Natl. J. Physiol. Pharm. Pharmacol., (2024), Vol. 14(12): 2636–2651
ISSN: 2320-4672 (Print)
ISSN: 2231-3206 (Online)
Original Research
DOI: *10.5455/NJPPP.2024.v14.i12.23*

Submitted: 30/04/2024 Accepted: 03/11/2024 Published: 31/12/2024

Second trimester placental location as a predictor of adverse pregnancy outcomes

Bini VS[1*], Anitha K Gopal[2] and Bindhu K M[3]

[1]*Assistant Professor, Department of Obstetrics and Gynaecology, Government Medical College Trivandrum, Kerala*
[2]*Additional Professor, Department of Obstetrics and Gynaecology, Government Medical College Kottayam, Kerala*
[3]*Associate Professor, Department of Obstetrics and Gynaecology, Government Medical College Kottayam, Kerala*

ABSTRACT

Background: Placenta forms an important anatomical and physiological connection between the mother and fetus. Placenta is associated with many fetal and maternal problems. It has a key role in the nutrition of the fetus, oxygenation, endocrine, and other body functions. Hence it is important for a healthy pregnancy and its outcomes. The uterine blood supply is not uniformly distributed. The site of the implantation and the resultant location of the placenta within the uterus are likely to be the important determinants of placental blood flow and therefore pregnancy success. Placental location has been implicated in preterm birth, fetal mal-position and mal-presentation, IUGR, Low APGAR score, and development of preeclampsia.

Objectives: The objective of the study is to find out whether there is any significant correlation between second trimester placental location with the following Fetal outcomes like intra uterine growth restriction (IUGR), preterm birth, low APGAR at 1 minute (i.e. <7), NICU admission, abnormal presentation and maternal outcomes such as preeclampsia, abruption, ante partum hemorrhage due to placental praevia, postpartum hemorrhage.

Materials and Methods

Study Design: A prospective observational study was conducted at the Department of Obstetrics and Gynaecology, Government Medical College Hospital in Kerala for a period of 12 months after getting the approval of the Institutional Ethic Committee (IEC)/Institutional Review Board (IRB) of the Institution. Population for the study is antenatal women of the age group 18 to 35 years without any high risk factors. Sample size is calculated as 390 with criteria for inclusion and exclusion. Sampling procedure used is simple random sampling (SRS). The main tools used for the study were case records, obstetric USG, clinical examination findings, and lab reports. The placental location was identified by Ultra Sonography done between 18 and 24 weeks. Location of the placenta was classified as fundal, unilateral, and low for the study. The outcome variables selected for the study comes under two categories i.e. maternal outcomes and fetal outcomes. The maternal outcomes are preeclampsia, placental abruption, ante partum hemorrhage due to placental praevia and post-partum hemorrhage and fetal outcomes are intra uterine growth restriction (IUGR), preterm birth, low APGAR at 1 minute (i.e. <7), NICU admission and abnormal presentation.

Analysis: In chi square test we get a p value. If p value is less than 0.05 then we treat it as a significant association between the variables. If the p value is less than 0.01 then there is highly significant association between the variables in the study.

Results: There is a significant relation between the placental location in the second trimester and the maternal outcomes such as preeclampsia, APH in placental praevia and fetal outcomes such as IUGR, low APGAR at 1 minute (i.e. <7), and NICU admission. It is also found that there is no significant association between placental location in the second trimester and maternal outcomes such as placental abruption, postpartum hemorrhage, and fetal outcomes such as preterm birth and abnormal presentation.

Conclusion: With the help of Ultra Sonography, a simple, noninvasive, easy to perform, cost effective diagnostic method, the location of placenta can easily made out and thereby identifying the above high-risk cases such as IUGR, low APGAR at 1 minute (i.e. <7), preeclampsia and APH in placental praevia. By identifying such patients appropriate treatment can be initiated and regular follow-up can be done in advance.

Keywords: Placenta location, Second trimester, Preeclampsia, Placental abruption, Ante partum hemorrhage due to placental praevia, Post partum hemorrhage, IUGR, Preterm birth, Low APGAR, Abnormal presentation.

***Corresponding Author:** Dr. Bini V. S. Assistant Professor, Department of Obstetrics and Gynaecology, Government Medical College Trivandrum, Kerala, India. Email: *drbinigirish@gmail.com*

INTRODUCTION

Placenta forms an important connecting organ between the mother and fetus. Placenta is connected to the fetus through the umbilical cord. Placenta provides oxygen and nutrients to the developing fetus. It also plays an important role in metabolic exchange, endocrine, and other body functions. It is critical for maternal and fetal wellbeing. So many fetal and maternal problems are associated with uteroplacental insufficiency. The blood supply to the uterus is not uniformly distributed. If the placenta implants in a poorly vascularized area in the uterus, there is a chance of uteroplacental insufficiency. Hence placental location forms an important determining factor in fetal and maternal outcomes (Hadley *et al*., 1990; Kalanithi *et al*., 2007; Hoogland and Haan, 1980; Magann *et al*., 2007).

Placental location in the uterus and its significance in obstetric outcomes were extensively studied. Location of the placenta has been related with fetal position, fetal presentation, prematurity, and pre-eclampsia. Ultrasonography is the most accurate and simple method for determining the location of placenta. Ultrasonography helps us to detect abnormal placental locations and its abnormalities in the later weeks of pregnancy. High-resolution trans vaginal sonography (TVS) has revolutionized the proper identification of placental location especially in low lying placenta.

Routine Ultrasonography is now an integral component of antenatal care. During a routine obstetric ultrasonography, fetus is primarily evaluated along with placenta, umbilical cord, and amniotic fluid. It is a simple, cost effective, easy to perform and noninvasive tool to diagnose high risk factors included in a pregnancy. Appropriate treatment can be started promptly for such patients with high risk factors and they can be followed up regularly so that they will get a better pregnancy outcome.

Blood supply to the uterus is from uterine arteries on both sides, which is a branch of internal iliac artery. Each uterine arteries supplies the corresponding side of the uterus with many of its branches. Also, there are anastomoses between the two uterine arteries and also with the ovarian artery. There is no proof that all the anastomoses are functional. When the placenta is located centrally it will receive almost equal supply from both the uterine arteries. But when the placenta is located laterally either to the left or right it is more supplied by the ipsilateral uterine artery with some contribution form the contralateral uterine artery through collateral circulation. The fact is that the extend of collateral circulation is not the same in all individuals. Poor collateral circulation will lead to uteroplacental insufficiency.

In normal placentation cytotrophoblastic invasion is high. But in preeclampsia, cytotrophoblasts fail to adopt a vascular adhesion phenotype leading to restricted invasion of trophoblastic cells (Khong *et al*., 1986). This may explain the reduced trophoblastic invasion in laterally situated placenta when the uteroplacental blood flow needs are mainly met with by that side uterine artery. Thus, preeclampsia occurs only in the presence of placenta (Walker *et al*., 1998).

Trophoblastic invasion of spiral arteries occurs in two phases. The first phase occurs twelve weeks after fertilization and extends into the decidua basalis. The second phase of invasion extends into the myometrium and is completed by sixteen weeks of post fertilization. Failure of trophoblastic invasion of spiral arteries results in a reduction of placental blood flow and is implicated in the causation of preeclampsia, fetal growth restriction, placental abruption, Prelabour Rupture of Membrane (PROM) and preterm labor (Seshadri and Arjun, 2015).

Remodeling converts narrow lumen, muscular spiral arteries into dilated low resistance uteroplacental vessels. Molecular mechanisms of these crucial events and their significance in the pathogenesis of preeclampsia and fetal growth restriction have been reviewed by Kaufman in 2003 and Red-horse in 2006 (Cunningham *et al*., 2014).

