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Research Article

PLACENTAL MORPHOLOGY AND ITS CO-RELATION WITH FOETAL OUTCOME IN PREGNANCY-INDUCED HYPERTENSION

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ABSTRACT
The Aim was to undertake a detailed study of placental morphology in pregnancies complicated by hypertension to assess the spectrum of placental changes and to correlate these findings with severity and duration of maternal disease and with the foetal outcome. A study of sixty placentae was done with the collaboration of Department of Obstetrics and Gynaecology, to find out the morphological changes of placenta in 30 women suffering from pregnancy induced hypertension in comparison to 30 women with uncomplicated gestation and their fetal outcome. Mean placental weight and volume was found to be much lower in the study group. Macroscopic features like retroplacental haematoma, grossly discernable infarcts and calcification was found to be more in the placentae of mothers suffering from PIH. The mean birth weight of babies in PIH was less as compared to the control group; also the incidence of still births was more.

Key Words: *Pregnancy Induced Hypertension, Retroplacental Haematoma, Foetal Outcome*

INTRODUCTION

The placenta is the most accurate record of infant's prenatal experiences. Generally physicians are uncomfortable with the task of examining the placenta, but it is a task they should willingly undertake because submitting this organ to a knowledgeable look and touch can provide much insight into prenatal life. According to Park (2009) the hypertensive disorders are responsible for 5-8 % of all maternal deaths. A wide variation in placental size has been reported in pre-eclamptic women. Usually the placental size is described as smaller than normal but unduly large and occasionally hydropic placentae have also been reported. As the placenta is the direct link between mother and foetus, the examination of placenta should give the clear idea of what had happened with it, when it was in the mother's womb and what is going to happen with the foetus in the future. With this objective the present study was carried out.

Brosens *et al.,* (1977) said that the retro placental haemorrhage is due to rupture of maternal decadal arteriole, the wall of which is weakened because of the changes that occur in pre-eclampsia. Fox (1973) said that in pre-eclampsia the functional reserve capacity of the placenta is diminished by the utero-placental ischaemia. Under these conditions, a further loss of functional tissue by extensive infarction as a result of a large haematoma may compromise placental function to the extent that it fails to provide the foetus with an adequate supply of oxygen and nutrients. Fox (1978) postulated that extensive placental infarction is associated with a high incidence of foetal hypoxia, intra-uterine growth retardation and death.

Tindall and Scott (1965) found that calcification occurs more commonly in first pregnancies and its incidence is directly related to low maternal age, high maternal socio-economic status and delivery during the summer months. Brown *et al.,* (1988) found that placental calcification occurs earlier in pregnancy in cigarette smokers. Udainia and Jain (2001) found that a linear correlation exists between weight of newborn baby and weight of placenta in uncomplicated pregnancies. For mild and severe pregnancy induced hypertension cases also, they found that linear relationship exists between the weight of newborn baby and the weight of placenta. Templeton and Campbell (1977) estimated the prenatal death rate in eclamptic women as 136/1000. Whereas Chamberlain *et al.,* (1970) showed a prenatal mortality rate of 33.7/1000 in severe pregnancy induced hypertension and eclampsia as compared to the rate of 19.2/100 in normotensive pregnancies.

MATERIALS AND METHODS

The present study is based on the observations made on the placentae of patients suspected to be the cases of PIH. Due clearance was taken from the hospital ethical committee before proceeding with the study. For the study, 60 placentae were selected at random from the women delivered at the Department of Obstetrics and

