

The Relation between development of Pregnancy Induced Hypertension and location of placenta among the pregnant women in Bastar Region

Rajeshree A. Nandanwar^{1,*}, Abhilasha M. Wahane², Narendra S. Dange³

¹Assistant Professor, Department of Anatomy, GMC, Jagdalpur

²Associate Professor, Department of Anatomy, GMC, Nagpur

³Associate Professor, Department of Biochemistry, GMC, Jagdalpur

***Corresponding Author:**

E-mail: rajeshreedng@yahoo.com

ABSTRACT:

Objective: To find out the relation between developments of Pregnancy-Induced Hypertension (PIH) and location of placenta.

Material and Method: 900 pregnant women were selected from antenatal clinic with gestational age of 18 to 24 weeks without any complication. Ultrasonic examination was done to find out site of placenta. Development of sign and symptoms of PIH were followed.

Result: Among 900 women, 365 develop PIH out of 549 with laterally located placenta and 128 women develop PIH out of 351 with centrally located placenta. So the risk for development of PIH with laterally located placenta was 3.45 (odds ratio) and 95% confidence interval (2.62-4.57). The difference was statistically significant, p value ($p < 0.0001$) by chi-square test.

Conclusion: There is significant relationship between laterally located placenta and the development of PIH. There was three times increase risk for development of PIH in females with laterally located placenta. To achieve favourable outcome and reduce the maternal and perinatal morbidity and mortality associated with PIH, these subjects may require special obstetric care and management.

Keywords: Placental laterality, PIH, Ultrasound, Central placenta.

Access this article online	
Quick Response Code:	Website: www.innovativepublication.com
	DOI: 10.5958/2394-2126.2015.00014.6

INTRODUCTION

Many anatomists, embryologists and obstetricians have curiosity about placenta due to its role in the intrauterine development of life. The ancient Egyptians believed that the soul enter the child at birth through the placenta. The term 'Placenta' was derived from Greek word 'Plakuos' meaning 'Flat cake' and was coined for the first time by 'Realdus Columbus' in 1559.

As per the meaning, placenta is circular, discoid organ, the growth of which is directly influenced by the maternal health conditions and accordingly it affects the intra-uterine status of the foetus. It is a structure where maternal and foetal tissues come in direct contact without rejection, suggesting immunological acceptance of foetal graft by the mother.^[7] Placenta is a potent endocrine, immunologic and metabolic organ besides being responsible for nutrition, respiration and excretion for the foetus.

The placenta begins its function of providing nourishment to foetus very early in development. Placenta and umbilical cord are essentially a foetal

organs functioning to support growth of foetus and interacts with two individuals - mother and foetus. The placenta and umbilical cord remains connected to the uterus till the baby is out of womb.

Whatever changes are taking place in placenta, it reflects intrauterine status of foetus. So in adverse foetal outcome, it gives valuable clues. A complete picture of placental function emerges through team work of an obstetrician, a paediatrician, a pathologist, an anatomist, a biochemist and an endocrinologist. Now a days, PIH is one of the commonest causes of maternal and foetal morbidity and mortality,^[10] other than haemorrhage and infection.^[28] These hypertensive disorders are classified into three clinical types:-

1. Pre-eclampsia
2. Eclampsia
3. Gestational Hypertension

Pre-eclampsia is a syndrome complex characterized by increase of blood pressure upto 140/90 mm of Hg recorded on two occasions 6 hours apart with proteinuria (0.3 gms or more protein in 24 hour collected sample with 1+ or greater on urine dipstick test) after 20th weeks of gestation involving multiple organ system. It is also called as toxemia. It is more common in primigravidae (10%) than multigravidae (5%).^[10] Following delivery these signs disappear fairly quickly.

