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RESEARCH ARTICLE

Lateral position of placenta as a predictor for development of pregnancy induced hypertension and adverse pregnancy outcomes

Anshul Rana, Rama Thakur, Rohini Rao, Neeti Aggarwal

Corresponding author: Dr Anshul Rana, Senior resident, Department of Obstetrics and Gynecology, Lady Hardinge Medical College, New Delhi, India;
Email : anshulrana10july@gmail.com

ABSTRACT

Objectives: The purpose of this study was to determine if lateral placentation and pregnancy induced hypertension are linked. To assess the incidence of adverse maternal outcomes in laterally located placenta. **Methods:** This prospective study was conducted over a period of 1 year. Singleton uncomplicated pregnancies irrespective of parity and after exclusion of high risk factors underwent ultrasound between 18 – 24 weeks gestation. All subjects were then classified in two groups according to placental locations – central placenta and lateral placenta. All pregnancies were followed and only those subjects that delivered at our institute were included. **Results:** Baseline characteristics, maternal outcomes and development of pregnancy induced hypertension were recorded. We included 300 women, 204 (68%) had centrally located placenta, and 96 (32%) had laterally located placenta. The risk of developing pregnancy induced hypertension in those with laterally located placenta was 3.8 times than those with centrally located placenta. Women with lateral placenta had significantly higher risk of abruption ($p = 0.001$), preterm delivery ($p = 0.009$), induction of labor ($p = 0.0004$) and postpartum complications ($p < 0.0001$). **Conclusions**: Our findings suggest that pregnant women with lateral placenta are at higher risk of developing pregnancy induced hypertension and adverse pregnancy outcomes.

Keywords: Placental laterality, pregnancy induced hypertension, ultrasonography, preterm delivery.

Pregnancy induced hypertension (PIH) is one of the leading causes of maternal mortality worldwide. It has been estimated that toxemia of pregnancy complicates 2 - 8% of pregnancies globally. In Africa and Asia hypertensive disorders of pregnancy contribute to 9% of deaths [1]. The cause of preeclampsia remains debatable, clinical and pathological studies suggest that the placenta is central to its pathogenesis [2]. Placentation most commonly develops in the anterior or posterior uterine walls, or the fundus (central placentation). In some pregnancies, however, the placenta implants in the lateral wall of the uterus. Evidence is scarce on the impact of lateral placentation on pregnancy outcomes [3,4]. This study was designed to seek the relation of placental laterality in ultrasound to the development of PIH and adverse pregnancy outcomes. By doing this, we will be able to screen patients at risk of developing PIH and pregnancy complications so as to keep them under close surveillance and manage them timely.

Methodology

This prospective observational study was conducted at the Department of Gynecology and Obstetrics, Indira Gandhi Medical College, Shimla which is a tertiary care teaching institute in the state of Himachal Pradesh. This study was conducted between 1st July 2019 to 30th June 2020. Written informed consent was obtained from all the participants at the time of enrollment for the study.

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Inclusion criteria -

1. Singleton uncomplicated pregnancies irrespective of parity
2. Period of gestation between 18 to 24 weeks

Exclusion criteria -

1. Chronic hypertension
2. Early onset gestational hypertension
3. Renal diseases
4. Severe anaemia
5. Connective tissue disorders
6. Diabetes mellitus (pregestational and gestational)
7. Positive lupus anticoagulant
8. Thyrotoxicosis
9. Anticardiolipin antibody positive
10. Rhesus incompatibility
11. Multiple pregnancies
12. H/O smoking, alcohol intake and drug addiction
13. Pregnancies with the congenitally malformed fetus
14. Placenta previa

All the cases were subjected to a detailed history, general physical, and systemic as well as obstetrical examination at the time of their antenatal visits. The location of placenta was determined by ultrasound at 18 - 24 weeks in all the selected women and followed subsequently for the development of complications. The placenta was classified as central when it was in anterior, posterior, or fundal position. When 70 % or more of the placental mass was to one side of the midline, it was classified as lateral (right or left) placenta. All women were followed throughout the pregnancy for the development of the signs and symptoms of PIH as per ACOG (American College of Obstetricians and Gynecologists) guidelines [5] maternal outcomes were also noted.

