ORIGINAL RESEARCH PAPER

Volume-7 | Issue-5 | May-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

# COULD LATERAL PLACENTA BE A PREDICTOR OF POOR PREGNANCY OUTCOMES: A PROSPECTIVE STUDY AND LITERATURE REVIEW?

Gynecology

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## ABSTRACT

**Objective:** The purpose of this study was to find out the placental laterality as predictor of pregnancy induced hypertension, baby weight and low Apgar score.

**Methods:** This was prospective study conducted from Feb. 2015 to May 2016, in department of obstetrics and gynecology, University College of medical sciences, New Delhi. 522 antenatal patients attending obstetrics OPD without high risk factors were enrolled. After enrollment 62 patients were lost to follow up. Ultrasonography for placental localization was done at 18-24 wks of pregnancy. Placenta locations were divided into lateral (either right or left) and central (anterior, posterior or fundal). Patients were followed till delivery for pregnancy outcomes such as pregnancy induced hypertension, baby weight and Apgar score.

**Results:** A total of 460 patients were analyzed, 82 (17.8%) had lateral placenta and among them 40 (48.7%) developed preeclampsia and 63 (76.8%) had small for gestational age baby ($p<0.001$). 378 (82.17%) had central placenta and among them 79 (20.9%) developed preeclampsia and 102(27%) had small for gestational age baby ($p<0.001$).

**Conclusion:** Laterally located placenta could be a predictor of poor pregnancy outcomes.

## KEYWORDS

Placental location, pregnancy induced hypertension, small for gestational age.

**Introduction:**

Only a few studies have dealt with placental implantation site and pregnancy outcomes (1-2).Preeclampsia has complex pathopysiology. Various tests have been proposed to identify women at risk of developing preeclampsia such as the cold pressor test, the isometric hand grip exercise, and the roll over test which depends on the presence of some pathophysiological changes that occur in preeclampsia. Other tests such as the measurement of urinary calcium or plasma fibronectin are based on the presence of biochemical alterations peculiar to this disease.[2] Preeclampsia occurs only in the presence of placenta [3]. The placental location by ultrasound at 18–24 weeks is noninvasive, cost effective, and has a good positive predictive value among the various predictors for preeclampsia [4]. Uterus blood supply is not uniformly distributed. The site of implantation and location of the placenta within the uterus are important determinants of placental blood flow and pregnancy outcomes like intrauterine growth restriction and preeclampsia [5]. Both uterine arteries demonstrate similar resistance in the women with centrally located placenta. When the placenta is laterally located, the uterine artery close to the placenta has lower resistance than the one opposite from it. In lateral placenta, the uteroplacental blood flow needs are to be met primarily by one of the uterine arteries with some contribution by the other uterine artery via collateral circulation [4, 6]. The degree of collateral contribution may not be the same in all women, and deficient contribution leads to the development of preeclampsia, IUGR, or both.

**Material and Methods:** This prospective study was carried out in the Department of Obstetrics and Gynecology, GTB hospital, University College of medical sciences, New Delhi from Feb. 2015 to May 2016.

**Inclusion Criteria**

All pregnant women at 18–24 weeks of gestation without any high risk factors, attending the antenatal clinic were included in this study.

**Exclusion Criteria:**

Pregnant women were excluded from the study if they were having risk factors like thyroid disoders, diabetes mellitus, chronic hypertension, renal disease, severe anemia, connective tissue disorder, positive lupus anticoagulant and anticardiolipin antibodies, RH incompatibility, twin pregnancy, low lying placenta and accrete and positive VDRL test.

Definition of lateral and central placenta: The placenta was classified as lateral (either right or left) and central (anterior, posterior, or fundal position) according to position of attachment with uterus.

Cases were subjected to history, general physical, systemic and obstetrical examination at the time of their antenatal visit and at the time of admission. The location of placenta was determined by ultrasound at 18–24 weeks in all the selected women and they were followed till delivery for the development of preeclampsia, mode of delivery, baby birth weight and APGAR. Preeclampsia was diagnosed on the basis of the American Congress of Obstetricians and Gynecologists criteria for preeclampsia and is defined as new-onset hypertension (BP is ≥140 mmHg systolic and/or ≥90 mmHg diastolic) occurring in a pregnant woman after 20 weeks gestation, with proteinuria (defined as urinary excretion of ≥0.3 g protein in 24 h). Patients who developed preeclampsia in follow up period were managed as per institutional protocol.

