Original Article

Comparison of Placental Location on Ultrasound in Preeclampsia and Normotensive Pregnancy in Third Trimester

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Abstract

Background: Hypertensive disorders in pregnancy account for 15%–20% maternal and 20%–25% perinatal mortality. There is interest in predicting preeclampsia (PE) early in pregnancy to reduce PE and its subsequent complications. There is no cheap and easily available, reliable predictor for PE. Some studies have shown that the lateral location of placenta is associated with adverse pregnancy outcomes due to PE. The lateral placenta is yet to be proven as a strong predictor of PE to initiate preventive measures. Placental localization can be easily done on routine ultrasonography during pregnancy. In the light of these observations, a prospective study was done to study any association between PE and placental location by ultrasound in third trimester. Research Question: Is there any association between placental location on ultrasound and preeclampsia in third trimester? The objective is to study association between location of placenta and preeclampsia and compare placental location in normotensive pregnancies with that in PE in third trimester. **Methods:** A prospective comparative, case–control, observational study was conducted in the Department of Obstetrics and Gynecology at North DMC Medical College and Hindu Rao Hospital, Delhi, India, from August 2019 to April 2020. The study population included 200 pregnant women with singleton pregnancy in third trimester, without any medical disorders such as diabetes mellitus, hypertension, renal disease, cardiac disease, and coagulation disorder or smoking. One hundred women had preeclampsia and 100 were normotensive controls. Ultrasound was done after filling F form as per the Government of India guidelines to rule out sex determination, and placenta was localized by ultrasound. Placenta was classified as central when it was equally distributed between the right and left sides of the uterus irrespective of anterior, posterior, or fundal position and lateral when 75% or more of the placental mass was on one side of the midline. Placental location was compared in hypertensive and normotensive pregnancies. **Results:** Out of the total 200 women, 152 (76%) had central and 48 (24%) had lateral placenta. Ninety-two percent of controls and 60% of cases had central placenta. Forty percent of cases and only 8% normotensive women had lateral placenta. Lateral placenta was five times more frequent in presence of PE as compared to normotensive controls. Out of 152 women with central placenta, 92 (60.5%) women were normotensive but with lateral placenta, only 8 (16.7%) had normal blood pressure. PE was present in 83% of women with lateral placenta and in only 39.47% with central placenta. This difference was statistically significant as $P < 0.0001$ as per Chi-square test. This reflects a significant association between lateral position of placenta and occurrence of PE. As per odds ratio (0.1304) patients without lateral placenta had 90% protection against preeclampsia. **Conclusion:** Central placenta is more common than lateral placenta. Lateral placenta is seen five times more frequently among hypertensive women and this difference is statistically significant. The absence of lateral placenta provides 90% protection against PE but the severity of PE was not affected by placental location..

Keywords: Lateral placenta, placental location, prediction, preeclampsia, sonography, ultrasound

Introduction

Hypertensive disorders remain the most common medical complication during pregnancy and affect 2%–8% of all the pregnancies globally.[1] Every year, preeclampsia (PE) leads to 50,000–60,000 maternal deaths worldwide and accounts for 8%–14% of all maternal deaths in India.[2,3] Preeclampsia is major contributor to prematurity. Neonatal mortality increases 5-fold in the presence of severe PE.[4] Preeclampsia is also a risk factor for future cardiovascular and metabolic diseases in women. Prediction, prevention, early diagnosis, and management of PE can reduce maternal and perinatal

Received: 07-04-2023 **Revised:** 13-07-2023 **Accepted:** 10-08-2023 **Available Online:** 27-04-2024

Access this article online

Quick Response Code:

Website: https://journals.lww.com/jmut

DOI: 10.4103/jmu.jmu_39_23

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How to cite this article: Aggarwal M, Mittal R, Chawla J. Comparison of placental location on ultrasound in preeclampsia and normotensive pregnancy in third trimester. J Med Ultrasound 2024;32:161-6.