Eclampsia is considered as the most dangerous form of these disorders. It is defined as

the pre-eclampsia complicated with convulsions and/or coma. The term 'eclampsia' is derived from Greek word meaning 'like a flash of lightning' as it usually occurs quite abruptly without any warning manifestations.^[10]

Gestational hypertension is defined as sustained rise of blood pressure to 140/90 mm of Hg or more on at least two occasions, four or more hours apart beyond 20th week of pregnancy or during the first 24 hours after delivery in a previously normotensive woman.^[10]

In PIH, resistance to flow in utero-placental circulation is increased, affecting the growth of placenta in terms of weight, thickness, surface area, volume and location. These placental abnormalities ultimately result in reduction of foetal weight.^[21] So its examination gives a clear idea of what had happened with it, when it was in the mother womb and what is going to happen with the foetus in the future.^[28] Several studies were done to find out the significance of placental location in the uterine cavity. Placental location has been found to correlate with foetal position and presentation,^[14,16] length of gestation,^[13] course of labour,^[9] presence of preeclampsia^[3,20] and pregnancy outcome.^[5] Several methods have been used to document placental location, including manual exploration of the uterus, soft tissue x-ray films, and isotopic placenta-graphy.^[8,16,26,27,30] In the past two decades ultrasonography has proved to be the safest, easiest, and most accurate method for assessing placental location.^[1,14,17]

The relationship between location of placenta and the development of PIH has been examined previously with controversial results.^[3,20] In the two previous reports the placental location was examined with regard to its proximity to the uterine fundus. The two groups used different methods. There has been no study to examine the clinical significance of placental laterality and its association with the development of PIH and intrauterine growth retardation (IUGR). There is significant association between location of placenta and uterine artery resistance.^[18] It is possible that placental location may influence uterine artery blood flow distribution and predispose the pregnancy to such adverse outcome as preeclampsia, premature birth, and IUGR.

The aim of this study was to examine the association of placental laterality and the development of preeclampsia.

MATERIAL AND METHOD

The study was carried out in the Department of Obstetrics and Gynaecology, GMC, Jagdalpur from Jan 2012 to 2015.

Inclusion criteria -All pregnant women attending the antenatal clinic at 18 to 24 weeks of gestation without any high risk factors.

Exclusion criteria -Pregnant women having chronic hypertension or essential hypertension, severe anaemia, twin pregnancy, RH incompatibility, diabetes mellitus, thyrotoxicosis, renal disease, connective tissue disorder or positive VDRL test.

Ethical clearance was taken as per the guidelines of the institute. Written consent was taken. Detail history was obtained; general, physical and systemic as well as obstetrical examination was done at the time of their antenatal visit. Ultrasonography was done to determine location of placenta at 18 to 24 weeks in all selected women and followed subsequently for the development of PIH.

The placenta was classified as central when it was equally distributed between the right and left side of the uterus irrespective of anterior, posterior or fundal position. When 75% or more of the placental mass was to one side of the midline, it was classified as unilateral right or left placenta. All women were followed throughout the pregnancy for the development of the sign and symptoms of PIH. It was diagnosed on the basis of the American Congress of Obstetricians and Gynaecologists criteria for preeclampsia. The patients were treated according to the severity of the disease.

Statistical Analysis: The statistical analysis was done by using Chi-Square Test. $P < 0.05$ was considered as statistically significant. Data was analyzed by using window Microsoft and statistical software Graph Pad Prism 5.01.

RESULTS

Data of 900 patients was analysed. Incidence of preeclampsia was found to increase with increasing age. In our study 42% patients over 30 years developed preeclampsia (Figure 1). Out of total 900 women, 73% (657) were in age group 21 to 30 years (Table 1). On ultrasonographic examination done at 18 to 24 weeks of gestation, 549 (61%) women had laterally placed placenta and 351 (39%) had centrally located placenta (Table 2). Out of 549 women with laterally placed placenta, 365 (66.4%) developed PIH, while 128 women (36.4%) out of remaining 351 with centrally placed placenta developed PIH. So the risk for development of PIH with laterally located placenta was three times more (odds ratio-3.45) than centrally located placenta and 95% confidence interval (CI) 2.62-4.57. The difference was statistically significant, p value ($p < 0.0001$) by chi-square test.