Baby birth weight was small for gestational age if weight less than 10$^{th}$ percentile for that gestational age. [7]

**Results:** A total of 460 patients were analyzed, out of them 82 (17.8%) had lateral placenta and 378 (82.17%) had central placenta with a mean age was 24.39 years and 24.75 years respectively. Among lateral and central placenta the mean BMI was 22.487 kg/m2 and 22.29kg/m$^2$ respectively (Table 1). Hence, age and BMI of both the groups were matched.

Among lateral placenta (n=82), 40 (48.7%) developed preeclampsia with 24(29%) had non severe and 16(19.5%) have severe preeclampsia. Among central placenta (n=378), 79 (20.9%) developed preeclampsia with 69(18.2%) have non severe and 10(2.6%) had severe preeclampsia ($p<0.001$) with 3.6 odds ratio (Table 3, Fig. 2). Preeclampsia developed 3.6 fold in lateral group of placenta. Babies birth weight were compared, in laterally situated placental group 63 (76.8%) patients developed small for gestation age (SGA) with mean birth weight was 2.259 kg (Table 1, Table 2, Fig.1). In central placenta group (n=378), mean baby birth weight was 2.73 kg (Table 1), 102(27%) patients have SGA babies ($p<0.001$) with the odd ratio 8.97 (table 2). SGA babies developed 8.97 fold among laterally situated placenta. Apgar scores both at 1 min. and 5 min. were lower in lateral placenta group.

There was no significant difference in the mode of delivery in both the groups of placenta (Table 4).

**Table 1: Profile of women**

| Placenta | N | Mean ± Std. Deviation |
|---|---|---|
| Age (yrs) Lateral | 82 | 24.39±3.65 |
| Central | 378 | 24.75 ± 3.57 |
| BMI(Kg/m2) Lateral | 82 | 22.487 ± 3.07 |
| Central | 378 | 22.297 ± 3.041 |
| Primigravida Lateral | 46 | |
| Central | 198 | |
| Multigravida Lateral | 36 | |
| Central | 180 | |
| Baby wt. Lateral | 82 | 2.259 ± .687 |
| Central | 378 | 2.73 ± .479 |

**Table2: Relationship between placental location and birth weight.**

| Placental location (n) | Birth weight | | Apgar Score | |
|---|---|---|---|---|
| | Small for gestational age | Average for gestational age | <5 at 1min. | <5 at 5min. |
| Lateral (82) | 63 (76.8%) | 19 | 23(28%) | 10(12%) |
| Central (378) | 102(27%) | 276 | 50(13%) | 6(1.5%) |

p<0.001

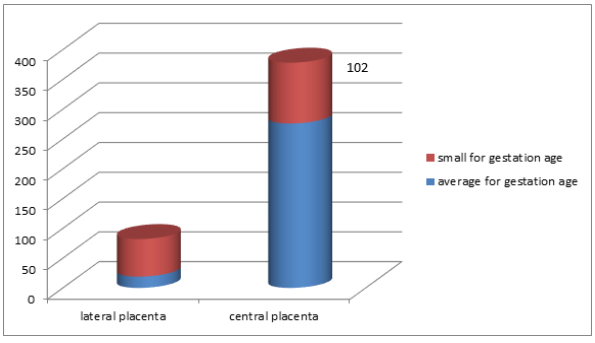


**Fig.1: Association between placental location and birth weight in both the groups**

**Table 3: Relationship between placental location and PIH**

| Placental location | No. | With PIH | Without PIH | Non severe preeclampsia | Severe preeclampsia |
|---|---|---|---|---|---|
| **Lateral** | 82(17.8%) | 40 | 42 | 24 | 16 |
| **Central** | 378 (82.17%) | 79 | 299 | 69 | 10 |
| **Total** | 460 | 119 | 341 | 93 | 26 |

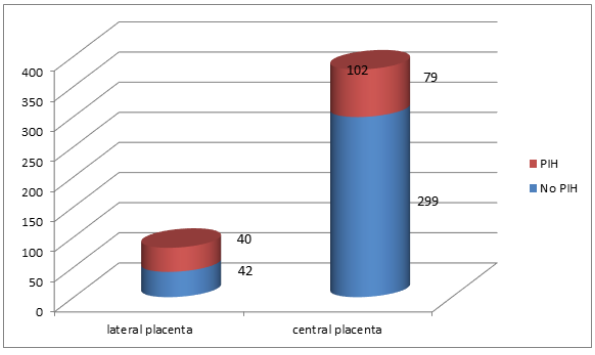


**Fig.2: Association between placental location and pregnancy induced hypertension in both the groups**

**Table 4: Location of placenta and mode of delivery.**

| Placental location (n) | Mode of delivery | |
|---|---|---|
| | Caesarean section | Normal vaginal |
| Lateral (82) | 7(8.5%) | 75 (91.4%) |
| Central (378) | 38 (10%) | 340 (90%) |
| Total (460) | 45 | 415 |