Gynecology. Thirty out of sixty placentae were from controls and thirty were from cases of hypertensive pregnancies. The placentae were divided into four groups viz;
Group 1 Eclampsia.
Group 2 Moderate pre–eclampsia.
Group 3 Mild pre–eclampsia.
Group 4 Control group.
The criteria adopted for grouping of these cases were defined according to The International Society for the Study of Hypertension in Pregnancy Classification followed by the American College of Obstetrics and Gynecologists (ACOG, 1988). The alterations in blood pressure were observed on at least two different occasions, at least six hours apart.
The placentae were collected soon after delivery and kept in10 percent formalin. After the placental membranes were trimmed off, the umbilical cord was cut to a length of 2.5 cms, the placenta were washed gently to remove any adherent clots and then gently dried with filter paper. The weight of the placenta was then obtained on the weighing machine. The volume of the placenta was obtained by water displacement method in a graduated one-liter beaker. Detailed gross examination of the placenta was done to note the shape and any abnormalities in the placental shape. The fetal surface of the placenta was scrutinized, the state of membranes was noted and a search was made for subamniotic hematoma. The maternal surface was examined for presence of any retro placental hematoma and calcification. The mode of insertion of the umbilical cord was noted along with any umbilical cord abnormalities.

RESULTS

Out of 30 patients of hypertensive pregnancies, there were 6 cases (20%) of eclampsia, 10 cases (33.33%) of moderate pre-eclampsia and 14 cases (46.67%) of mild pre-eclampsia. In the control group, there were 30 disease free women with uncomplicated gestations. On gross examination the term placenta was seen to be a disc shaped mass with circular or oval outline. The maternal surface was observed to be made up of a number of cotyledons, the number of which ranged from 12-20. The fetal surface had a bluish colour in all the placentae.
Assuming 400-600 gms as the normal placental weight, 83.33% of cases of eclampsia had placental weight lower than or equal to 400gms with a mean of 329.17 grams. Placental weight in cases of moderate and mild pre-eclampsia was recorded below or equal to 400 gms in 60% of cases and 50% of cases respectively. Further in the control group this finding was observed in only 36.67% of cases. The mean placental volume in the control group was 431.17±79.13 cu.cm, which was much more as compared to the study group. The mean placental volume in Group 1 was 328.33±122.38 cu.cm; in Group 2 it was 371±65.74 cu.cm. and slightly more in Group 3 to the tune of 409.64±57.19 cu.cm..

Table 1: Placental weight and volume in cases and controls

GROUP	PLACENTAL WEIGHT (gms) RANGE	PLACENTAL WEIGHT (gms) MEAN±SD	PLACENTAL VOLUME (cu.cm.) RANGE	PLACENTAL VOLUME (cu.cm.) MEAN±SD
GROUP 1	210-540	329.17±123.47	200-530	328.33±122.38
GROUP 2	300-490	379.5±67.35	290-480	371±65.74
GROUP 3	300-520	412.5±60.47	310-520	409.64±57.19
GROUP 4	320-615	439.50±73.90	305-615	431.17±79.13

Grossly discernible infarcts were seen in 20 out of 30 cases in the study group as compared to 4 out of 30 controls and their incidence ranged from 64.29% in mild pre-eclampsia to 66.67% in cases of eclampsia. Retroplacental haematoma was seen in 2 out of 6 cases of eclampsia, 3 out of 10 cases of moderate pre-eclampsia and 4 out of 14 cases of mild pre-eclampsia. The controls did not show retroplacental haematoma. Calcification of varying degree was noted in 100% cases of eclampsia, 70% cases of moderate pre-eclampsia and to the tune of 64.29% cases in mild pre-eclampsia and in only 10% of the placentae in the control group. The manner of insertion of the umbilical cord was seen to be eccentric in almost 80% of the cases and controls, the rest being central attachment.

Table 2: Macroscopic placental abnormalities

Pathologic feature		Group 1 Eclampsia N = 6	Group 2 Mod.Pre Eclampsia N =10	Group 3 Mild Pre Eclampsia N =14	Group 4 Controls N =30
Placental weight (gms)	≤ 400	5(83.33%)	6(60%)	7(50%)	11(36.67%)
	Mean	287	335.83	365	364.55
	400-600	1(16.67%)	4(40%)	7(50%)	18(60%)
	Mean	540	457.50	460	475.56
	>600	--	--	--	1(3.33%)
	Mean	--	--	--	615
Infarct	Absent	2(33.33%)	3(30%)	5(35.71%)	26(86.67%)
	Present	4(66.67%)	7(70%)	9(64.29%)	4(13.33%)
Retro placental Haematoma	Absent	4(66.67%)	7(70%)	10(71.42%)	30(100%)
	Present	2(33.33%)	3(30%)	4(28.58%)	--
Calcifi-cation	Absent	--	3(30%)	5(35.71%)	27(90%)
	Present	6(100%)	7(70%)	9(64.29%)	3(10%)

The shape of the placenta was discoidal in 73.33% of cases in the study group (figure 1) and 83.33% in the control group (figure 2). Other shapes that were observed were irregular (16.67% in study group and 10% in control group) and bidiscoidal, lobed and diffused (3.33% each) in both study and control group.