Out of the total 900 cases, 244 developed mild PIH (Diastolic Blood pressure DBP 90 to 99 mmHg). Out of these 244 cases, 100 had centrally located placenta and 144 had laterally located placenta (Table 3, 4). 161 cases developed moderate

PIH (D.B.P.100 to 109mmHg). Out of these 161 cases, 42 were with centrally located placenta, while 119 women were with laterally located placenta (Table 3, 4). 88 women developed severe PIH (D.B.P.>110mmHg) and out of these 8 had centrally located placenta and 80 had laterally located placenta.

Table 1: Distribution of cases according to age

Age (yrs)	n	%
< 20	45	5 %
21-25	414	46 %
26-30	243	27 %
31-35	198	22%

Table 2: Relationship between placental location and PIH

Placental location	Outcome		Odds ratio	95% CI
	PIH	No PIH		
Lateral n=549	365 (66.4%)	184 (33.5%)	3.45	(2.61-4.57)
Central n=351	128 (36.4%)	223 (63.5%)		

P<0.0001

Table 3: Distribution of cases according to the severity of hypertension

Severity of Hypertension based on DBP(mm of Hg)	Number of cases n-493
Mild (90-99)	244
Moderate (100-109)	161
Severe (>110)	88

Table 4: Distribution of severity of PIH between different placental groups

Severity of Hypertension based on DBP(mm of Hg)	Centrally located Placenta	Laterally located Placenta
Mild (90-99)	100	144
Moderate (100-109)	42	119
Severe (>110)	8	80

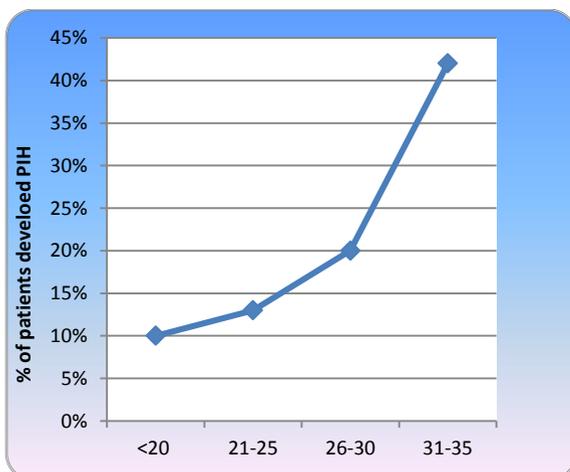


Fig. 1: Percentage of patients developing PIH in accordance with age

DISCUSSION

Preeclampsia involves multiple organ system and still remains the principal cause of maternal and perinatal mortality and morbidity^[12] In humans, uterine arteries are divided into significant number of branches supplying corresponding side of the uterus. There is no proof that anastomosis between two uterine arteries are functional. In laterally located placenta, the uterine artery closer to placenta has lower resistance than the one opposite from it. But in centrally located placenta, both uterine arteries has similar resistance^[4,11,25] In centrally located placenta, the uteroplacental circulation is equal from both uterine arteries. But in laterally located placenta, the uteroplacental circulation is mainly through one of the uterine arteries. Other uterine artery has less contribution through collateral circulation. This collateral circulation may not be the same in all patients and deficient contribution may facilitate the development of preeclampsia.^[29]

The existence of major vascular anastomosis in some patients may explain the normal uterine flow velocity waveform and absence of preeclampsia even if placenta is unilateral. In normal gravid uterus, the spiral arterioles supplying placental bed undergo trophoblast induced conversion to uteroplacental arterioles. The significance of normal placentation for this cytotrophoblastic invasion is high and the cytotrophoblast fail to adopt a vascular adhesion phenotype in preeclampsia.^[6] In preeclampsia, conversion of the spiral arterioles is incomplete.^[24] It involves only the sub placental venules. In cases with laterally located placenta, there is no functional anastomosis between right and left uterine arteries, so that systolic / diastolic ratio of ipsilateral uterine artery is more than the contralateral. So uteroplacental circulation is through one side uterine artery causing reduced trophoblastic invasion in laterally located placenta.^[23]