p=0.675

**Discussion:** Preeclampsia is still remains the principal cause of maternal and perinatal mortality and morbidity and there is continuous search for an ideal predictive test and preventive measure. It has been shown that in humans, both uterine arteries have a significant number of branches and that each supply the corresponding side of the uterus. Although anastomosis between the two uterine arteries exists, there is no proof that they are functional. When the placenta is laterally located, the uterine artery closer to the placenta has lower resistance than the one opposite to it. In women with centrally located placenta, both uterine arteries have similar resistance [2] and the uteroplacental blood flow needs are met by equal contribution from both uterine arteries. However, when the placenta is laterally located, in the majority of the cases, the uteroplacental blood flow needs are met primarily by one of the uterine arteries with some contribution from the other uterine artery via the collateral circulation. The degree of collateral circulation may not be the same in all the women and deficient contribution may facilitate the development of preeclampsia, IUGR, or both. Lateral placentation may thus predispose to uteroplacental insufficiency and IUGR in some women. Indeed, data from several computer simulation models of the uteroplacental circulation [5] and from studies examining the association between placental location and uterine artery Doppler velocimetry [5] support the hypothesis that the 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 trophoblast invasion leading to uteroplacental insufficiency, has been associated with lateral placentation. [5]

Results of our is in accordance with Kakkar et al (2013) prospective study [2], in which total 150 patients were taken out of which 84 had lateral placenta and 66 had central placenta. In lateral placenta 66.6% developed pregnancy induced hypertension and the odd ratio was 5.09. Lucy et al (2007) studied [5] the relationship between intrauterine growth restriction (IUGR) and laterality of placenta, with four fold increased risk (odd ratio 3.8) of IUGR in lateral placenta. Lateral placentas were significantly more common in the IUGR group than in the non-IUGR group (17.9% [12/67] versus 5.9% [12/205], respectively; P = .047). Seckin et al (2015) prospective study [8] also showed the relationship between lateral placenta and development of preeclampsia (4.5% vs. 1.6% in lateral and central placenta respectively) and fetal growth restriction. Gonser et al (1996) study (n=148) also showed odd ratio 3.1 in lateral (n=115) vs. central (n=33) group for development of preeclampsia. Fung et al (2011) also studied prospectively the location of placenta and pregnancy outcome and found the odds ratio of 2.04 for preeclampsia and 1.82 for small for gestational age (SGA) in lateral group. Results of our study were similar to above studies with odd radio of 3.6 for preeclampsia and 8.97 for SGA in lateral placentas.

But the results of Magann et al [1] reported no association between IUGR and placental location. However, that study grouped placental location into 3 broad categories (low, high lateral, and high fundal), which did not separate out central (anterior or posterior) and fundal placentas. Devarajan et al showed that relative to central/fundal location, laterally located placentas had an adjusted OR of 0.81 for SGA and 0.62 for preeclampsia/gestational hypertension and hence no relation between lateral placenta and adverse pregnancy outcomes[9].The results were contradictory to ours study.

**Table 5: Comparison with previous studies in literature.**

| Study author | Study group (n) | Type of study | Results (PIH) | Results (SGA) |
|---|---|---|---|---|
| Gonser et al (1996) | 148 (lateral=115, central=33) | Prospective | 3.1 (odds ratio) | |
| Lucy et al (2007) | 272 | Prospective | | 3.8 (odds ratio) |
| Fung et al (2011) | 16236 | Retrospective | 2.04 (odds ratio) | 1.82 (odds ratio) |
| Devarajan et al (2012) | 796 | Retrospective | 0.62 (odds ratio) | 0.81 (odds ratio) |
| Kakkar et al (2013) | 150 ( lateral =84, central= 66) | Prospective | 5.09 (odds ratio) | |
| Seckin et al (2015) | 1052 (lateral =133, central =919) | Retrospective | 4.5% vs. 1.6% | |
| Present study | 460 ( lateral =82, central = 378) | Prospective | 3.6 | 8.97 |

**Conclusion:** Location of placenta as it is done by sonography, can be a simple and non invasive test for prediction of poor antenatal outcomes in terms of pregnancy induced hypertension, small for gestational age and low APGAR scores.

**Acknowledgments:** The author declare that She has no conflicts of interest related to this manuscript. This study was not funded by any one. The study was approved from institutional ethical committee and an informed written consent was taken from the patient.

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