Preeclampsia developing before 32 weeks is early onset type and developing after 32 weeks is late onset type. Early onset type is mainly associated with maternal and fetal high-risk factors. Late onset type is mainly due to uteroplacental insufficiency. It is said that pre-eclampsia and eclampsia contribute to the death of a woman every 3 minutes worldwide (Vidyadhar *et al*., 2011). Infants of women with severe pregnancy induced hypertension have a 5-fold increase in mortality compared to infants of normotensive women (Vidyadhar *et al*., 2011). This has led to the interest in screening tests, in preventing this disorder. There are many clinical, biophysical, and biochemical tests for the early detection of preeclampsia. Some of these tests are simple, whereas others are invasive; some have been studied extensively, while others are still under clinical investigation. There are considerable disagreement regarding the sensitivity and predictive values of the various tests studied. The reported differences in the predictive values of these tests may be attributed to one or more of the following: populations studied, definition and prevalence of the disorder, techniques, and methodology used in performing these tests, etc.

Poor trophoblastic invasion of spiral arterioles leads to the development of preeclampsia, intra uterine growth retardation, or both conditions (Khong *et al*., 1986). Also, unilateral placental location may predispose to the development of preeclampsia and intrauterine growth retardation due to poor vasculature. In the presence of preeclampsia or intrauterine growth retardation, up to 75% of patients had unilaterally located placenta (Kofinas *et al*., 1989).

Intrauterine growth restriction (IUGR), defined most commonly as estimated fetal birth weight (EFBW) below the 10th percentile for gestational age using population-specific fetal growth curves and is associated with high perinatal mortality and morbidity (Chessex *et al*., 1984; Sung *et al*., 1993). IUGR can be early onset and late onset types. Early onset IUGR is one occurring before 32 weeks of gestation and can be due to fetal factors (such as infection and chromosomal or structural anomalies), placental factors (placental abruption), and maternal factors (toxin or drug exposure, illicit drug use, and chronic medical conditions such as hypertension and anemia) (Kuzma and Sokol, 1982; Khoury *et al*., 1988; Holzman and Paneth, 1994; Odegard *et al*., 2000; Resnik*et al*., 2002). Late onset IUGR starting after 32 weeks of gestation is sometimes difficult to diagnose and can cause fetal mortality and morbidity. Main cause for late onset IUGR is uteroplacental insufficiency. Many cases of IUGR remain unexplained.

As we already stated the blood supply of the uterus is not uniformly distributed. Placental location within the uterus is one of the important determinants of placental blood flow and therefore pregnancy success. For example, implantation in the region of a uterine septum is a known cause of miscarriage and recurrent abortion (Proctor and Haney, 2003), and placental location has been implicated in preterm birth, in fetal malposition and malpresentation, and in the development of preeclampsia (Hoogland and de Haan, 1980; Gonser *et al*., 1996). However, the association between placental location and fetal growth has not been well characterized. For example, initial observational studies suggested that placenta previa may be associated with IUGR (Newton *et al*., 1984; Dommisse, 1985) but subsequent larger and better-designed studies have refuted this association (Wolf *et al*., 1991). Reports on the association between IUGR and other placental locations have been similarly conflicting (Vaillant *et al*., 1993). So, this study was designed to investigate further the association between IUGR and placental location.

In complete placenta previa and marginal previa there is a chance for migration of placenta over the course of the pregnancy, and if that location persists, are delivered by cesarean section. If the leading edge of the placenta remains within 2 cm of the internal OS over the course of the pregnancy, this frequently leads to a cesarean delivery because of the unacceptable high risk of hemorrhage with a vaginal delivery (Bhide *et al*., 2003). Pregnancies complicated by placenta previa are reported to have a reduced risk of hypertensive disorders of pregnancy compared with normally implanted placentas (Leiberman *et al*., 1991) and low lying placentas (placentas in the lower uterine segment but which are not placenta previa) are associated with an increased risk of postpartum hemorrhage (Bobrowski and Jones, 1995). Other placental implantation sites have been associated with adverse maternal and neonatal outcomes in small investigations. Unilateral placental implantations (placentas where the bulk of the placenta is implanted over the right or left lateral aspect of the uterus) have been linked with an increased incidence of preeclampsia, fetal distress in labor, abdominal deliveries and IUGR (Gonser *et al*., 1996). Cornual implantations have been linked with an increased risk of breech presentations (Fianu and Vaclavinkova, 1978), persistent placenta previa with fetal growth restriction, abruption placenta, and third trimester bleeding (Newton *et al*., 1984).

Even though the placental location is documented regularly at the time of the second trimester ultrasound evaluation of the fetal anomalies, a detailed appraisal of that location, with the exception of placenta previa, and its predictability of adverse perinatal and maternal outcomes in a large prospective study has not been undertaken. The purpose of this investigation is to find out whether the initial site of placental implantation, as determined by ultrasound between 14 and 22 weeks of gestation, is predictive of ante partum complications, intra partum events, and neonatal outcomes.

The changes that take place in the uterine artery blood flow in pregnancy are of utmost importance. Various studies have been undertaken to assess the effect of various factors on the uterine blood flow in normal and abnormal pregnancies (Caton and Kalra, 1986).

Several methods were used to document placental location, including manual exploration of the uterus, soft tissue x-ray films, and isotopic placentography in the past. Nowadays, ultrasound is the safest, easiest, and most accurate method for assessing placental location. Also, doppler provides a noninvasive method for the study of uteroplacental circulation and fetal hemodynamics. The ratio of the systolic peak velocity and end-diastolic velocity (S/D) has been used as an expression of uterine vascular resistance.

Screening ultra sonography (USG) is a part of routine antenatal care to all pregnant women and it usually focuses on gestational age, amniotic fluid volume, fetal anatomic survey, and placental location. Yet then a limited number of investigators have dealt with placental implantation sites and pregnancy outcomes (Newton *et al*., 1984; Leiberman *et al*., 1991). There has been extensive research on low placental implantation because of the importance of detecting placenta previa. Only a few studies have been undertaken on other aspects of placental position and possible impact on pregnancy outcome. These studies reported that placental location might have implications for poor pregnancy outcomes including preterm birth (Hadley *et al*., 1990), small for gestational age (SGA) (Kalanithi *et al*., 2007) fetal malposition, malpresentation, and the development of pre-

eclampsia (Hoogland and de Haan, 1980). In theory, lateral placental location could contribute a higher risk of fetal IUGR. A case-control trial conducted in USA revealed that women with their placenta located in the fundus carry an increased risk of premature rupture of membranes with all the consequential adverse sequelae (Hadley *et al*., 1990). Reports on an association between IUGR and placental locations (other than placenta previa) have been conflicting (Kofinas *et al*., 1989; Vaillant *et al*., 1993).

Thus, a limited number of studies were there to establish the relationship of second trimester placental location and adverse fetal and maternal outcomes. It is designed to study this topic so that preeclampsia, IUGR, etc. can be effectively detected early and provide appropriate preventive and therapeutic measures.

Based on these observations a prospective study to find out whether the placental location as seen by ultrasound between 18 and 24 weeks of gestation can be used in the prediction of adverse pregnancy outcomes. Results of this study may provide insight that second trimester placental localization by USG can be used as an effective screening modality for various adverse pregnancy outcomes.

The objective of the study, second trimester placental location as a predictor of adverse pregnancy outcomes is to find out whether there is any significant correlation between second trimester placental location the fetal outcomes such as intra uterine growth restriction (IUGR), preterm birth, low APGAR (Appearance Pulse Grimace Activity Respiration) at 1 minute (i.e. <7), NICU Admission and Abnormal Presentation and maternal outcomes such as preeclampsia, abruption, antepartum hemorrhage due to placenta previa and postpartum hemorrhage.