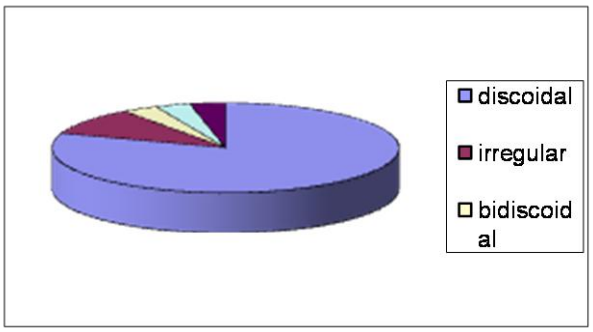

Figure 1: Shape of placenta in study group

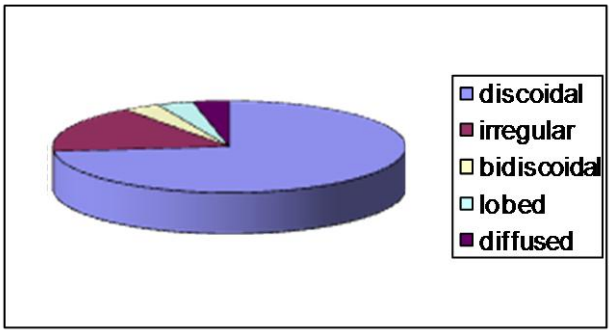

Figure 2: Shape of placenta in control group

One out of 6 cases of eclampsia delivered a stillborn baby. 2 stillborn babies were delivered in moderate pre-eclampsia and three in mild pre-eclampsia. All the controls delivered healthy normal babies. The mean birth weight of babies in eclampsia, moderate pre-eclampsia, mild pre-eclampsia was lower (2.16 ± 0.72 kg, 2.59 ± 0.28 kg and 2.79 ± 0.42 kg respectively) as compared to the control group, which had mean foetal weight of 3.27 ± 0.46kg. The decrease in birth weight in group 1 when compared with that of control group, was significant ($p<0.05$), whereas, though there was decrease in the birth weight in group 2 & 3, but this decrease in weight was non-significant.

Table 3: Foetal outcome in all the four groups

Group	Live Birth	Still Birth	Range Mean Birth Weight (Kg)	Apgar Score at 1min <7	≥7
Group 1	5	1	1.5-3.2 2.16 ± 0.72	-	5
Group 2	8	2	2.18-3.14 2.59 ± 0.28	1	7
Group 3	11	3	2.0-3.5 2.79 ± 0.43	2	9
Group 4	30	-	2.14-4.4 3.27 ± 0.46	30	-

Table 4: Correlation of macroscopic placental changes with foetal outcome in control and study group

Macroscopic placental Changes	Criteria	Foetal outcome In study group No. of cases	Foetal outcome In control group No. of cases
Placental weight (gms)	≤400	8/18	10/11
	400 – 600	6/12	17/18
	>600	--	1/1
Infarcts	Absent	5/10	24/26
	Present	9/20	4/4
Retro placental	Absent	10/21	28/30
Haematoma	Present	4/9	--
Calcification	Absent	4/8	25/27
	Present	10/22	3/3

Foetal outcome was taken as defined by the following criterion:
Live born
Birth weight ≥2.5Kg Apgar score ≥7

DISCUSSION

Fox and Langley (1973) described the placenta as the mirror of the perinatal period. Londhe and Manel (2011) also observed that placental weights were lower in cases of pre-eclampsia than in controls. Kher and Zawar (1981), Kalo *et al.,* (1979) and Bhatia *et al.,* (1981) have reported reduced placental weight in cases of higher grades of hypertension, while placentae from cases of mild pre-eclampsia were reported to weigh within normal range. The study of Udainia *et al.,* (2001) found the mean placental weight of 435.63gm in mild hypertension and 371.43gm in severe hypertension. It is well established by Browne and Veall (1953) that the placenta in toxaemia receives reduced maternal blood flow, which according to Little (1960) is responsible for some reduction in its weight.

