.21	1.19	1.18	1.17	0.78	0.76	0.74	0.73
62	32	IL	Lower	2	Anaemia	Mild	5.8	1485	1572			10.4	8.2			R	AB			1.21	1.19			0.92	0.9		

Middle Cerebral Artery (MCA)				Cerebroplacental Ratio (CPR)				Obstetric outcome						Neonatal outcome				
PI	PI	PI	PI	MCA PI/UA PI														
34 wks	35 wks	36 wks	37 wks	34 wks	35 wks	36 wks	37 wks	Delivered @Weeks of gestation	Induced/ Spontaneous	Mode of Delivery (Vaginal/LSCS)	Indication	Live/IUFD/ FSB	Morbidity	Birth weight (gm)	APGAR Score @1 min	APGAR Score @ 5 min	Indication of NICU stay	Mortality
1.58	1.3			1.31	0.98			35		Emer.LSCS	AB CTG+CPR reversal+Oligo	Live		1498	3.00	6.00	VLBW+Birth Asphyxia+MAS	Y
1.48	1.38	1.18		1.25	1.17	0.99		36		Elect.LSCS	CPR reversal+Oligo	Live	Wound Gap	1650	4.00	6.00	Hypoglycemia+hyperbilirubinemia	N
1.34	1.28	1.28	1.17	1.14	1.09	1.08	0.99	37	Sponatneous	Vaginal Delivery	CPR reversal	Live		1900	5.00	8.00	MAS+Birth Asphyxia	N
1.4	1.38	1.17		1.19	1.18	1.01		36		Elect.LSCS	CPR borderline+MCA redistribution	Live		1850	7.00	9.00		N
1.3	1.19			1.10	0.99			35		Emer.LSCS	AB CTG+MCA redistribution+CPR reversal	Live	Wound Gap	1550	7.00	9.00		N
1.46	1.3	1.24	1.14	1.45	1.33	1.29	1.24	37		Emer.LSCS	AB CTG+Oligo	Live		1940	7.00	9.00		N
1.3	1.25	1.19	1.06	1.16	1.14	1.09	0.98	37		Emer.LSCS	AB CTG+CPR reversal+MCI redistribution	Live	Wound gap	1810	4.00	6.00	MAS+hypocalcemia	N
1.32	1.29	1.25	1.16	1.13	1.11	1.09	1.02	37		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution+Oligo	Live		1910	6.00	7.00		N
1.4	1.3	1.18		1.16	1.09	0.98		36	Induced	Vaginal Delivery	CPR Reversal	Live	Fever	1860	5.00	7.00	Birth Asphyxia+hypoglycemia	N
1.3	1.18			1.18	1.08			35		Emer.LSCS	AB CTG+MCA redistribution+Oligo	Live	Fever+Wound Gap	1540	6.00	8.00	Hyperbilirubinemia+Hypoglycemia	N
1.35	1.3	1.28	1.14	1.12	1.09	1.08	0.97	37	Induced	Vaginal Delivery	CPR Reversal	Live	Fever	1780	5.00	8.00	Hyperbilirubinemia	N
1.08				0.96				34		Emer.LSCS	AB CTG+CPR reversal+MCA redistribution+Oligo	Live	Fever+Wound Gap	1450	4.00	6.00	VLBW+Birth Asphyxia	Y
1.31	1.26	1.08		1.19	1.17	1.07		36		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution	Live		1720	5.00	9.00		N
1.36	1.28	1.18	1.05	1.30	1.24	1.17	1.07	37		Elect.LSCS	CPR borderline+MCA redistribution	Live		1920	7.00	8.00		N
1.42	1.26	1.15		1.18	1.12	1.02		36		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution	Live	Wound Gap	1710	4.00	8.00		N
1.36	1.29	1.16		1.14	1.09	0.99		36		Emer.LSCS	AB CTG+CPR reversal+MCA redistribution	Live		1700	4.00	7.00	Birth Asphyxia+hypothermia	N
1.45	1.32	1.16		1.20	1.11	0.99		36		Emer.LSCS	AB CTG+CPR reversal+MCA redistribution	Live	Fever+Wound Gap	1690	5.00	7.00	Birth Asphyxia+hypocalemia	N
