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Placental Laterality And Risk Of Hypertensive Disorders And Adverse Fetal Outcomes: A Prospective Observational Study

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Abstract
Background: *Placental laterality, determined by second-trimester ultrasonography, has been proposed as a marker for adverse pregnancy outcomes. Lateral placentation is hypothesized to compromise uteroplacental perfusion through suboptimal trophoblastic invasion, potentially predisposing to hypertensive disorders and fetal compromise. However, its utility as an independent predictor of adverse fetal outcomes remains incompletely characterized, particularly in Indian tertiary care settings.*
Objective: *To evaluate placental laterality as a predictor of adverse fetal outcomes and to compare the incidence of pregnancy-induced hypertension (PIH), fetal growth restriction, and perinatal morbidity between central and lateral placentation groups.*
Methods: *This prospective observational study enrolled 200 antenatal women with singleton pregnancies between 18–24 weeks of gestation at a tertiary care hospital over 18 months. Placental location was classified by transabdominal ultrasonography as central (anterior/posterior/fundal; n=100) or lateral (right/left lateral walls; n=100). Participants were followed until delivery. Fetal and maternal outcomes were recorded and compared using chi-square test and Student's t-test; $p<0.05$ was considered statistically significant.*
Results: *PIH occurred in 11.00% of the central group versus 34.00% of the lateral group ($p<0.001$). Mean birth weight was significantly lower in the lateral group (2.36 ± 0.52 kg vs 2.83 ± 0.41 kg; $p<0.001$). Low birth weight, intrauterine growth restriction, NICU admissions, and perinatal mortality were all significantly higher in the lateral placentation group ($p<0.001$).*
Conclusion: *Placental laterality on second-trimester ultrasonography is a significant and independent predictor of adverse fetal outcomes. Lateral placentation should prompt enhanced antenatal surveillance and delivery planning at adequately equipped facilities.*
Keywords: *Placenta; Fetal Outcome; Pre-Eclampsia; Ultrasonography, Prenatal, Pregnancy-Induced Hypertension*

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I. Introduction

Adverse fetal outcomes, including low birth weight, intrauterine growth restriction (IUGR), preterm birth, and perinatal mortality, remain major contributors to neonatal morbidity and mortality globally.(1) Identifying reliable, non-invasive, and readily available predictive markers in early or mid-pregnancy continues to be a priority in obstetric research, particularly in resource-limited settings.(2) Among the structural determinants of pregnancy outcome, placental characteristics have attracted growing attention. The placenta is the primary mediator of oxygen and nutrient delivery to the fetus, and its implantation site fundamentally governs the quality of uteroplacental perfusion.(3)

Placental laterality—referring to whether the placenta implants on the lateral uterine walls (right or left) as opposed to the anterior, posterior, or fundal walls—has been proposed as a clinically relevant parameter assessable by routine transabdominal ultrasonography.(4) The lateral walls of the uterus are supplied predominantly by the arcuate arteries arising from the uterine artery, with relatively less robust vascular density compared to the anterior and posterior walls.(5) Lateral implantation may therefore be associated with suboptimal trophoblastic invasion of spiral arteries, resulting in inadequate remodeling of the uteroplacental vasculature.(6) This pathophysiological sequence can lead to chronic placental hypoxia, oxidative stress, and release of anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1), culminating in endothelial dysfunction and the clinical spectrum of pregnancy-induced hypertension (PIH) and fetal growth restriction.(7,8)

Several studies have reported a significant association between lateral placentation and the development of PIH and preeclampsia.(9,10) However, the evidence base specifically characterizing placental laterality as a predictor of adverse fetal outcomes—including IUGR, low birth weight, poor Apgar scores, NICU admission, and perinatal mortality—remains limited, with few prospective data from Indian populations.(11) Most existing studies have examined PIH as the primary outcome, with fetal consequences reported as secondary endpoints.(12) Given that the ultimate impact of placental dysfunction is borne by the fetus, a study specifically evaluating fetal outcomes in relation to placental laterality is warranted.