morbidity and mortality and its long-term complications. Unfortunately, there has been little progress in predicting PE as compared to advances made in eliminating other serious medical conditions. The various predictors of PE include features on history, biochemical factors, and ultrasonographic parameters. Biochemical predictors of PE are not very useful because of high cost and limited availability. Hence, there is search of a predictor which is reliable, cheap, noninvasive, easy to perform, and causes least discomfort to women.[5]

Placenta is the culprit for the occurrence of preeclampsia as it is well documented that PE is cured by delivery of the placenta.[1,6] Some studies have shown that the lateral location of placenta is associated with adverse pregnancy outcomes due to PE.[7-9] However, the lateral placenta has not yet been proven as a strong predictor of PE to initiate preventive measures. In the light of these observations, this study was done to see if PE has any association with location of placenta on ultrasound in third trimester as compared to normotensive pregnancies.

Research question

Is there any association of placental location in third trimester with preeclampsia?

Aims and objectives

We aimed to study placental location in pregnancy by ultrasonography and find any association between placental location and preeclampsia by comparing location of placenta in normotensive pregnancies with that in PE in third trimester.

Materials and Methods

This prospective comparative, case–control, observational study was carried out in the Department of Obstetrics and Gynecology at NDMC Medical College and Hindu Rao Hospital, Delhi, India, from August 2019 to April 2020. Due clearance from the ethical and scientific committee of our hospital was obtained for this study (Registration Number: ECR/979/Inst/DL/2017; Approval number: Dean/North/DMC/2019/3126; Approval date: 11/09/2019). Additional informed consent was obtained from all the patients for which identifying information is not included in this article. We included women in third trimester with singleton pregnancy attending antenatal clinic or admitted to the hospital who met inclusion criteria. There were 100 cases and 100 controls. Cases had PE and controls were normotensive women selected randomly. Women with multiple pregnancy and those with a history of chronic hypertension, diabetes mellitus, renal disease, and coagulation disorder were excluded from the study.

A detailed history was taken. General and systemic examination was done. Blood pressure (BP) of pregnant women was measured by a conventional mercury sphygmomanometer, which has been the gold standard for measurement of BP as per NICE guidelines.[10] The BP was measured in the sitting position with the arm at the level of heart, the cuff was long enough to encircle the arm and wide enough to cover at least two-thirds of upper arm. BP cuff was firmly applied and inflated

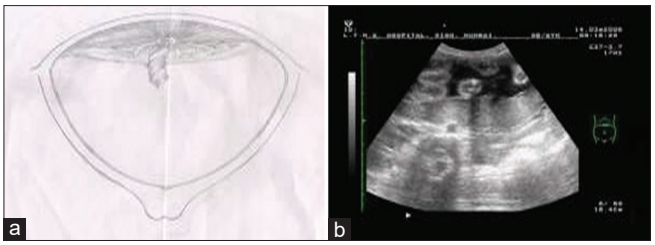

Figure 1: Central placenta-(a) schematic diagram, (b) ultrasound image

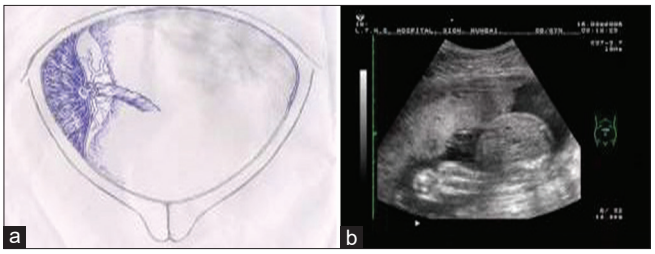

Figure 2: Lateral placenta-(a) schematic diagram, (b) ultrasound image

till 20–30 mmHg above the disappearance of radial pulse; cuff was deflated at a rate of 2 mmHg per second. Diastolic BP was indicated by Korotkoff phase V or the disappearance of Korotkoff sounds. PE was diagnosed and classified as per the ACOG 2018 guidelines:[11]

- Mild PE: Systolic BP ≥140 mmHg and diastolic BP ≥90 mmHg
- Severe PE: Systolic BP ≥160 mmHg and diastolic BP ≥110 mmHg.