1.38	1.29	1.26	1.22	1.16	1.10	1.09	1.06	37	Induced	Vaginal Delivery	CPR borderline+Oligo	Live	Fever+Wound Gap	1860	5.00	6.00	Birth Asphyxia+hypoglycemia	N
1.48	1.36	1.28	1.22	1.26	1.18	1.11	1.07	37	Sponatneous	Vaginal Delivery	CPR borderline	Live		1950	7.00	8.00		N
1.36	1.28	1.24		1.14	1.08	1.05		36		Emer.LSCS	AB CTG+CPR borderline+Oligo	Live		1660	4.00	7.00	MAS+Birth Asphyxia+hypoglycemia	N
1.48	1.35	1.18		1.22	1.13	0.99		36	Induced	Vaginal Delivery	CPR Reversal	Live		1810	4.00	8.00	Hyperbilirubinemia	N
1.35	1.28	1.2		1.34	1.28	1.21		36		Emer.LSCS	AB CTG+Oligo	Live		1740	7.00	9.00		N
1.38	1.36	1.25	1.1	1.19	1.18	1.10	0.98	37	Induced	Emer.LSCS	CPR reversal	Live		1980	5.00	7.00	Birth Asphyxia+hypothermia	N
1.34	1.3	1.25	1.11	1.14	1.11	1.10	0.99	37		Elect.LSCS	CPR reversal+Oligo	Live		1750	6.00	8.00		N
1.34	1.23			1.10	1.03			35		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution+Oligo	Live		1620	6.00	9.00		N
1.46	1.36	1.28	1.2	1.23	1.16	1.11	1.04	37	Sponatneous	Vaginal Delivery	CPR borderline	Live		1950	8.00	9.00	Hyperbilirubinemia	N
1.13				0.97				34		Emer.LSCS	AB CTG+CPR reversal+MCA redistribution+Oligo	Live		1442	4.00	5.00	VLBW+Birth Asphyxia	Y
1.38	1.27	1.1		1.28	1.25	1.09		36	Induced	Vaginal Delivery	MCA redistribution+Oligo	Live	Fever+Wound Gap	1660	7.00	9.00		N
1.32	1.18			1.18	1.08			35		Emer.LSCS	AB CTG+MCA redistribution+Oligo	Live		1498	5.00	8.00	VLBW+Hypothermia	N
1.43	1.35	1.28	1.14	1.19	1.14	1.09	0.98	37	Induced	Emer.LSCS	CPR reversal	Live		1870	4.00	9.00	Birth Asphyxia	N
1.58	1.36	1.28	1.19	1.33	1.16	1.10	1.03	37	Sponatneous	Vaginal Delivery	CPR borderline	Live	Fever	1830	4.00	8.00	MAS+hypocalcemia	N
1.38	1.28	1.12		1.15	1.08	1.02		36		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution	Live		1750	4.00	7.00	MAS+Hypocalcemia	N
1.59	1.2			1.35	1.03			35		Elect.LSCS	CPR borderline+MCA redistribution+Oligo	Live		1550	6.00	9.00	Hypoglycemia+Hypocalcemia	N
1.3	1.28	1.18	1.02	1.18	1.20	1.15	1.01	37		Elect.LSCS	CPR borderline+MCA redistribution	Live		1890	5.00	9.00	Hypoglycemia	N
1.68	1.45	1.31	1.16	1.39	1.21	1.10	0.97	37	Induced	Emer.LSCS	CPR reversal	Live	Wound gap	1950	6.00	8.00		N
1.49	1.35	1.28	1.12	1.23	1.13	1.08	0.95	37		Emer.LSCS	AB CTG+CPR reversal+Oligo	Live		1850	4.00	6.00	MAS+Birth Asphyxia	N
1.34	1.2			1.13	1.03			35		Emer.LSCS	AB CTG+Oligo+MCA redistribution+CPR Borderline	Live		1640	8.00	9.00		N
1.82	1.65	1.48	1.25	1.50	1.38	1.24	1.05	37	Sponatneous	Vaginal Delivery	CPR borderline	Live		1800	6.00	9.00		N