OBJECTIVES

The objective of the study, second trimester placental location as a predictor of adverse pregnancy outcomes is to find out whether there is any significant correlation between second trimester placental locations with the following fetal outcomes such as

- Intra Uterine Growth Restriction (IUGR)
- Preterm Birth
- Low APGAR[1] at 1 minute (i.e. <7)
- NICU Admission
- Abnormal Presentation

and maternal outcomes such as

- Preeclampsia
- Placental Abruption
- Ante Partum Haemorrhage (APH) due to Placental Praevia
- Post Partum Hemorrhage

MATERIALS AND METHODS

This prospective observational study was conducted at the Department of Obstetrics and Gynaecology, in a Tertiary Care Medical College Hospital for a period of 12 months after getting the approval of the Institutional Ethic Committee (IEC)/ Institutional Review Board (IRB). Population for the study is Antenatal women of the age group 18 to 35 years without any high-risk factors who receive antenatal care at the same hospital. Incidence of adverse outcomes varies considerably from one study to another. Due to wide variability, a conservative estimate of 50% was used in the sample size calculation. As the outcome is measured as proportions, the sample size is calculated using the formula n= [(Z1-α/2)2p(1-p)]/ d 2, where Z1-α/2= 1.96, p=0.5, d=0.05 and the sample size is 390. Simple Random Sampling (SRS) was the sampling method used for this study.

The criteria for inclusion of pregnant ladies in this study were Intra-uterine pregnancy of gestational age 18–24 weeks, maternal age 18–35 years, and singleton pregnancy. Pregnant ladies excluded from this study were women in the high-risk pregnancy category (due to chronic hypertension, diabetes, renal disease, previous history of preeclampsia/eclampsia, previous history of preterm labour or preterm delivery, fetal anomalies, uterine anomalies, etc) and Patients not willing for follow up.

Exposure variable for the study is the location of placental implantation. For this study, the site of implantation of placenta was classified as fundal, low, and unilateral. The outcome variables are preeclampsia, placental abruption, ante partum hemorrhage due to placental praevia, postpartum hemorrhage, intra uterine growth restriction (IUGR), preterm birth, low APGAR at 1 minute (i.e. <7), Neonatal Intensive Care Unit (NICU) admission and abnormal presentation. All these outcome variables have values yes or no.

Study procedure

This study was conducted as an observational study and the main tools used for the study were case records, obstetric USG, clinical examination findings and lab reports. Sample size for this study was 390. After applying inclusion and exclusion criteria, women will be taken up for the study through simple random sampling after getting their consent. A detailed history was taken and clinical examinations were done according to the case record form given in the annexure. The location of placenta was identified by ultrasound sonography done between 18 and 24 weeks in all 390 women in the OBG department. Location of placenta

[1] APGAR means Score of the Baby on Activity (muscle tone), Pulse, Grimace (reflex irritability), Appearance (Skin color), and Respiration.

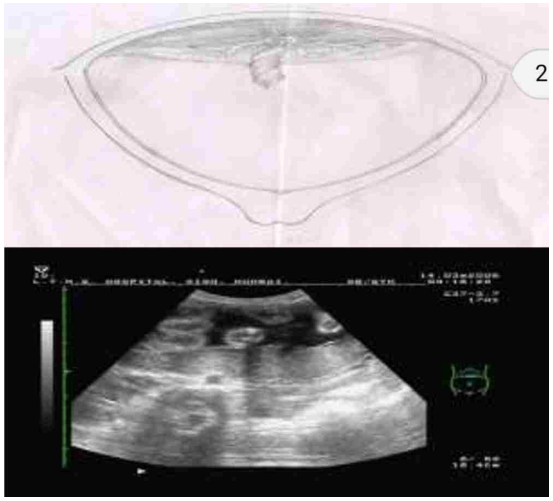

Fig. 1. Placental location – Fundal.

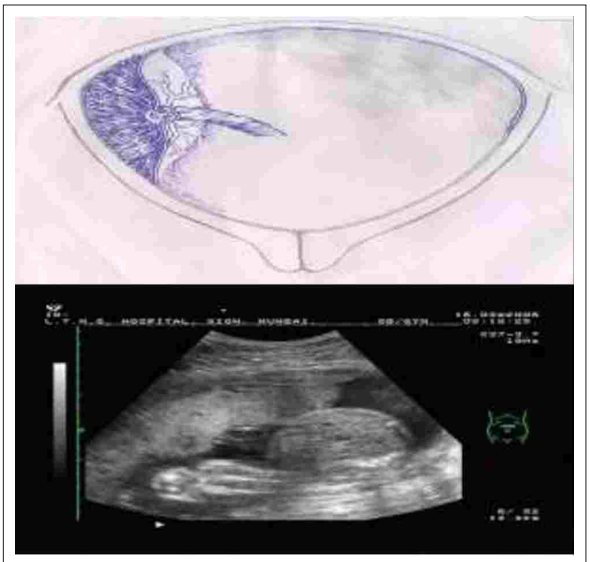

Fig. 2. Placental location – Unilateral.

was classified as follows for the study. Fundal: placenta equally distributed between the right and left side of uterus irrespective of anterior or posterior (Figure 1), Unilateral: more than 75% of placental mass is towards one side of the midline it is classified as unilateral right or left placenta (Figure 2) and Low: placenta is in lower uterine segment.

The outcome variables selected for the study come under two categories i.e. maternal outcomes and fetal outcomes. The possible values for all the outcome variables are yes or no. The values for the outcome variables were collected at the time of follow-up and delivery for all the women included in the study.

The maternal outcome variables selected for the study are as follows. **Preeclampsia:** gestational hypertension associated with proteinuria. (Seshadri and Arjun, 2015), **Placental Abruption:** separation of the placenta either partially or totally from its implantation site before delivery (Cunningham et al., 2014), **Ante Partum Hemorrhage due to placental praevia:** the painless vaginal bleeding after 28 weeks in low lying placenta (Khong et al., 1986) and **Post Partum Haemorrhage (PPH):** the excessive bleeding from the genital tract after delivery (Kofinas et al., 1989). The fetal outcome variables selected for the study are as follows. **Intra Uterine Growth Restriction (IUGR):** IUGR babies are those whose weights were below 10th percentile for their gestational age (Chessex et al., 1984), Preterm Birth: Preterm infants were those delivered before 37 completed weeks of gestation (Sung et al., 1993), **Low APGAR at 1 minute (i.e. <7):** APGAR score provides a convenient short hand for reporting the status of the newborn infant and the response to the resuscitation and APGAR score comprises of five components; Activity (muscle tone), Pulse (Heart rate), Grimace (reflex irritability), Appearance (Skin color), and Respiration. Each of which is given a score of 0, 1 or 2. The score is reported at one and five minutes after birth. A score of seven to ten is considered normal (Kuzma and Sokol, 1982), **NICU Admission:** Neonates admitted to NICU within 24 hours of birth and **Abnormal Presentation:** Presentation other than vertex is considered as abnormal presentation.

The above mentioned four maternal outcomes and five fetal outcomes in these three placental location groups were collected and tabulated using Microsoft Excel. For studying the relation between outcomes and the placental location the tool Chi square test was used. Chi square test of independence was used to find out the dependence of the two variables. The data in MS Excel file export into SPSS (Statistical Package for Social Sciences software. With the help of IBM SPSS software, the chi square values were calculated and the graphs for various analyses were generated. The variable placenta location has three possible values and each pregnancy outcome has two possible values. Therefore, the degree of freedom is $(r-1) \times (c-1)$ i.e. 2. The result of Chi square test is a p value. If p value is less than 0.05 then there exists a significant association between the variables. If the p value is less than 0.01 then there is a highly significant association between the variables in the study.