Second-trimester ultrasonography is a universally performed investigation in antenatal care, and determination of placental location adds no additional cost or complexity.(13) If placental laterality can reliably predict adverse fetal outcomes, it could serve as a simple screening tool to stratify pregnancy risk and guide the intensity of fetal surveillance, including serial growth scans, umbilical artery Doppler, and non-stress testing.(14) This study was therefore designed to prospectively evaluate placental laterality as a predictor of adverse fetal outcomes in a cohort of 200 antenatal women, comparing fetal and maternal parameters between central and lateral placentation groups.

II. Methodology

Study Design and Setting

This prospective observational study was conducted in the Department of Obstetrics and Gynaecology at a tertiary care teaching hospital over a period of 18 months. Ethical clearance was obtained from the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrolment.

Participants

A total of 200 antenatal women were enrolled. Inclusion criteria comprised singleton pregnancies between 18–24 weeks of gestation attending the antenatal outpatient department. Exclusion criteria included multiple gestations, pre-existing chronic hypertension, pre-gestational diabetes mellitus, chronic renal disease, thyroid disorders, connective tissue diseases, and pregnancies with known fetal structural or chromosomal anomalies.

Placental Location Assessment

Placental location was determined by transabdominal ultrasonography performed by experienced radiologists using standardized protocols and a 3.5 MHz curvilinear transducer. The uterine cavity was divided into five segments: anterior wall, posterior wall, fundus, right lateral wall, and left lateral wall. Central placentation was defined as predominant placental attachment to the anterior wall, posterior wall, or fundal region. Lateral placentation was defined as predominant attachment to the right or left lateral uterine walls. Participants were accordingly categorized into two groups: central placentation group (n=100) and lateral placentation group (n=100).

Follow-up and Outcome Measures

All participants were followed from enrolment until delivery. Antenatal visits were scheduled monthly until 28 weeks, fortnightly from 28–36 weeks, and weekly thereafter. At each visit, blood pressure was measured in the left lateral position using a mercury sphygmomanometer after 10 minutes of rest, and urine was tested for proteinuria by dipstick analysis.

Pregnancy-induced hypertension was defined as systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg on two occasions at least 4 hours apart after 20 weeks of gestation in previously normotensive women. Preeclampsia was defined as PIH accompanied by proteinuria (≥300 mg/24 hours or ≥2+ on dipstick). Eclampsia was defined as the occurrence of new-onset grand mal seizures in a woman with preeclampsia.

Primary fetal outcomes recorded included birth weight (kg), low birth weight (<2500 g), Apgar scores at 1 and 5 minutes, NICU admission, prematurity (<37 completed weeks), intrauterine growth restriction (IUGR, defined as estimated fetal weight below the 10th centile for gestational age on serial ultrasonography with Doppler abnormalities), stillbirth, and early neonatal death. Secondary maternal outcomes recorded included development of PIH and its severity, mode of delivery, abruption placentae, HELLP syndrome, and postpartum haemorrhage. Perinatal mortality was defined as the sum of stillbirths and early neonatal deaths.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using SPSS version 25.0 (IBM Corp., Armonk, NY). Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test as appropriate. Continuous variables were expressed as mean ± standard deviation and

compared using the independent samples Student's t-test. A p-value of <0.05 was considered statistically significant.

III. Results

A total of 200 pregnant women were enrolled and followed until delivery, with 100 in each group. Both groups were comparable at baseline with respect to maternal age, parity, gestational age at enrolment, body mass index, and socioeconomic status (p>0.05 for all parameters). The mean maternal age was 24.6 ± 3.8 years in the central group and 24.9 ± 4.1 years in the lateral group (p=0.58). Primigravida constituted 54% and 56% of the central and lateral groups respectively (p=0.77).