Statistical analysis - The presentation of the categorical variables was done in the form of number and percentage (%). On the other hand, the quantitative data with normal distribution were presented as the means ± Standard Deviation (SD) and the data with non-normal distribution as median with 25th and 75th percentiles (interquartile range). The data normality was checked by using Kolmogorov-Smirnov test. The cases in which the data was not normal, we used non parametric tests. The following statistical tests were applied for the results -

1. The association of the variables which were quantitative and not normally distributed in nature were analyzed using Mann-Whitney Test (for two groups) and independent t test was used for comparison of normally distributed data between two groups.

2. The association of the variables which were qualitative in nature was analyzed using chi-square test. If any cell had an expected value of less than 5 then fisher's exact test was used.

3. Univariate and multivariate logistic regression was used to find out significant risk factors of PIH.

The data entry was done in the Microsoft EXCEL spreadsheet and the final analysis was done with the use of Statistical Package for Social Sciences (SPSS) software version 21.0.

Results

Demographic and other baseline characteristics of our study population are depicted in table 1.

Distribution of age, gravidity, body mass index (BMI) and residence was comparable among the two groups. The distribution of placental laterality is depicted in table 2 and incidence of PIH in each group is given in table 3. Out of the total 300 women, 96(32%) had laterally located placentas and of them 51(53.13%) developed PIH, while the remaining 204(68%) had centrally located placentas and of them 35(17.16%) developed PIH (p <0.0001).

Table 1: Demographic and other baseline characteristics

Age (years)	Central placenta (n=204)	Lateral placenta (n=96)	Total	P value
≤20	30 (14.71%)	17 (17.71%)	47 (15.67%)	0.211‡
21-29	113 (55.39%)	52 (54.17%)	165 (55%)	
31-39	61 (29.90%)	25 (26.04%)	86 (28.67%)	
≥40	0 (0%)	2 (2.08%)	2 (0.67%)	
Gravidity				
Primigravida	96 (47.06%)	53 (55.21%)	149 (49.67%)	0.097‡
Gravida 2	66 (32.35%)	32 (33.33%)	98 (32.67%)	
Gravida 3	33 (16.18%)	6 (6.25%)	39 (13%)	
Gravida 4 or more	9 (4.41%)	5 (5.21%)	14 (4.67%)	
Body mass index BMI(kg/m²)				
<18.5{Underweight}	15 (7.35%)	10 (10.42%)	25 (8.33%)	0.159§
18.5 to 22.9{Normal}	135 (66.18%)	51 (53.13%)	186 (62%)	
23 to 24.9{Overweight}	35 (17.16%)	25 (26.04%)	60 (20%)	
≥25{Obese}	19 (9.31%)	10 (10.42%)	29 (9.67%)	
Residence				
Rural	144 (70.59%)	62 (64.58%)	206 (68.67%)	0.296§
Urban	60 (29.41%)	34 (35.42%)	94 (31.33%)	

Data are n, % or mean ± standard deviation (SD) unless otherwise specified, † Mann Whitney test, ‡ Fisher's exact test, § Chi square test, * Independent t test

Table 2: Distribution of placental location of study subjects

Placental laterality	Frequency	Percentage
Central placenta	204	68.00%
Lateral placenta	96	32.00%
Total	300	100.00%

Adverse pregnancy outcomes are given in table 4. The occurrence of abruption (p = 0.001), preterm delivery <37 weeks (PTD) (0.009), induction of labor (IOL) (p = 0.0004)

Table 3: Association of PIH with placental laterality

PIH	Central placenta (n=204)	Lateral placenta (n=96)	Total	P value
No	169 (82.84%)	45 (46.88%)	214 (71.33%)	<.0001§
Yes	35 (17.16%)	51 (53.13%)	86 (28.67%)	
Total	204 (100%)	96 (100%)	300 (100%)	

§ Chi square test, PIH pregnancy induced hypertension

al [7], Ambastha V et al [8] and Racher ML et al [9]. In the past several studies have been conducted on the similar grounds. However, each of them had their own strengths and limitations. The study by Yousef et al [6] was substantiated with doppler velocimetry of uterine arteries, however were