1.32	1.25			1.08	1.04			35		Emer.LSCS	AB CTG+CPR borderline+Oligo	Live		1550	7.00	8.00		N
1.36	1.32	1.3	1.21	1.12	1.10	1.09	1.02	37	Sponatneous	Vaginal Delivery	CPR borderline	Live		1920	7.00	9.00		N
1.39	1.3	1.15		1.18	1.13	1.01		36		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution+Oligo	Live		1650	7.00	8.00		N
1.34	1.3	1.27	1.2	1.13	1.10	1.09	1.03	37	Induced	Vaginal Delivery	CPR borderline	Live		1990	7.00	8.00		N
1.32	1.22			1.14	1.07			35		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution+Oligo	Live		1520	5.00	7.00	Birth Asphyxia	N
1.45	1.25			1.22	1.06			35		Emer.LSCS	AB CTG+CPR borderline+Oligo	Live		1510	4.00	6.00	Birth Asphyxia	N
1.36	1.2			1.15	1.02			35		Emer.LSCS	AB CTG+CPR Borderline+MCA redistribution+Oligo	Live	Wound Gap	1550	6.00	9.00	Birth Asphyxia+Hypothermia	N
1.58	1.34	1.19		1.33	1.15	1.03		36		Emer.LSCS	AB CTG+CPR borderline+Oligo	Live		1810	6.00	9.00		N
1.58	1.36	1.28	1.21	1.44	1.14	1.08	1.03	37	Sponatneous	Vaginal Delivery	CPR borderline	Live		1950	7.00	9.00		N
1.45	1.38	1.28	1.22	1.20	1.16	1.09	1.03	37	Induced	Vaginal Delivery	CPR borderline	Live		1930	7.00	8.00		N
1.32	1.25			1.11	1.07			35		Emer.LSCS	AB CTG+CPR borderline+Oligo	Live		1610	6.00	8.00		N
1.34	1.28	1.18		1.12	1.09	0.98		36	Induced	Vaginal Delivery	CPR reversal	Live	Fever	1780	4.00	7.00	MAS+Birth Asphyxia	N
1.31	1.2			1.08	1.01			35		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution	Live		1560	7.00	9.00		N
1.41	1.32	1.22		1.18	1.13	1.04		36	Induced	Vaginal Delivery	CPR borderline	Live		1820	5.00	6.00	MAS+Hyothermia	N
1.67	1.52	1.31	1.27	1.38	1.27	1.10	1.07	37	Sponatneous	Vaginal Delivery	CPR borderline	Live		1930	8.00	9.00		N
1.42	1.36	1.28	1.18	1.18	1.14	1.09	1.02	37	Induced	Vaginal Delivery	CPR borderline	Live		1950	6.00	9.00		N
1.4	1.32	1.24		1.17	1.11	1.06		36		Emer.LSCS	AB CTG+CPR borderline+Oligo	Live	Wound Gap	1890	6.00	9.00		N
1.38	1.32	1.25		1.14	1.10	1.05		36	Induced	Vaginal Delivery	CPR borderline	Live		1740	5.00	8.00		N
1.02				0.95				34		Emer.LSCS	AB CTG+CPR reversal+MCA redistribution	Live	Fever+Wound Gap	1420	3.00	6.00	VLBW+Birth Asphyxia	Y
1.32	1.3	1.29	1.17	1.10	1.09	1.09	1.00	37	Sponatneous	Vaginal Delivery	CPR borderline	Live		1750	8.00	9.00		N
1.31	1.28	1.16		1.10	1.09	0.99		36		Elect.LSCS	CPR reversal+MCA redistribution+Oligo	Live		1680	7.00	9.00		N
1.38	1.31	1.22		1.13	1.08	1.02		36	Induced	Vaginal Delivery	CPR Borderline	Live		1850	6.00	8.00		N
1.4	1.36	1.28	1.23	1.16	1.14	1.08	1.05	37	Sponatneous	Vaginal Delivery	CPR borderline	Live		1980	6.00	8.00		N
1.35	1.2			1.12	1.01			35		Emer.LSCS	AB CTG+CPR borderline+MCA redistribution	Live		1550	8.00	9.00		N