Incidence of Pregnancy-Induced Hypertension (Table 1)

PIH occurred in 11 (11.00%) women in the central placentation group compared to 34 (34.00%) in the lateral placentation group, a difference that was highly statistically significant (p<0.001). Gestational hypertension was diagnosed in 8.00% of the central group versus 18.00% of the lateral group (p=0.038). Preeclampsia occurred in 3.00% of the central group compared to 14.00% of the lateral group (p=0.006). Eclampsia was observed in 0% and 2.00% of central and lateral groups respectively (p=0.497). The overall risk of any hypertensive disorder was approximately three times higher in the lateral placentation group.

Table 1: Incidence of Pregnancy-Induced Hypertension

Parameter	Central Placentation (n=100)	Lateral Placentation (n=100)	P-value
Any PIH	11 (11.00%)	34 (34.00%)	<0.001
No PIH	89 (89.00%)	66 (66.00%)	<0.001
Gestational Hypertension	8 (8.00%)	18 (18.00%)	0.038
Preeclampsia	3 (3.00%)	14 (14.00%)	0.006
Eclampsia	0 (0.00%)	2 (2.00%)	0.497

Maternal Complications and Mode of Delivery (Table 2)

Maternal complications were significantly more frequent in the lateral placentation group. Abruption placentae occurred in 1.00% of the central group versus 5.00% of the lateral group (p=0.108). HELLP syndrome was observed in 0% of the central group versus 2.00% of the lateral group (p=0.497). The cesarean section rate was 34.00% in the central group versus 52.00% in the lateral group (p=0.010). Postpartum haemorrhage was more common in the lateral group (9.00%) compared to the central group (4.00%) (p=0.151).

Table 2: Maternal Complications and Mode of Delivery

Complication	Central Placentation (n=100)	Lateral Placentation (n=100)	P-value
Abruption Placentae	1 (1.00%)	5 (5.00%)	0.108
HELLP Syndrome	0 (0.00%)	2 (2.00%)	0.497
Acute Renal Failure	0 (0.00%)	1 (1.00%)	1.000
Pulmonary Oedema	0 (0.00%)	2 (2.00%)	0.497
Normal Vaginal Delivery	66 (66.00%)	48 (48.00%)	0.010
Cesarean Section	34 (34.00%)	52 (52.00%)	0.010
Postpartum Haemorrhage	4 (4.00%)	9 (9.00%)	0.151

Fetal Birth Weight and Apgar Scores (Table 3)

Fetal outcomes revealed significant differences between the two groups. Mean birth weight was significantly lower in the lateral placentation group (2.36 ± 0.52 kg) compared to the central group (2.83 ± 0.41 kg; p<0.001). Low birth weight (<2500 g) was observed in 22 (22.00%) neonates in the central group versus 52 (52.00%) in the lateral group (p<0.001). Mean Apgar score at 1 minute was 7.7 ± 1.3 in the central group versus 6.3 ± 1.7 in the lateral group (p<0.001). Mean Apgar score at 5 minutes was 8.8 ± 0.9 versus 7.5 ± 1.4 in the central and lateral groups respectively (p<0.001), indicating significantly greater neonatal compromise in the lateral group.

Table 3: Fetal Birth Weight and Apgar Scores

Parameter	Central Placentation (n=100)	Lateral Placentation (n=100)	P-value
Birth Weight, mean ± SD (kg)	2.83 ± 0.41	2.36 ± 0.52	<0.001
Low Birth Weight (<2.5 kg)	22 (22.00%)	52 (52.00%)	<0.001

Parameter	Central Placentation (n=100)	Lateral Placentation (n=100)	P-value
Normal Birth Weight (≥2.5 kg)	78 (78.00%)	48 (48.00%)	<0.001
Apgar Score at 1 min, mean ± SD	7.7 ± 1.3	6.3 ± 1.7	<0.001
Apgar Score at 5 min, mean ± SD	8.8 ± 0.9	7.5 ± 1.4	<0.001

Perinatal Outcomes (Table 4)