Table 4: Adverse pregnancy outcomes

Antenatal events	Central placenta (n=204)	Lateral placenta (n=96)	Total	P value
IUFD	1 (0.49%)	3 (3.13%)	4 (1.33%)	0.098‡
PPROM	4 (1.96%)	4 (4.17%)	8 (2.67%)	0.273‡
Abruption	0 (0%)	6 (6.25%)	6 (2%)	0.001‡
Gestation at the time of delivery in weeks				
28-31+6 (Very preterm)	1 (0.49%)	0 (0%)	1 (0.33%)	1‡
32-33+6(Early preterm)	2 (0.98%)	2 (2.08%)	4 (1.33%)	0.595‡
34-36+6(Late preterm)	10 (4.90%)	13 (13.54%)	23 (7.67%)	0.009§
37-38+6(Early term)	123 (60.29%)	61 (63.54%)	184(61.33%)	0.590§
39-40+6(Full term)	68 (33.33%)	20 (20.83%)	88 (29.33%)	0.027§
Onset of labor				
Spontaneous	171 (83.82%)	63 (65.63%)	234 (78%)	0.0004§
Induced	33 (16.18%)	33 (34.38%)	66 (22%)	
Mode of delivery				
Vaginal delivery	163 (79.9%)	73 (76.04%)	236(78.67%)	0.446§
LSCS	41 (20.10%)	23 (23.96%)	64 (21.33%)	
Postpartum complications				
No	192 (94.12%)	74 (77.08%)	266(88.67%)	<.0001§
Yes	12 (5.88%)	22 (22.92%)	34 (11.33%)	

‡ Fisher's exact test, † Mann Whitney test, § Chi square test, IUFD - Intrauterine fetal demise, PPROM - Preterm premature rupture of membranes, IOL - Induction of labor, IHCP - Intrahepatic cholestasis of pregnancy, PE - Preeclampsia, FGR - Fetal growth restriction, PROM - Premature rupture of membranes, LSCS - Lower segment cesarean section.

and postpartum complications ($p < 0.0001$) were significantly more in subjects with lateral placentas. Logistic regression analysis for the dependent variables of preeclampsia is given in table 5. In our study, subjects with lateral placenta had significantly higher risk of developing

done only for subjects with lateral placentation. Observations of Yousuf et al [6] and Ambastha V et al [8] included primigravidas only, they are more prone to developing PIH and this is well established fact in literature and could be a confounding factor. Study by Granfors M et al [7] had the

Table 5: Logistic regression to find out significant risk factors of PIH

PIH	Beta coefficient	Standard error	P value	Odds ratio	Odds ratio lower bound (95%)	Odds ratio upper bound (95%)
Age (years)						
≤18 years				1.000		
19-35 years	-2.228	0.858	0.009	0.108	0.020	0.580
>35 years	-2.858	1.334	0.032	0.057	0.004	0.785
Primigravida	0.324	0.334	0.332	1.382	0.719	2.658
IVF + OI conceived	3.069	0.939	0.001	21.512	3.416	135.456
Family history of hypertension	2.455	0.667	0.0001	11.642	3.152	42.997
Previous obstetric history of PIH	2.177	0.761	0.004	8.819	1.986	39.152
Placental laterality						
Central placenta				1.000		
Lateral placenta	1.341	0.320	<0.0001	3.823	2.040	7.166

PIH - Pregnancy induced hypertension, IVF - In vitro fertilization, OI - Ovulation induction

PIH as compared to those who have central placenta ($p < 0.0001$).

Discussion

Our study revealed that subjects with lateral placenta had significantly higher risk of developing PIH as compared to those who have central placenta ($p < 0.0001$). This is consistent with the findings of Yousef et al [6], Granfors M et

largest sample size collected prospectively, their finding of association of lateral placentation with preeclampsia (PE) (adjusted odds ratio 1.30, 95% confidence interval 1.03 – 1.65) was consistent with our finding. They included only primigravidas in their analysis and considered other placental locations such as fundal, anterior and posterior. It is known that there is no official classification regarding placental

location, other than placenta previa and their categorization into many groups might therefore be imprecise. Recent meta-anlysis by Racher M et al [9] combined the data of central and or fundal versus (vs.) lateral placenta, identified 14 studies with the sample size of 24968. They found lower risk of hypertension during pregnancy in central and or fundal group.

Studies with no association have also been published in the past. In contrast to our finding, Salama – Bello R et al [3] and Porto L et al [4] observed no significant difference in development of PIH between the 2 groups (p value 0.71 and 0.34 respectively). They had major limitations; both included wider gestation age for laterality scan. If placental laterality as screening method for PIH has to be established then it should be in earlier gestations. Both were retrospective study designs which also included high risk pregnancies (chronic hypertension and gestational diabetes), which could be confounding factor.