Consent was taken. PNDT form, i.e., form F was filled to rule out any sex determination for all the pregnant patients as per guidelines of the government of India. Ultrasonography was performed in supine position using MINDRAY ultrasound scanner using a 3.5 MHz curvilinear probe. Placental localization was done by ultrasonography and classified as central or lateral:

- Central – When placenta was equally distributed between the right and left sides of uterus. The central placenta could be anterior, posterior, or fundal [Figure 1]
- Lateral – When 70% or more of placenta was on one side of midline. It could be right or left [Figure 2].[12]

Placental location was compared in hypertensive and normotensive pregnancies. The data were entered into MS EXCEL spreadsheet and analysis was done using GraphPad instat 3 software. Variables were correlated using the Chi-square test. Pearson coefficient was used to assess the association of various parameters with each other. A $P < 0.05$ was considered statistically significant.

Results

Most of the women were between 20 and 30 years (20–35 years). The basic demographic profile of our patients is given in Table 1. Age, parity, and gestational age were comparable among cases and controls.

The distribution of placental location is depicted in Table 2 and Figure 3. The central placenta was overall more common than lateral placenta. The lateral placenta was present in 40% of cases with PE and only 8% of controls. This difference is statistically significant as $P < 0.05$. Hence, the central placenta was much more frequent among normotensive women as compared to PE and lateral placenta was seen five times more frequently among hypertensive women.

Out of 152 patients with central placenta, 60.5% (n = 92) women had normal BP but with lateral placenta, only 16.7% (n = 8) women were normotensive. In other words, preeclampsia was present in 39.47% of women with central placenta and 83% of women with lateral placenta [Table 2 and Figure 4]. This difference was statistically highly significant as the [$P < 0.0001$, Table 2]. In the present study 50% women had preeclampsia. However, actually only 8% pregnancies have PE. Equating the current results to make PE 8%, will bring down incidence of PE to 5% in central placenta and 30% in lateral placenta. Hence, PE is almost six times more frequent in the presence of lateral placenta as compared to central placenta. This reflects a significant association between lateral position of placenta and occurrence of preeclampsia.

The risk of PE was 9.87 in the presence of lateral placenta. Odds ratio was 0.1304 with 95% confidence interval (CI) (0.05710–0.2979) and Correlation Coefficient (r) was 0.8575. This meant that patients who did not have lateral placenta had 90% protection against preeclampsia. All the patients with left anterolateral placenta had PE.

As shown in Table 3 and Figure 4, out of 100 cases with PE, 67 had mild and 33 patients had severe PE. Out of 60 hypertensive patients with central placenta, 71.66% (n = 43) had mild preeclampsia and 28.33% (n = 17) had severe preeclampsia, this difference was statistically significant P = 0.0091. On the other hand, out of 40 cases of PE with lateral placenta, PE was mild in 60% (n = 24) and severe in 40% (n = 16) but this difference was statistically not significant as [P = 0.4003, Table 3 and Figure 4]. Mild PE was almost 1.8 times more frequent with central placenta than lateral though this difference was statistically not significant.

In 67 patients with mild PE, 64.2% (n = 43) had central placenta and 35.8% (n = 24) had lateral placenta. In patients with severe PE placenta was almost equally distributed between central and lateral groups. Hence, the severity of PE was not affected by placental location.

Table 1: Distribution of patients according to demographic parameters

	Cases	Controls
Age (years), mean±SD	24.61±2.63	25.27±2.74
Gravida		
Primigravida	37	21
Multigravida	63	79
Gestational age (weeks), mean±SD	35.16±2.701	36.03±2.96

SD: Standard deviation

Table 2: Comparison of placental location in hypertensive cases and normotensive controls

Location of placenta	Cases	Controls	Total, *n* (%)	*P*
Central placenta	60	92	152 (76)	0.0429
Lateral placenta	40	8	48 (24)	0.0002
Total	100	100	200 (100)	