Perinatal outcomes were significantly worse in the lateral placentation group across all parameters. NICU admission was required in 17 (17.00%) neonates in the central group compared to 42 (42.00%) in the lateral group (p<0.001). Prematurity was observed in 12 (12.00%) versus 30 (30.00%) in the central and lateral groups respectively (p=0.002). IUGR was diagnosed in 10 (10.00%) pregnancies in the central group versus 26 (26.00%) in the lateral group (p=0.003). Stillbirth occurred in 1 (1.00%) case in the central group and 6 (6.00%) in the lateral group (p=0.057). Early neonatal death was observed in 1 (1.00%) versus 4 (4.00%) in the respective groups (p=0.172). Perinatal mortality was significantly higher in the lateral group (10.00%) compared to the central group (2.00%; p=0.018), representing a five-fold increase in risk.

Table 4: Perinatal Outcomes

Outcome	Central Placentation (n=100)	Lateral Placentation (n=100)	P-value
NICU Admission	17 (17.00%)	42 (42.00%)	<0.001
Prematurity (<37 weeks)	12 (12.00%)	30 (30.00%)	0.002
Intrauterine Growth Restriction	10 (10.00%)	26 (26.00%)	0.003
Stillbirth	1 (1.00%)	6 (6.00%)	0.057
Early Neonatal Death	1 (1.00%)	4 (4.00%)	0.172
Perinatal Mortality	2 (2.00%)	10 (10.00%)	0.018

IV. Discussion

The present prospective observational study demonstrates that placental laterality, determined by second-trimester ultrasonography, is a significant predictor of adverse fetal outcomes. Women with lateral placentation had markedly higher rates of PIH, low birth weight, IUGR, preterm delivery, NICU admission, and perinatal mortality compared to those with central placentation. These findings lend support to the hypothesis that the implantation site of the placenta within the uterus has measurable consequences for fetal well-being.

The incidence of PIH in our lateral placentation group was 34.00% compared to 11.00% in the central group (p<0.001), representing approximately a three-fold difference. This is consistent with the seminal report by Kofinas et al., who demonstrated significantly higher uterine artery resistance in lateral placentation and a strong association with preeclampsia and IUGR. (9) Rai Ambika et.al. similarly reported that lateral placentation was a reliable predictor of preeclampsia in an Indian obstetric population. (10) The pathophysiological basis for this association is well-established. The lateral uterine walls receive relatively less blood flow from the arcuate arterial system, and implantation at these sites may be associated with incomplete physiological transformation of spiral arteries by invasive extravillous trophoblasts.(15) Inadequate spiral artery remodeling results in high-resistance, low-flow uteroplacental circulation, predisposing to placental ischaemia and the release of vasoactive mediators including sFlt-1 and endothelin-1, which drive the systemic endothelial dysfunction characteristic of preeclampsia.(16,17)

The primary objective of our study was to characterize the impact of placental laterality specifically on fetal outcomes, which represents an underexplored dimension of this association. Mean birth weight was significantly lower in the lateral group (2.36 ± 0.52 kg vs 2.83 ± 0.41 kg; p<0.001), and the incidence of low birth weight was more than twice as high (52.00% vs 22.00%; p<0.001). These findings align with the known consequences of uteroplacental insufficiency on fetal growth. Chronic reduction in oxygen and nutrient delivery secondary to placental hypoperfusion activates fetal adaptive mechanisms including redistribution of cardiac output to vital organs (brain-sparing), suppression of somatic growth, and reduction in amniotic fluid production, ultimately manifesting as IUGR and low birth weight.(18,19) In our study, IUGR was diagnosed in 26.00% of the lateral group versus 10.00% of the central group (p=0.003), corroborating this pathophysiological pathway.