For testing the independence of the variables, a null hypothesis and an alternative hypothesis are to be defined in chi square test. **Null Hypothesis:** There is no association between the two variables; and **Alternative Hypothesis**: There is a significant association between the variables.

If the p value is >0.05 we accept the null hypothesis otherwise alternative hypothesis will be accepted. In this study, the aim is to check the association of 9 outcome variables with the location of placenta in the

second trimester. So, 9 null hypotheses and 9 alternative hypothesis were designed for the study. The hypotheses are defined as follows:

1. Null Hypothesis: No association between second trimester placental location and IUGR. Alternative Hypothesis: Significant association between second trimester placental location and IUGR.
2. Null Hypothesis: No association between second trimester placental location and preterm birth. Alternative Hypothesis: Significant association between second trimester placental location and preterm birth.
3. Null Hypothesis: No association between second trimester placental location and low APGAR at 1 minute. Alternative Hypothesis: Significant association between second trimester placental location and low APGAR at 1 minute.
4. Null Hypothesis: No association between second trimester placental location and NICU admission. Alternative Hypothesis: Significant association between second trimester placental location and NICU admission.
5. Null Hypothesis: No association between second trimester placental location and abnormal presentation. Alternative Hypothesis: Significant association between second trimester placental location and abnormal presentation.
6. Null Hypothesis: No association between second trimester placental location and preeclampsia. Alternative Hypothesis: Significant association between second trimester placental location and preeclampsia.
7. Null Hypothesis: No association between second trimester placental location and placental abruption. Alternative Hypothesis: Significant association between second trimester placental location and placental abruption.
8. Null Hypothesis: No association between second trimester placental location and ante partum hemorrhage due to placental previa. Alternative Hypothesis: Significant association between second trimester placental location and ante partum hemorrhage due to placental previa.
9. Null Hypothesis: No association between second trimester placental location and postpartum hemorrhage. Alternative Hypothesis: Significant association between second trimester placental location and postpartum hemorrhage.

RESULTS AND OBSERVATIONS

With the help of IBM SPSS software, chi square test was conducted and the result of the study is furnished below.

Placental location in second trimester vs IUGR

The data tabulation and analysis of Placental location in second trimester verses IUGR is shown in Table 1. The p value obtained for the chi square test conducted between the variables placental location in second trimester and IUGR is 0.0001 (Table 2). The p value is very less than 0.01 so the null hypothesis was rejected and the alternate hypothesis was accepted. That is there is highly significant association between

Table 1. Crosstab for Placental_Location_II_Trimester X IUGR.

Placental location in second trimester		IUGR		Total
		N	Y	
Fundal	Fundal cases	219	27	246
	% of IUGR w.r.t. total fundal cases	89.0%	11.0%	
	% of fundal cases w.r.t. IUGR	70.0%	35.1%	
	% of fundal w.r.t. sample size	56.2%	6.9%	63.1%
Low	Low cases	41	9	50
	% of IUGR w.r.t. total low cases	82.0%	18.0%	
	% of low cases w.r.t. IUGR	13.1%	11.7%	
	% of low w.r.t. sample size	10.5%	2.3%	12.8%
Unilateral	Unilateral cases	53	41	94
	% of IUGR w.r.t. total unilateral cases	56.4%	43.6%	
	% of unilateral cases w.r.t. IUGR	16.9%	53.2%	
	% of unilateral w.r.t. Sample size	13.6%	10.5%	24.1%
Total	Cases irrespective of position	313	77	390
	% of IUGR w.r.t. sample size	80.3%	19.7%	

Table 2. Chi-square tests result for Placental_Location_II_Trimester X IUGR.

Chi-square tests: Placental_Location_II_Trimester X IUGR			
	Value	df	Asymptotic significance (2-sided)
Pearson chi-square	45.842	2	0.000
Likelihood ratio	41.376	2	0.000
No of valid cases	390		

Table 3. Crosstab for Placental_Location_II_Trimester X preterm birth.

Placental location in second trimester		Preterm birth		Total
		N	Y	
Fundal	Fundal cases	218	28	246
	% of preterm birth w.r.t. total fundal cases	88.6%	11.4%	
	% of fundal cases w.r.t. preterm birth	64.1%	56.0%	
	% of fundal w.r.t. sample size	55.9%	7.2%	63.1%
Low	Low cases	42	8	50
	% of preterm birth w.r.t. total low cases	84.0%	16.0%	
	% of low cases w.r.t. preterm birth	12.4%	16.0%	
	% of low w.r.t. sample size	10.8%	2.1%	12.8%
Unilateral	Unilateral cases	80	14	94
	% of preterm birth w.r.t. total unilateral cases	85.1%	14.9%	
	% of unilateral cases w.r.t. preterm birth	23.5%	28.0%	
	% of unilateral w.r.t. sample size	20.5%	3.6%	24.1%
Total	Cases irrespective of position	340	50	390
	% of preterm birth w.r.t. sample size	87.2%	12.8%	

Table 4. Chi-square tests result for Placental_Location_II_Trimester X preterm birth.

Chi-square tests: Placental_Location_II_Trimester X preterm birth			
	Value	df	Asymptotic significance (2-sided)
Pearson chi-square	1.269	2	0.530
Likelihood ratio	1.241	2	0.538
No of valid cases	390		

the second trimester placental location and the fetal outcome IUGR.

The study reveals a strong association between the second trimester placental location and IUGR. Out of the 390 samples studied, unilateral placenta location is observed in 94 cases. 43.6% of the unilateral cases have subsequent IUGR (Table 1). As the association of placental location and IUGR had statistically proved, the high prevalence of IUGR in unilateral cases will exist in the population also. By identifying such patients earlier, better care and treatment can be given.

Placental_Location_II_Trimester X preterm birth

The data tabulation and analysis of Placental location in second trimester versus preterm birth is shown in Table 3. The p value obtained for the chi square test conducted between the variables placental location in second trimester and preterm birth is 0.530 (Table 4). The p value is higher than 0.01 so the null hypothesis was accepted and the alternate hypothesis was rejected. That is there is no significant association between the second trimester placental location and the fetal outcome of preterm birth.

Placental_Location_II_Trimester X low APGAR at 1 minute (i.e. <7)

The data tabulation and analysis of Placental location in second trimester verses low APGAR at 1 minute (i.e. <7) is shown in Table 5. The p value obtained for the chi square test conducted between the variables

Table 5. Crosstab for Placental_Location_II_Trimester X Low APGAR at 1 minute (i.e. <7).

Placental_Location_II_Trimester		Low APGAR at 1 minute (i.e. <7)		Total
		N	Y	
Fundal	Fundal cases	239	7	246
	% of low APGAR at 1 minute w.r.t. total fundal cases	97.2%	2.8%	
	% of fundal cases w.r.t. Low APGAR at 1 minute	64.4%	36.8%	
	% of fundal w.r.t. Sample size	61.3%	1.8%	63.1%
Low	Low cases	49	1	50
	% of low APGAR at 1 minute w.r.t. total low cases	98.0%	2.0%	
	% of low cases w.r.t. Low APGAR at 1 minute	13.2%	5.3%	
	% of low w.r.t. sample size	12.6%	0.3%	12.8%
Unilateral	Unilateral cases	83	11	94
	% of low APGAR at 1 minute w.r.t. total unilateral cases	88.3%	11.7%	
	% of unilateral cases w.r.t. Low APGAR at 1 minute	22.4%	57.9%	
	% of unilateral w.r.t. sample size	21.3%	2.8%	24.1%
Total	Cases irrespective of position	371	19	390
	% of low APGAR at 1 minute w.r.t. sample size	95.1%	4.9%	

Table 6. Chi-square tests result for Placental_Location_II_Trimester X Low APGAR at 1 minute (i.e. <7).