Table 3: Distribution of severity of pregnancy-induced hypertension between different placental groups

Severity of HT based on DBP	Central placenta	Lateral placenta	Total
Mild PE	43 (71.66)	24 (60)	67
Severe PE	17 (28.33)	16 (40)	33
Total	60 (100)	40 (100)	100
P	0.0091	0.4003	

DBP: Diastolic blood pressure, HT: Hypertension, PE: Preeclampsia

Discussion

PE occurs only in the presence of placenta.[13] In normal placenta, cytotrophoblasts invade tunica media of maternal spiral arteries and replace its endothelium, a process called pseudo vascularization. As a result, these vessels undergo transformation from small muscular arterioles to large capacitance, low-resistance vessels. This allows increased blood flow to maternal fetal interface. In PE, this invasion of decidual arterioles is incomplete and the placentation is

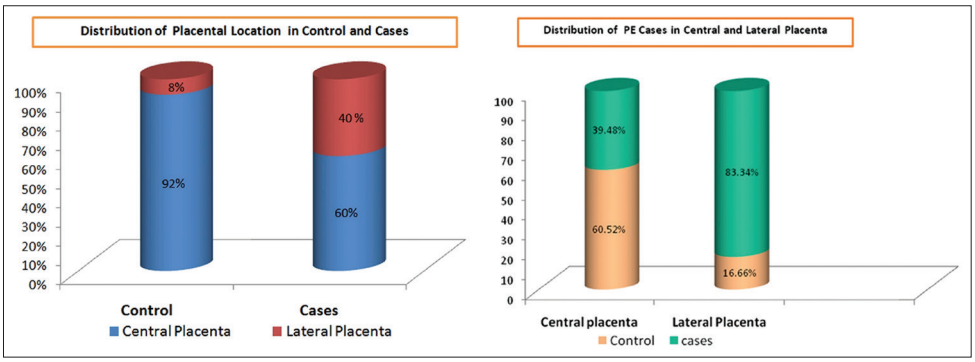

Figure 3: Comparison of placental location in hypertensive cases and normotensive controls

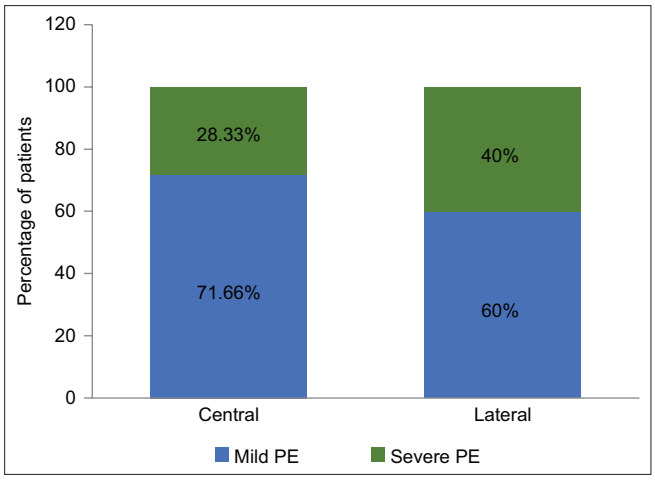

Figure 4: Distribution of severity of pregnancy-induced hypertension between different placental groups

shallow. This leads to faulty remodeling of uteroplacental arteries, inadequate perfusion, release of cytokines, leukotriene, immunomodulators, free radicals, and reactive oxygen species into the maternal circulation. This results in alteration between angiogenic and anti-angiogenic factors, vasodilators, and vasoconstrictors and causes PE and intra-uterine growth restriction.[14-16] Hence, placenta and PE are closely related to each other.