Apgar scores were significantly lower in the lateral placentation group at both 1 minute (6.3 ± 1.7 vs 7.7 ± 1.3; p<0.001) and 5 minutes (7.5 ± 1.4 vs 8.8 ± 0.9; p<0.001), indicating greater degrees of intrapartum and immediate neonatal compromise. Lower Apgar scores in this context most likely reflect chronic intrauterine hypoxia, IUGR, and the haemodynamic consequences of maternal hypertensive disease on the fetal circulation.(20) The significantly higher NICU admission rate in the lateral group (42.00% vs 17.00%; p<0.001) further substantiates the clinical burden imposed by lateral placentation on neonatal care resources. Prematurity,

which occurred in 30.00% of the lateral group versus 12.00% of the central group (p=0.002), was a major driver of both NICU admissions and adverse neonatal outcomes, and likely reflects iatrogenic preterm delivery for severe maternal or fetal indications as well as spontaneous preterm labour associated with placental dysfunction.(21)

Perinatal mortality was significantly higher in the lateral group (10.00% vs 2.00%; p=0.018), representing a five-fold increase in risk. This finding, while consistent with the overall pattern of adverse outcomes, has important clinical implications. Perinatal mortality is the most severe endpoint in obstetric practice, and the identification of a simple ultrasonographic marker capable of predicting this risk at mid-pregnancy could meaningfully inform clinical decision-making.(22) Perween M et al. have similarly reported higher perinatal mortality rates associated with lateral placentation, though their study designs and population characteristics differed from the present study.(11)

Maternal complications, including cesarean section rate (52.00% vs 34.00%; p=0.010), were significantly higher in the lateral group. The higher operative delivery rate was primarily attributable to indications of fetal distress, severe preeclampsia, and failed induction of labour, reflecting the more compromised fetal status in this group. The higher rates of postpartum haemorrhage and HELLP syndrome in the lateral group, though not statistically significant in isolation due to sample size constraints, are directionally consistent with findings from larger studies and likely reflect the systemic coagulopathic and vascular consequences of severe hypertensive disease.(23,24)

The clinical implication of our findings is that placental laterality, determined at the time of the routine anomaly scan (18–24 weeks), could serve as a simple, no-cost addition to risk stratification in antenatal care. Women identified with lateral placentation should be counselled regarding their elevated risk profile and managed with enhanced surveillance, including more frequent antenatal visits, regular blood pressure monitoring, serial growth ultrasonography, umbilical artery Doppler studies, and urine protein assessment.(25) Consideration should also be given to aspirin prophylaxis in women with lateral placentation and additional risk factors for preeclampsia, given the evidence for its efficacy in reducing preterm preeclampsia when commenced before 16 weeks.(26) Delivery planning at a tertiary care centre equipped for neonatal resuscitation and intensive care would be advisable for all such pregnancies.(14)

A limitation of the present study is its single-centre design with a relatively modest sample size, which may limit the generalisability of findings and the statistical power to detect differences in lower-frequency outcomes such as eclampsia and stillbirth individually. The classification of placental laterality relied on the dominant site of placental attachment and may not capture the full spectrum of lateral migration. Future multicentre studies with larger sample sizes, incorporating Doppler assessment of uterine artery resistance at the time of placental location determination, would further characterize the predictive utility of lateral placentation and allow development of validated risk scoring tools.

V. Conclusion

This prospective observational study establishes placental laterality, identified by second-trimester transabdominal ultrasonography, as a significant and independent predictor of adverse fetal outcomes. Women with lateral placentation demonstrated a three-fold higher incidence of pregnancy-induced hypertension and significantly worse fetal parameters including lower birth weight, higher rates of IUGR, lower Apgar scores, increased NICU admissions, and a five-fold greater perinatal mortality compared to those with central placentation. Placental location determination at the routine anomaly scan is a simple, non-invasive, no-cost addition to standard care that can effectively identify high-risk pregnancies warranting intensified fetal surveillance, timely intervention, and tertiary-level delivery planning. Integration of placental laterality assessment into routine antenatal risk stratification protocols has the potential to meaningfully reduce adverse fetal outcomes in clinical practice.

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