Chi-square tests: Placental_Location_II_Trimester X Low APGAR at 1 minute			
	Value	df	Asymptotic significance (2-sided)
Pearson chi-square	12.532	2	0.002
Likelihood ratio	10.591	2	0.005
No of valid cases	390		

placental location in second trimester and low APGAR at 1 minute (i.e. <7) is 0.002 (Table 6). The p value is very less than 0.01 so the null hypothesis was rejected and the alternate hypothesis was accepted. That is there is a significant association between the second trimester placental location and the fetal outcome low APGAR at 1 minute (i.e. <7).

Second trimester placental location and low APGAR in one minute is highly correlated. So, the observation seen in the study is applicable to the population also. About 11.7% of unilateral placenta location has an outcome of low APGAR at one minute (Table 5).

Placental_Location_II_Trimester X NICU admission

The data tabulation and analysis of Placental location in second trimester verses NICU admission is shown in Table 7. The p value obtained for the chi square test conducted between the variables placental location in second trimester and NICU admission is 0.011(Table 8). The p value is very less than 0.05 so the null hypothesis was rejected and the alternate hypothesis was accepted. That is there is relatively significant association between the second trimester placental location and the fetal outcome NICU admission.

In this study, there is a significant association between placental location and NICU admission. About 24.5% of unilateral placenta location has an outcome of NICU admission (Table 7).

Placental_Location_II_Trimester X abnormal presentation

The data tabulation and analysis of Placental location in second trimester verses abnormal presentation is shown in Table 9. The p value obtained for the chi square test conducted between the variables placental location in second trimester and abnormal presentation is 0.787(Table 10). The p value is higher than 0.01 so the null hypothesis was accepted and the alternate hypothesis was rejected. That is there is no significant association between the second trimester placental 4 trlocation and the fetal outcome abnormal presentation.

Table 7. Crosstab for Placental_Location_II_Trimester X NICU admission.

Placental_Location_II_Trimester		NICU Admission		Total
		N	Y	
Fundal	Fundal cases	218	28	246
	% of NICU admission w.r.t. total Fundal cases	88.6%	11.4%	
	% of fundal cases w.r.t. NICU admission	65.9%	47.5%	
	% of fundal w.r.t. Sample size	55.9%	7.2%	63.1%
Low	Low cases	42	8	50
	% of NICU admission w.r.t. total low cases	84.0%	16.0%	
	% of low cases w.r.t. NICU admission	12.7%	13.6%	
	% of low w.r.t. sample size	10.8%	2.1%	12.8%
Unilateral	Unilateral cases	71	23	94
	% of NICU admission w.r.t. total unilateral cases	75.5%	24.5%	
	% of unilateral cases w.r.t. NICU admission	21.5%	39.0%	
	% of unilateral w.r.t. sample size	18.2%	5.9%	24.1%
Total	Cases irrespective of position	331	59	390
	% of NICU admission w.r.t. sample size	84.9%	15.1%	

Table 8. Chi-square tests result for Placental_Location_II_Trimester X NICU admission.

Chi-square tests: Placental_Location_II_Trimester XNICU admission			
	Value	df	Asymptotic significance (2-sided)
Pearson chi-square	9.105	2	0.011
Likelihood ratio	8.490	2	0.014
No of valid cases	390		

Table 9. Crosstab for Placental_Location_II_Trimester X abnormal presentation.

Placental_Location_II_Trimester		Abnormal presentation		Total
		N	Y	
Fundal	Fundal cases	237	9	246
	% of abnormal presentation w.r.t. total fundal cases	96.3%	3.7%	
	% of fundal cases w.r.t. abnormal presentation	63.4%	56.3%	
	% of fundal w.r.t. sample size	60.8%	2.3%	63.1%
Low	Low cases	48	2	50
	% of abnormal presentation w.r.t. total low cases	96.0%	4.0%	
	% of low cases w.r.t. abnormal presentation	12.8%	12.5%	
	% of low w.r.t. sample size	12.3%	0.5%	12.8%
Unilateral	Unilateral cases	89	5	94
	% of abnormal presentation w.r.t. total unilateral cases	94.7%	5.3%	
	% of unilateral cases w.r.t. abnormal presentation	23.8%	31.3%	
	% of unilateral w.r.t. sample size	22.8%	1.3%	24.1%
Total	Cases irrespective of position	374	16	390
	% of abnormal presentation w.r.t. sample size	95.9%	4.1%	

Table 10. Chi-square tests result for Placental_Location_II_Trimester X abnormal presentation.

Chi-square tests: Placental_Location_II_Trimester X abnormal presentation			
	Value	**df**	**Asymptotic Significance (2-sided)**
Pearson chi-square	0.478	2	0.787
Likelihood ratio	0.454	2	0.797
No of valid cases	390		

Table 11. Crosstab for Placental_Location_II_Trimester X preeclampsia.

Placental_Location_II_Trimester		**Preeclampsia**		**Total**
		N	**Y**	
Fundal	Fundal cases	240	6	246
	% of preeclampsia w.r.t. total fundal cases	97.6%	2.4%	
	% of fundal cases w.r.t. preeclampsia	64.7%	31.6%	
	% of fundal w.r.t. sample size	61.5%	1.5%	63.1%
Low	Low cases	49	1	50
	% of preeclampsia w.r.t. total low cases	98.0%	2.0%	
	% of low cases w.r.t. preeclampsia	13.2%	5.3%	
	% of low w.r.t. sample size	12.6%	0.3%	12.8%
Unilateral	Unilateral cases	82	12	94
	% of preeclampsia w.r.t. total unilateral cases	87.2%	12.8%	
	% of Unilateral cases w.r.t. preeclampsia	22.1%	63.2%	
	% of unilateral w.r.t. sample size	21.0%	3.1%	24.1%
Total	Cases irrespective of position	371	19	390
	% of preeclampsia w.r.t. sample size	95.1%	4.9%	

Table 12. Chi-square tests result for Placental_Location_II_Trimester X preeclampsia.

Chi-square tests: Placental_Location_II_Trimester X preeclampsia			
	Value	**df**	**Asymptotic significance (2-sided)**
Pearson chi-square	16.671	2	0.000
Likelihood ratio	13.865	2	0.001
No of valid cases	390		

Placental_Location_II_Trimester X preeclampsia

The data tabulation and analysis of placental location in second trimester verses preeclampsia is shown in Table 11. The p value obtained for the chi square test conducted between the variables Placental location in second trimester and Preeclampsia is 0.0001 (Table 12). The p value is very less than 0.01 so the null hypothesis was rejected and the alternate hypothesis was accepted. That is there is significant association between the second trimester placental location and the maternal outcome preeclampsia.

Second trimester placental location and preeclampsia is highly correlated. So, the observation seen in the study is applicable to the population also. About 12.8% of unilateral placenta location has an outcome of preeclampsia (Table 11).

Placental_Location_II_Trimester X placental abruption

The data tabulation and analysis of Placental location in Second Trimester verses Placental Abruption is shown in Table 13. The p value obtained for the chi square test conducted between the variables placental location in second trimester and Placental Abruption 0.427 (Table 14).The p value is higher than the 0.01 so the null hypothesis was accepted and the alternate

Table 13. Crosstab for Placental_Location_II_Trimester X placental abruption.

Placental_Location_II_Trimester		Placental abruption		Total
		N	Y	
Fundal	Fundal cases	242	4	246
	% of placental abruption w.r.t. total fundal cases	98.4%	1.6%	
	% of fundal cases w.r.t. placental abruption	63.2%	57.1%	
	% of fundal w.r.t. Sample size	62.1%	1.0%	63.1%
Low	Low cases	48	2	50
	% of placental abruption w.r.t. total low cases	96.0%	4.0%	
	% of low cases w.r.t. placental abruption	12.5%	28.6%	
	% of low w.r.t. sample size	12.3%	0.5%	12.8%
Unilateral	Unilateral cases	93	1	94
	% of placental abruption w.r.t. total unilateral cases	98.9%	1.1%	
	% of unilateral cases w.r.t. placental abruption	24.3%	14.3%	
	% of unilateral w.r.t. sample size	23.8%	0.3%	24.1%
Total	Cases irrespective of position	383	7	390
	% of placental abruption w.r.t. sample size	98.2%	1.8%	

Table 14. Chi-square tests result for Placental_Location_II_Trimester X placental abruption.