The uterus is supplied by uterine artery which is a branch of the hypogastric artery. Each uterine artery supplies corresponding side of uterus through several branches. Each uterine artery anastomoses with branches from opposite side.[17] The distribution of uterine blood flow is in-homogeneous and hence placental location is important.[18,19] With central placenta, resistance is similar in both uterine arteries and both uterine arteries contribute to uteroplacental flow. In the presence of lateral placenta, the uterine artery closer to placenta has lower resistance and it primarily meets the uteroplacental blood flow with some contribution from other uterine arteries through collateral circulation. All women do not have the same amount of collateral circulation and trophoblastic invasion may be reduced in lateral placenta. There are reports of higher resistance in the presence of lateral placenta.[20] This deficient uteroplacental flow may contribute to PE and fetal growth restriction or both in the presence of lateral placenta.[21] The risk of PE is higher in pregnancies with increased resistance in uterine vessels in the presence of lateral placenta.[22-24]

PE incurs a huge impact on health system because of high morbidity and mortality in mother, newborn, and long-term sequelae of prematurity and growth restriction besides increased cost. Maternal and fetal complications due to PE can be reduced by predicting PE early in pregnancy. Various predictors of PE include history, biochemical markers, ultrasound parameters, and others such as cold pressor test, the isometric handgrip exercise, and rollover test.[10,11,25] Among ultrasonographic parameters, uterine artery pulsatility index, resistivity index have been used to predict PE.[22]

None of these predictive tests are in wide use because of several limitations and hence, search for an ideal predictive test for PE is still on. Placenta is routinely evaluated during second and or third-trimester ultrasound for its location and morphology. Placental location is done to exclude low-lying placenta and is not associated with fetomaternal outcome.[26] Recently, there has been increased interest in studying placental location as a predictor of PE. The lateral location of placenta has been associated with increased incidence of PE by several authors.[7,19] Ultrasound location of placenta at 18–24 weeks can be a noninvasive, reliable, cost-effective predictive screening test for PE.

Kofinas *et al*. evaluated correlation between placental location on ultrasound and subsequent development of preeclampsia in 300 women. Placenta was lateral in 54% and central in 46% women. Patients who remained normotensive subsequently had equal distribution of placenta as central and lateral. Thirty-four patients developed PE subsequently and 74% of them had unilateral placenta and only 26% had central placenta. The incidence of PE was 2.8-fold higher in patients with unilateral placenta as compared to those with central placenta ($P < 0.03$). They concluded that unilaterally placed placenta may predispose to subsequent development of PE by affecting resistance in uterine arteries and thereby uterine artery circulation.[19]

In 2011, Fung *et al*. published a retrospective study done over 7 years from 2000 to 2007 involving 16,236 patients. Out of these, 609 women had low-lying placenta. Out of remaining 15,627 women, they classified placenta as fundal, central, and lateral. 1.77% and 3.34% of women with central and lateral placenta developed PE. They observed a 2.2-fold and 2-fold higher risk of PE in the presence of fundal and lateral placenta as compared to central placenta, respectively.[27]

Seckin *et al*. did a retrospective study on 1057 patients. They found that placenta was central in 87.4% ($n = 919$) and lateral in 12.6% ($n = 133$) patients. PE was found to be significantly higher in the lateral placental location group (4.5% vs. 1.6%; $P = 0.027$). They suggested that pregnant women with lateral placenta should be closely monitored for the development of PE.[7]

On the contrary, in the same year, Gizzo *et al*. did not find any case of gestational hypertension in women with lateral placenta in a prospective cohort observational study on 1056 women. Placenta was central in 93.46% and lateral in 6.53% of cases.[26]

Yousuf *et al*. in 2016, did a prospective cohort observational study on 201 singleton pregnant women with no high-risk factor for PE. Placenta was localized between 18 and 22 weeks of pregnancy during a routine ultrasound. Doppler was done if placenta was lateral. These pregnancies were followed for the development of PE till 40 weeks of gestation. Placenta was central in 130 (75.5%) and lateral in 71 (24.5%) cases. PE occurred in 52% (37/71) and 10.8% (14/130) women with lateral and central placenta, respectively. This difference was statistically significant, $P < 0.001$. The odds ratio for

Table 4: Comparison of placental location overall and in preeclampsia in various studies