In this study, out of 900 women, 549 (55%) females had laterally located placenta and 351 (45%) had centrally located placenta. Out of 549 females with laterally located placenta 365 (66.4%) developed PIH as compared to 351 females with centrally located placenta where 128 (36.4%) developed PIH. So the risk of developing PIH was three times greater for females with laterally located placenta. So the risk for development of PIH with laterally located placenta was 3.45 (odds ratio) and 95% confidence interval (2.62-4.57). The difference was found to be highly significant statistically (p<0.0001).

In the previous study done by Kofinas et al, he found the incidence of PIH in laterally located placenta was 2.8 fold greater than those with centrally located placenta^[19] and our result is in accordance with this. The study of Muralidhar et al^[22] included 426 unselected singleton pregnant women, out of which 324 had centrally located placenta and

102 had unilateral placenta. A total of 71 women developed preeclampsia of which 52 (74%) had unilaterally located placenta. The relationship was found to be statistically significant $p < 0.0001$. The result of present study was also comparable to the study done by Kalanithi et al.^[15] The study showed that development of PIH and IUGR pregnancies were nearly fourfold more in lateral placentation.

Booth et al.^[3] concluded that there is a significant association between fundal placenta and; they explained it by Bieniarz's theory. Bieniarz^[2] theorized that when location of placenta is high in the uterine cavity, drainage of uteroplacental circulation might cause a pathologic redistribution of blood in the renal and visceral circulation, resulting in PIH. In contrast, Little and Friedman^[20] failed to find any association between highly implanted placenta and preeclampsia.

CONCLUSION

The pathophysiologic characteristics of preeclampsia are complex and the cause remains unknown. The study demonstrates a significant relationship between placental laterality and the presence of preeclampsia. It was speculated that insufficient collateral blood supply in patients with unilateral placentation may facilitate the development of preeclampsia. Females with laterality located placenta had a three times greater risk of developing PIH, so these pregnancies may require careful obstetric management to achieve a more favourable outcome and decrease the maternal and perinatal morbidity and mortality associated with preeclampsia.