Chi-square tests: Placental_Location_II_Trimester X placental abruption			
	Value	df	Asymptotic significance (2-sided)
Pearson chi-square	1.704	2	0.427
Likelihood ratio	1.400	2	0.497
No of valid cases	390		

hypothesis was rejected. That is there is no significant association between the second trimester placental location and the maternal outcome placental abruption.

Placental_Location_II_Trimester X APH in placental previa

The data tabulation and analysis of placental location in second trimester verses APH in placental praevia is shown in Table 15. The p value obtained for the chi square test conducted between the variables placental location in second trimester and APH in placental praevia is 0.0001 (Table 16). The p value is very less than 0.01 so the null hypothesis was rejected and the alternate hypothesis was accepted. That is there is significant association between the second trimester placental location and the fetal outcome APH in placental praevia. The APH in Placental Praevia is related to low lying placenta only and it comes about 8% of the low lying placenta in Second Trimester (Table 15).

In this study it is observed that 50 cases have low lying placenta in the second trimester. In such cases repeat USG was done at 34 weeks. Out of the 50 cases only 15 cases remained as low lying. That is 30% of low lying in second trimester remained as low lying in 34 weeks and remaining 70% of the cases had under gone placental migration by 34 weeks. Out of the 15 cases remained in low lying 4 cases had APH. That is 20% of the low lying cases in the third trimester had APH.

Placental_Location_II_Trimester X post partum hemorrhage

The data tabulation and analysis of Placental location in second trimester verses postpartum hemorrhage is shown in Table 17. The p value obtained for the chi square test conducted between the variables placental location in second trimester and postpartum hemorrhage is 0.552 (Table 18). The *p* value is higher than 0.01 so the null hypothesis was accepted and the alternate hypothesis was rejected. That is there is no significant association between the second trimester placental location and the maternal outcome postpartum hemorrhage.

Table 15. Crosstab for Placental_Location_II_Trimester X APH in placental praevia.

Placental_Location_II_Trimester		APH in placental praevia		Total
		N	Y	
Fundal	Fundal cases	246	0	246
	% of APH in placental praevia w.r.t. total fundal cases	100.0%	0.0%	
	% of fundal cases w.r.t. APH in placental praevia	63.7%	0.0%	
	% of fundal w.r.t. sample size	63.1%	0.0%	63.1%
Low	Low cases	46	4	50
	% of APH in placental praevia w.r.t. total low cases	92.0%	8.0%	
	% of low cases w.r.t. APH in placental praevia	11.9%	100.0%	
	% of low w.r.t. sample size	11.8%	1.0%	12.8%
Unilateral	Unilateral cases	94	0	94
	% of APH in placental praevia w.r.t. total unilateral cases	100.0%	0.0%	
	% of unilateral cases w.r.t. APH in placental praevia	24.4%	0.0%	
	% of unilateral w.r.t. sample size	24.1%	0.0%	24.1%
Total	Cases irrespective of position	386	4	390
	% of APH in placental praevia w.r.t. sample size	99.0%	1.0%	

Table 16. Chi-square tests result for Placental_Location_II_Trimester X APH in placental praevia.

Chi-square tests: Placental_Location_II_Trimester X APH in placental praevia			
	Value	df	Asymptotic Significance (2-sided)
Pearson chi-square	27.482	2	0.000
Likelihood ratio	16.721	2	0.000
No of valid cases	390		

Table 17. Crosstab for Placental_Location_II_Trimester X post partum hemorrhage.

Placental_Location_II_Trimester		Post Partum Hemorrhage		Total
		N	Y	
Fundal	Fundal cases	228	18	246
	% of APH in post partum hemorrhage w.r.t. total fundal cases	92.7%	7.3%	
	% of fundal cases w.r.t. post partum hemorrhage	62.6%	69.2%	
	% of fundal w.r.t. sample size	58.5%	4.6%	63.1%
Low	Low cases	46	4	50
	% of post partum hemorrhage w.r.t. total low cases	92.0%	8.0%	
	% of low cases w.r.t. post partum hemorrhage	12.6%	15.4%	
	% of low w.r.t. sample size	11.8%	1.0%	12.8%
Unilateral	Unilateral cases	90	4	94
	% of post partum hemorrhage w.r.t. total unilateral cases	95.7%	4.3%	
	% of unilateral cases w.r.t. post partum hemorrhage	24.7%	15.4%	
	% of unilateral w.r.t. sample size	23.1%	1.0%	24.1%
Total	Cases irrespective of position	364	26	390
	% of post partum hemorrhage w.r.t. sample size	93.3%	6.7%	

Table 18. Chi-Square Tests Result for Placental_Location_II_Trimester X post partum hemorrhage.

Chi-square tests: Placental_Location_II_Trimester X post partum hemorrhage			
	Value	**df**	**Asymptotic Significance (2-sided)**
Pearson chi-square	1.189	2	0.552
Likelihood ratio	1.297	2	0.523
No of valid cases	390		

DISCUSSION

There were multiple studies that established an association of placental location and adverse pregnancy outcomes like preeclampsia, IUGR, preterm, and still birth. Also there are studies about the changes in the uterine artery blood flow in normal pregnancies and their correlation with the placental location demonstrated a significant relationship between placental location and uterine artery Systole / Diastole (S/D) ratio in uncomplicated pregnancies. High uterine artery resistance leads to decreased uterine artery blood flow and adverse pregnancy outcomes.

In this study "Second Trimester Placental Location as a predictor of adverse pregnancy outcomes" proved that there is a statistically significant association between second trimester placental location and fetal outcomes like IUGR, low APGAR at 1 minute, NICU admissions and maternal outcomes like preeclampsia. Another observation from this study is that those with the low placenta in second trimester who were followed up at 34 weeks, 70% were found to have placental migration and 30% remain to have low lying placenta. Four cases of APH are reported from this 30%.

Placental location Vs IUGR

In this study association between the lateral location of the placenta and IUGR was statistically significant (p value << 0.001). That is IUGR is four times more in lateral placenta than fundal or low. This is comparable with Kalanithi *et al*. (2007) who studied that in IUGR babies' chances to find lateral placenta are more than 3.8 times than normal babies.

In contrast, a population-based case cohort study of more than 3000 pregnancies by Magann *et al*. (2007) reported no association between IUGR and placental location. Although some investigators have found some association between IUGR and low lying placenta (Newton *et al*., 1984; Dommisse, 1985; Ananth *et al*., 2001) others have been unable to confirm this association. This study did not find an association between a low lying placenta and IUGR because of the small number of low lying placenta and placenta previa studied.

It is not immediately clear why lateral placental location in the second trimester may be associated with IUGR. A possible explanation is that the lateral region of the uterus may receive less total blood flow from the single ipsilateral uterine artery than the central region, which can derive flow from both uterine arteries and the numerous vascular anastomoses that develop. Lateral placental location may thus predispose to uteroplacental insufficiency and IUGR in some women. Indeed data from several simulation models of uteroplacental circulation and from studies examining the association between placental location and uterine artery Doppler velocimetry (Kofinas *et al*., 1988, 1989; North *et al*., 1994; Ito *et al*., 1998) support the hypothesis that site of placental attachment in the uterus may be an important determinant of placental blood flow. This explanation is consistent with the fact that preeclampsia which shares with IUGR, the common pathologic mechanism of shallow endovascular trophoblastic invasion leading to uteroplacental insufficiency has been associated with lateral placental location (Gonser *et al*., 1996; Booth *et al*., 1962).