Placental location	Present study	Kofinas *et al*. (1989)[19]	Fung *et al*. (2011)[27]	Seckin *et al*. (2015)[7]	Gizzo *et al*. (2015)[26]	Yousuf *et al*. (2016)[28]	Salama bello *et al*. (2019)[29]
Total patients	200	300	15,627	1057	1056	201	464
Central, *n* (%)	152 (76)	138 (46)	14,999 (95.98)	919 (87.4)	987 (93.46)	130 (75.5)	411 (88.57)
Lateral, *n* (%)	48 (24)	162 (54)	628 (4.01)	133 (12.6)	69 (6.53)	71 (24.5)	53 (11.42)
Total cases of PE, *n* (%)	100 (50)	34 (11.33)	286 (1.83)	21 (2.28)	37 (3.5)	51 (25.37)	97 (20.9)
Percentage of PE in central placenta, *n* (%)	60 (39.5)	9 (6.52)	265 (1.77)	15 (1.2)	37 (3.74)	14 (10.8)	87 (21.17)
Percentage of PE in lateral placenta, *n* (%)	40 (83.3)	25 (15.43)	21 (3.34)	6 (4.5)	0	37 (52)	10 (19)
Percentage of central placenta in PE	60	26.47	92.65	71.4	100	27.4	89.6
Percentage of lateral placenta in PE	40	73.52	7.34	28.57	0	72.5	10.4

PE: Preeclampsia

developing PE with lateral placenta was 9.27 and 95% CI was (4.30–19.98). The risk for PE with lateral placenta was significantly higher (92%) when there were associated Doppler abnormalities as compared to 6% with normal Doppler.[28]

In 2019, Salama-Bello *et al*. evaluated placental location retrospectively in 464 patients. They found that the placenta was central in 411 (88.57%) patients and lateral in only 53 (11.42%) women. They did not find any difference in PE in central or laterally situated placenta (19% vs. 21%, respectively).[29]

In the present study, the overall central placenta was more common and preeclampsia was present in 83% of women with lateral placenta and 39.47% of women with central placenta. This difference was statistically highly significant as the $P < 0.0001$. This reflects a significant association between lateral position of placenta and occurrence of preeclampsia. This was in accordance with other studies by Seckin *et al*., Fung *et al*., Yousuf *et al*., Salama-Bello *et al*.[7,27-29] However on the contrary, various other studies found lateral placenta to be more common in around 60-70% of patients.[19,26] On the other hand, Kofinas et al. 1989, Kakkar et al. found central and lateral placenta almost equally distributed.[19,21] As per the odds ratio of 0.130, patients who did not have lateral placenta had 90% protection against preeclampsia.

Table 4 shows a comparison of our study with other studies. In the present study, PE was far more common in lateral placenta as compared to central placenta which was in accordance with the largest study by Fung *et al*. and several other studies.[7,27-29] However, our results were contrary to the study by Gizzo *et al*. and Salamella-Bello *et al*. as they did not find any difference in the incidence of PE according to placental location.[26,29]

In the present study, the central placenta was more common (60%) than lateral placenta (40%). This difference is statistically significant ($P < 0.001$).

Most of the studies done till date have found a higher incidence of PE in the presence of lateral placenta and our study was in accordance with them. In the present study, 40% of patients with central placenta and 83% with lateral placenta have PE and this difference is statistically significant with $P < 0.0001$.

As the placenta is routinely localized during ultrasound done in mid-pregnancy to rule out congenital malformation, the lateral placenta can be used as a common, easily available, and cost-effective predictor of PE and reduce its associated morbidity and mortality. Hence, placental location during mid-pregnancy has additional importance in predicting PE besides ruling out low-lying placenta.

Conclusion

From the above study, we conclude that central placenta is more common than lateral placenta (76% vs. 24%). Lateral placenta is seen five times more frequently in preeclampsia as compared to normotensive controls. PE is almost six times more frequent in the presence of lateral placenta as compared to central placenta. The absence of lateral placenta provides 90% protection against PE but the severity of PE was not affected by placental location.

Study limitations

Small sample size.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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