REFERENCES

1. Badria L, Young GB. Correlation of ultrasonic and soft tissue x-ray placentography in 300 cases. *J Clin Ultrasound* 1976;4:403
2. Bieniarz J. The patho-mechanism of late pregnancy toxemia and obstetrical haemorrhages. II. Placental site and venous drainage of the pregnant uterus. *AM J OBSTET GYNECOL* 1959; 78: 385)
3. Booth RT, Wood C, Beard RW, Gibson JRM, Pinkerton JHM. Significance of site of placental attachment in uterus. *Br Med J* 1962;1:1732
4. Campbell S, Bewbey S, Cohen-overbeek T. Investigation of the uteroplacental circulation by Doppler ultrasound. *Semin Perinatol.* 1987; 11:362.
5. Chapman MG, Furness ET, Jones WR, Sheat JH. Significance of the ultrasound location of placental site in early pregnancy. *Br J ObstetGynaecol* 1979;86:846.
6. Das Gupta S. Pathophysiology and prevention of pregnancy induced hypertension. New Delhi, JP Publications. Recent Adv. In Obstet and Gynaecol. 1:14-31.
7. Datta AK. Essentials of human embryology. 5th ed. Kolkata; Current Books International. 2007. p. 57-69.
8. Davison TW, Ewing KL, Sayat N, MullaNP, Ferguson JL. Liquid crystal thermographic placental location. *ObstetGynecol* 1973;42:574.
9. Davydov SN, Orlov VM, Samorodinova LA, Khrustalkov SV. Location of placenta and clinical course of labour. *ActaChir Hung* 1987;28:3.
10. Dutta DC. Textbook of obstetrics including perinatology and contraception. 6th Ed. New Central Book Agency (P) Ltd.; 2009. p. 221-42.
11. Flescher A, Schulman H, Farmakides G, et al. Uterine artery Doppler velocimetry in pregnant women with hypertension. *American J Obstet Gynecol.* 1986; 154:806-13.
12. Haris D. Robert, Alexander D. Roberta. Ultrasound of the placenta and umbilical cord. *Ultrasonodraphy in obstetrics and gynaecology by Callen: 4thed, 2000. Philadelphia: WB.SaundersCSompany, Philadelphia: 597.*
13. Harris VG. Relation between placental site and length of gestation. *Br J ObstetGynaecol* 1975;82:581.
14. Hoogland HJ, deHaan J. Ultrasonographic placental localization with respect to fetal position in utero. *Eur J ObstetGynecolReprodBiol* 1980;11:9.
15. Kalanithi LE, Illuzzi JL, Nossov VB, et al. Intrauterine growth restriction and placental location. *J Ultrasound Med S.* 2007; 26: 1481-9.
16. Kian LS. The role of the placental site in the aetiology of breech presentation. *J ObstetGynaecol Br Commonw* 1963;70:795.
17. Kobayashi M, Hellman LM, Fillisti L. Placental localization by ultrasound. *Am J OBSTET GYNECOL* 1970;106:279.
18. Kofinas AD, Penry M, Greiss FC Jr, Meis PJ, Nelson LH. The effect of placental location on uterine artery flow velocity waveforms. *AM J OBSTET GYNECOL* 1988;159:1504.
19. Kofinas AD, Penry M, Swain M, et al. Effect of placental laterality on uterine artery resistance and development of preeclampsia and intrauterine growth retardation. *American J Obstet Gynecol.* 1989; 161: 153-69).
20. Little WA, Friedman EA. Significance of placental position. *ObstetGynecol* 1964;23:804.
21. Majumdar S, Dasgupta H, Bhattacharya K, Bhattacharya A. A study of placenta in normal and hypertensive pregnancies. *J. Anat. Soc. India* 2005; 54(2):34-8.
22. Pai Muralidhar V, Pillia J. Placental laterality by ultrasound- A simple yet reliable predictive test for preeclampsia. *J ObstetGynecol India.* 2005; 55: 431-3.
23. Sandhya K, Madhavi G.B. and Chandramathi M. placental laterality as a predictor of preeclampsia. *AJPCT* (3)(03) (2015)231-236).
24. Roberis JM and Hubel CA. Is oxidative stress the link in the two stage model of preeclampsia. *Lancet.* 1999; 354:788-9.
25. Schulman H, Winter D, Farmakides G, et al. Pregnancy surveillance with Doppler velocimetry of uterine and umbilical arteries. *American J Obstet Gynecol.* 1989; 169:192-6.
26. Thompson W, Bell TK, Pinkerton JHM. Location of placenta by means of a colourautoscan technique. *Br Med J* 1967;4:390.
27. Townsend DE, Swanson LA. Determination of placental location with technetium-99M. *Am J OBSTET GYNECOL* 1969;104:560.
28. Udainia A, Jain ML. Morphological study of placenta in pregnancy induced hypertension with its clinical relevance. *J. Anat. Soc. India* 2001; 50(1):24-7.

29. Van E Beek, Peters LLH. Pathogenesis of Preeclampsia. A comprehensive model. *ObstetGynaecol survey*. 1998; 53(4):233-39.
30. Vrettos AS, Megapanos E, Costamis P, Binopoulos D. isotopic placentography using Cr⁵¹-tagged erythrocytes. *AMJ OBSTET GYNECOL* 1965;93:957.