Placental location vs Preeclampsia

Doppler studies have shown that abnormal uterine artery flow velocimetry waveforms are more likely to occur in pregnancies with lateral placental locations compared with placentas that are centrally located (Kofinas *et al*., 1989; Ito *et al*., 1998). Such abnormal uteroplacental velocimetry waveforms are known to be associated with placental ischemia and IUGR. The maternal blood supply to the placenta derives mainly from the uterine arteries, with additional supply from the ovarian arteries. The right and left uterine arteries each have arcuate branches supplying the ipsilateral side of the uterus, some of which cross the midline to form anastomoses with the contralateral circulation. In pregnancies with unilateral placental location, uterine artery resistance is lower in the ipsilateral compared with the contralateral uterine artery; conversely, in pregnancies with centrally located placentas, the resistance is similar in the left and right uterine arteries. These observations suggest that centrally located placentas receive adequate blood flow from both uterine arteries, whereas unilateral placentas depend to a far greater extent on anastomosis between the 2 uterine arteries such that any deficiency in collateral circulation may lead to uteroplacental insufficiency and IUGR.

In this study, it is proved that second trimester placental location and preeclampsia are highly correlated. There is a six times more chance of developing preeclampsia in the lateral location of the placenta compared to the low or fundal location. So the observation seen in the study is applicable to the population also.

In a study conducted by Singh *et al*. (2016) showed that the females with laterally located placenta were found to have two times greater risk of preeclampsia. Also, the study result is in accordance with Kofinas *et al*. (1988) who concluded that in women with unilateral placenta, the incidence of preeclampsia was 2.8 fold greater than those with centrally located placenta. Similar to this study Bhalero *et al*. (2013) reported that the risk of developing pregnancy induced hypertension was five times greater for the females with a lateral located placenta as compared to those with a centrally located placenta. The results of the present study were also comparable to the study done by Gouser *et al*. (1996) that lateral implantation increases the risk of preeclampsia.

Placental location Vs low APGAR at 1 minute and NICU admissions

This study also revealed a significant correlation between placental location and low APGAR at 1 minute and NICU admissions. It revealed six fold increase in low APGAR at one minute and two fold increases in NICU admissions for unilateral placental locations compared to fundal and low placental locations.

A study conducted by Al Dahlen in 2000 (Al Dahhan, 2006) revealed the association between low lying placenta and low APGAR and other neonatal complications. However, in this study, it is the unilateral location that is associated with low APGAR and NICU admissions. This is in accordance with the study conducted by Magnan *et al* who revealed that lateral location of the placenta was associated with low APGAR but no association with IUGR.

Placental location vs low APH

Other variables in this study are APH - due to abruption or placenta praevia. It is found that the location of the placenta in the second trimester has no significant relation with placental abruption. But of the fifty cases of low lying placenta, 70% had placental migration by 34 weeks and 30% remained to have low lying placenta. Out of this, there are four cases of APH due to placental praevia. The study conducted by Bronstain *et al*. (2009) regarding the effect of low lying placenta on delivery outcomes showed that maternal hemoglobin less than 8 gm/dl was the most common morbidity associated with low lying placenta. So indirectly APH is the most common complication of low lying placenta, which is observed in four cases in this study.

Placental location vs other variables

The remaining variables are preterm birth, abnormal presentation, and PPH which showed no significant relation with the location of the placenta in the second trimester. However, a study conducted by Sarah Osmundson in April 2013 (Osmundson *et al*., 2013) revealed that women with second trimester diagnosis of a low placental location are at increased risk of PPH, which needs more studies. A study by Neethu Singh *et al*. (2016) revealed that the posteriorly located placenta can be taken as an indicator of preterm labor. In this study fundal location of placenta is not separately studied as anterior and posterior.

Strength and limitations of this study

In short, this study is an effort to establish the association of placental location with different maternal and neonatal outcomes so that it can help us to determine various maternal and fetal complications in terms of preeclampsia, IUGR, low APGAR, NICU admissions, etc. Taken with the fact that the blood supply to the uterus is not uniformly distributed, the study finding suggests that placental location may be an important determinant of placental blood flow. Additional research is necessary to confirm this observation, elucidate its mechanism, and determine whether pregnancy with the lateral placenta may benefit from more intensive monitoring like Doppler velocimetry.

Recommendations

The significance of placental location in the uterine cavity and its association with adverse feto-maternal outcomes has been studied extensively. Ultrasound has been proved to be the safest, easiest, and most accurate method for assessing placental location. Placental location can be assessed along with the anomaly scan during the second trimester. So, no additional cost is involved in assessing the location of the placenta in the second trimester. Thus, the second trimester placental location can be used as a cheap, noninvasive predictor for feto-maternal adverse outcomes like preeclampsia, IUGR, low APGAR at 1 minute and NICU Admission, especially in low resource settings. Unilateral placental location in second trimester USG can be taken as a risk factor for predicting preeclampsia and IUGR and such persons can be advised doppler velocimetry or can be referred to a higher center for further management. Low APGAR at 1 minute and NICU admissions are highly

correlated with unilateral location of placenta in the second trimester and so delivery of such patients can be conducted at higher center with NICU facility.

CONCLUSION

In this study, it can be concluded that USG during the second trimester from 18 to 24 weeks in pregnant women can be used as an easy noninvasive, cost effective tool as a predictor of adverse pregnancy outcomes. This will help in its prevention. The results of this study are in accordance with many studies done before. By identifying such high-risk patients, appropriate treatment can be initiated and the patients can be regularly followed up.

Although placenta has been systematically included in standard ultrasound reports for the past three or four decades unfortunately only few studies have been conducted with the aim of investigating the impact of placenta on pregnancy outcomes, excluding research on low lying placenta/placenta praevia.

Comprehending the role that placental location in diminishing the incidence of certain maternal and fetal adverse events, may allow us to decrease the number of pregnancy and perinatal conditions currently treated by symptomatic therapy or by an empirical approach.

As per this study maternal outcomes such as preeclampsia, APH in placenta praevia and fetal outcomes such as IUGR, low APGAR at 1 minute (i.e. <7) and NICU admissions are closely associated with the position of the placenta in second trimester. With the help of ultra sonography, a simple, noninvasive, easy to perform, cost effective diagnostic method, the location of the placenta can easily identify and thereby identify the above high-risk cases such as IUGR, low APGAR at 1 minute (i.e. <7), Preeclampsia and APH in placenta praevia. By identifying such patient appropriate treatment can be initiated and regular follow-up can be done in advance.

ACKNOWLEDGMENTS

I thank the faculties in the Department of Obstetrics& Gynaecology, Government Medical College, Kottayam, Kerala for their valuable advice in the smooth conduction of this study.

FUNDING

No funding received for this study.

AUTHORS' CONTRIBUTION

Dr Bini VS is the principal investigator of this research and Dr. Anitha K. Gopal and Dr. Bindhu K.M. are the co-investigators of this research.

CONFLICT OF INTEREST

No conflict of interest for this research.

DATA AVAILABILITY

All data were collected using a case record form after getting informed consent from the patient. All these data are available with the principal investigator.

REFERENCES

Al-Dahhan, F.H. 2006. Low-lying placenta in basra: fetomaternal outcome. The Medical Journal of Basrah University, 1–2.

Ananth, C.V., Demissie, K., Smulian, J.C. and Vintzileos, A.M. 2001. Relationship among placenta previa, fetal growth restriction, and preterm delivery: a population-based study. Obstet Gynecol 98, 299–306.