In our study preterm premature rupture of membranes (PPROM) is observed in 4/204 (1.96%) in central placenta group and 4/96 (4.17%) of lateral placenta group. Consistent with findings of Racher ML et al [9], in which no difference was identified in central and or fundal vs. all lateral group (relative risk 0.93, 95% confidence interval 0.69-2.18). Our finding on abruption (p = 0.001) is also in accordance to Racher ML et al [9], where the risk of placental abruption was significantly reduced if location site was central and or fundal vs. all lateral and central (only) vs. all lateral. Our findings on preterm delivery (table 4) are consistent with Porto L et al [4], who observed that of 1203 women, 79/1144 (6.9%) central placenta and 11/59 (18.6%) in lateral placenta group (p 0.001) delivered prematurely (<37 weeks). Our observation of more number of induction of labor (IOL) in lateral placenta subjects (33/96, 34.38%) with p value 0.0004 is in contrast to Porto L et al [4], in which 439 subjects with central placenta (38.4%) and 26 subjects with lateral placenta (59.44%) needed IOL (p 0.39). Mode of delivery (vaginal delivery vs. cesarean delivery rate) in our study was comparable in both groups (p = 0.446), in accordance to Porto L et al [4] who observed cesarean rate of 34.6% in central vs. 39% in lateral placentation subjects (p 0.49).There is a significant association of postpartum complications in subjects with lateral placenta (p <0.0001). Amongst all complications observed, 20.59% subjects had postpartum hemorrhage (PPH), of these 2/12 (17.67%) and 5/22 (22.73%) subjects belonged to central and lateral placentation group. These findings were consistent with Granfors M et al [7], who calculated adjusted odds ratio (aOR) 1.42 with 95 % confidence interval of 1.27 – 1.82 for PPH (>1000 ml).

After logistic regression, we observed that in subjects with lateral placentas, the overall risk of developing PIH is 3.28 (odds ratio). Yousuf et al [6] also observed that the overall risk of developing PE with laterally located placenta was 9.275 (odds ratio) with 95% confidence interval of 4.306–19.978. The strengths of our study are: it was a prospective study, with placental laterality scan done between the gestations of 18 weeks to 24 weeks, the time when most antenatal ultrasonography scans are done for ruling out congenital anomalies. This was done so that the subjects were not burdened to come for a separate antenatal visit for a placental laterality scan. Also, since we wanted to establish the utility of placental laterality as a predictor for preeclampsia, the predictor should be in early gestations of pregnancy to be of some value. High risk factors like chronic hypertension, diabetes mellitus (pregestational and gestational) and smoking were excluded, as they could be confounding factors. Table 5 shows that subjects who conceived by in vitro fertilization (IVF) and ovulation induction (OI), family history of hypertension and previous obstetric history of PIH had significantly high risk of developing PIH with adjusted odds ratio of 21.512, 11.642 and 8.819 respectively. The limitations of our study were that these confounding factors were not excluded, although they are well established risk factors for development of PIH. Our subjects did not have uterine artery doppler assessment as was done in the analysis by Dagklis T et al [10], who concluded that lateral placenta with unilaterally increased uterine artery pulsatility index (UtA PI) and or increased mean PI had higher risk of PE than those with bilaterally normal UtA PI.

Conclusion

In our study, we found an association of lateral placental location during second trimester ultrasound examinations and development of PIH, abruption, preterm delivery (<37 weeks), induced labor and postpartum complications. Impaired trophoblastic invasion of uterine spiral arteries is involved in etiology of PIH, and persistent high resistance in UtA should reflect the etiology process [10]. In our opinion, further studies with incorporation of histopathological examination of placenta along with detailed uterine artery Doppler could be more informative in determining why lateral placental location is risk factor for PIH and pregnancy complications.

Conflict of interest: None. **Disclaimer:** Nil.

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Anshul Rana [1], Rama Thakur [2], Rohini Rao [3], Neeti Aggarwal [4]

[1] Senior resident, Department of Obstetrics and Gynecology, Lady Hardinge Medical College, New Delhi, India; [2] Professor, Departments of Obstetrics and Gynecology, Kamla Nehru State Hospital, Indira Gandhi Medical College (IGMC), Shimla, Himachal Pradesh, India; [3] Assistant Professor, Departments of Obstetrics and Gynecology, Kamla Nehru State Hospital, Indira Gandhi Medical College (IGMC), Shimla, Himachal Pradesh, India; [4] Associate Professor, Department of Radiology, Indira Gandhi Medical College (IGMC), Shimla, Himachal Pradesh, India.