Bhalerao, A.V., Kulkarni, S. and Somalwar, S. 2013. Lateral placentation by USG: a simple predictor of preeclampsia. J South Asian Feder Obst Gynae, 5(2):68–71.

Bhide, A., Prefumo, F., Moore, J., Hollis, B. and Thilanganathan, B. 2003. Placental edge to internal os distance in the late third trimester and mode of delivery in placenta previa. BJOG 110, 860–864.

Bobrowski, R.A. and Jones, T.B. 1995. A thrombogenic uterine pack for postpartum hemorrhage. Obstet Gynecol 85, 836–837.

Booth, R.T., Wood, C., Beard, R.W., Gibson, J.R. and Pinkerton, J.H. 1962. Significance of site of placental attachment in uterus. Br Med J 5294, 1732–1734.

Bronsteen, R., Valice, R., Lee, W., Blackwell, S., Balasubramaniam, M. and Comstock, C. 2009. Effect of a low-lying placenta on delivery outcome. Ultrasound Obstet Gynecol, 33(2):204–208.

Caton, D. and Kalra, P.S. 1986. Endogenous hormones and regulation of uterine artery blood flow during pregnancy. Am J Physiol Regul Integr Comp Physiol 250, 365–369.

Chessex, P., Reichman, B. and Verellen, G. 1984. Metabolic consequences of intrauterine growth retardation in very low birthweight infants. Pediatr Res, 18(8):709–713.

Cunningham, G., Leveno, K.J. and Bloom, S.L. 2014. Williams Obstetrics. 24th edn.

Dommisse, J. 1985. Placenta praevia and intra-uterine growth retardation. S Afr Med J 67, 291–292.

Fianu, S. and Vaclavinkova, V. 1978. The site of placental implantation as a factor in the aetiology of breech presentation. Acta Obstetr Gynecol Scand 57, 371–372.

Fleischer, A., Schulman, H., Farmakides, G., Bracero, L.,Grunfeld, L., Rochelson, B. *et al*. 1986. Uterine artery Doppler velocimetry in pregnant women

with hypertension. Am J Obstet & Gynecol 154, 806–813.
Gonser, M., Tillack, N., Pfeiffer, K.H. and Mielke, G. 1996. Placental location and incidence of pre-eclampsia. Ultraschall Med 17, 236–238.
Hadley, C.B., Main, D.M. and Gabbe, S.G. 1990. Risk factors for premature rupture of the fetal membranes. Am J Perinatol, 7(4):374–379.
Holzman, C. and Paneth, N. 1994. Maternal cocaine use during pregnancy and perinatal outcomes. Epidemiol Rev 16, 315–334.
Hoogland, H.J. and de Haan, J. 1980. Ultrasonographic placental localization with respect to fetal position in utero. Eur J Obstet Gynecol Reprod Biol, 11(1):15.
Ito, Y., Shono, H., Shono, M., Muro, M., Uchiyama, A. and Sugimori, H. 1998. Resistance index of uterine artery and placental locationin intrauterine growth retardation. Acta Obstet Gynecol Scand 77, 385–390.
Kalanithi, L.E., Illuzzi, J.L., Nossov, V.B., Frisbaek, Y., Abdel-Razeq, S., Copel, J.A. *et al*. 2007. Intrauterine growth restriction and placental location. J Ultrasound Med 26, 1481–1489.
Khong, T.Y., De Wolf, F., Robertson, W.B. and Brosens, I. 1986. Inadequate maternal vascular response to placentation in pregnancies complicated by preeclampsia and by small-for-gestational age infants. Br J Obstet Gynaecol 93, 1049–1059.
Khoury, M.J., Erickson, J.D., Cordero, J.F. and McCarthy, B.J. 1988. Congenital malformations and intrauterine growth retardation: a population study. Pediatrics 82, 83–90.
Kofinas, A.D., Penry, M., Greiss, F.C. Jr., Meis, P.J. and Nelson, L.H. 1988. The effect of placental location on uterine artery flow velocity waveforms. Am J Obstet Gynecol 159, 1504–1508.
Kofinas, A.D., Penry, M., Swain, M. and Hatjis, C.G. 1989. Effect of placental laterality on uterine artery resistance and development of preeclampsia and intrauterine growth retardation. Am J Obste Gyne, 161(6 Pt 1):1536–9
Kuzma, J.W. and Sokol, R.J. 1982. Maternal drinking behavior and decreased intrauterine growth. Alcohol Clin Exp Res, 6(3):396–402.
Leiberman, J.R., Fraser, D., Kasis, A. and Mazor, M. 1991. Reduced frequency of hypertensive disorders in placenta previa. Obstet Gynecol 77, 836–839.
Magann, E.F., Doherty, D.A., Turner, K., Lanneau, G.S., Jr, Morrison, J.C. and Newnham, J.P. 2007. Second trimesterplacental location as a predictor of an adverse pregnancy outcome. J Perinatol 27, 914.
Newton, E.R., Barss, V. and Cetrulo, C.L. 1984. The epidemiology and clinical history of asymptomatic midtrimester placenta previa. Am J Obstet Gynecol 148, 743–748.
North, R.A., Ferrier, C., Long, D., Townend, K. and Kincaid-Smith, P. 1994. Uterine artery Doppler flow velocity waveforms in the second trimester for the prediction of preeclampsia and fetal growth retardation. Obstet Gynecol 83, 378–386.
Odegard, R.A., Vatten, L.J., Nilsen, S.T., Salvesen, K.A. and Austgulen, R. 2000. Preeclampsia and fetal growth. Obstet Gynecol 96, 950–955.
Osmundson, S.S., Wong, A.E. and Gerber, S.E. 2013. Second-trimester placental location and postpartum haemorrhage. J Ultrasound Med 32(4):631–6.
Proctor, J.A. and Haney, A.F. 2003. Recurrent first trimester pregnancy loss is associated with uterine septum but not with bicornuate uterus. Fertil Steril 80, 1212–1215.
Resnik, R. 2002. Intrauterine growth restriction. Obstet Gynecol 99, 490–496.
Schulman, H., Winter, D., Farmakides, G., Ducey, J., Guzman, E., Coury, A. *et al*. 1989. Pregnancy surveillance with Doppler velocimetry of uterine and umbilical arteries. Am J Obstet Gynecol 160, 192–196.
Seshadri, L. and Arjun, G. 2015. *Essentials of Obstetrics*. First edn: Wolters Kluwer.
Singh, N., Gupta, R., Pandey, K., Gupta, N., Chandanan, A. and Singh, P. 2016. To study second trimester placental location as a predictor of adverse pregnancy outcome. Int J Reproduct Cont Obst Gynecol, 5(5):1414–1417.
Sung, I.K., Vohr, B. and Oh, W. 1993. Growth and neurodevelopmental outcome of very low birth weight infants with intrauterine growth retardation: comparison with control subjects matched by birth weight and gestational age. J Pediatr 123, 618–624.
Vaillant, P., Best, M.C., Cynober, E. and Devulder, G. 1993. Pathological Doppler uterine readings when the placenta is laterally situated. J Gynecol Obstet Biol Reprod (Paris) 22, 301–307.
Vidyadhar, B., Giri, P.A., Mahajan, A.S. *et al*. 2011. Maternal and foetal outcomes in pregnancy induced hypertension – a study from rural tertiary care teaching hospital in India. Int Jour of Biomed Res 2(12):595-9
Walker JJ. 1998 Current thoughts on the pathophysiology of preeclampsia/eclampsia. Progress in obstetrics and gynecology. Edinburgh: Livingstone-Churchill; 1998:177–88.
Wolf, E.J., Mallozzi, A., Rodis, J.F., Egan, J.F., Vintzileos, A.M. and Campbell, W.A. 1991. Placenta previa is not an independent risk factor for a small for gestational age infant. Obstet Gynecol 77, 707–709.