Contradictory findings of heavier placentae in cases of hypertensive pregnancies have been explained by different hypothesis. Fox (1997) and Wigglesworth (1962) have observed some degree of compensatory hypertrophy in the placental mass leading to increase in the placental weight. This may be a consequence of placental insufficiency, secondary to inadequate utero-placental blood flow. Similar to our findings Kishwara *et al.,* (2008) found the placental volume lower in the hypertensive women.

Wide variations in the incidence of placental infarcts have been reported by Fox and Langley (1973), ranging from 34% in women with mild pre-eclampsia to 60% in women with the severe form of the disease. Salvatore (1968) also noted a higher incidence of red (34.3-66.6%) and white (40.6-65.5%) infarcts depending upon the severity of toxaemia as compared to 23.3% and 33.3% respectively in normal placentae. Various other workers like Wigglesworth (1962); Maqueo *et al.,* (1961); Kher and Zawar (1981); Kalo *et al.,* (1979); Bhatia *et al.,* (1981); Little (1960); Brosens and Ranaer (1972) and Mathews *et al.,* (1973) have reported similar higher incidence of placental infarction in cases of toxaemia of pregnancy.

Shanklin (1959) and Torpin and Swain (1966) have made a contradictory observation of no association of placental infarcts with hypertensive pregnancies. To evaluate the significance of placental infarction in hypertensive pregnancies workers like Little (1960); Wigglesworth (1962); Brosens and Renaer (1972); Mathews *et al.,* (1973) and Masodkar *et al.,* (1985) have studied the extent of infarction and have highlighted that placental infarcts involving more than 10% surface area are significant.

However, diagonally opposite views undermining the significance of this observation, have been expressed by Earn (1950), who stated that the placenta can withstand infarction of more than one half of its substance without any deleterious effect on the fetus. On the basis of our data, no definite comment can be offered on the significance of placental infarction as far as its extent is concerned.

Retroplacental haematoma were observed in 33.33% (2/6) cases of eclampsia, 30% (3/10) cases of moderate pre-eclampsia and 28.58% (4/14) cases of mild pre-eclampsia.

Fox (1997) has reported the incidence of retro placental haematoma in toxaemic subjects as ranging from 12-15%, but a correlation with the severity of disease has not been well established. Salvatore (1968) reported an incidence of retro placental haematoma in mild and severe pre-eclampsia as 3.1% and 25.8% respectively.

There are conflicting views regarding the relationship between this lesion and toxaemia of pregnancy. Barthalomew and Kracke (1932) maintain that retro- placental haematoma occurs as a complication of pre-eclampsia, while others like Hibbard and Jeffcoate (1966) considered that the haematoma causes the pre-eclampsia.

Placental calcification was noted in 73.34% (22/30) cases of various grades of hypertensive pregnancies. However, 10% (3/30) of the placentae from the control group also showed calcification. Thus, placental calcification was seen more commonly in placentae from hypertensive patients. Similar findings have been reported in this regard by Fox (1997), Shanklin (1959) and Mehrotra *et al.*, (1972).

A correlation between placental calcification and primigravidity has been noted in the present study. 72.73% (16/22) placentae in the study group, showing calcification, were from primigravidae as compared to 33.3% (2/6) from the control group. This relationship between placental calcification and primigravidity has reported by Fox (2007) and Wentworth (1963).

Kher and Zawar (1981) have reported a significant reduction in the feto placental weight ratio. Wigglesworth (1962) has experimentally showed it in rats that reduced placental blood flow results in a smaller fetus. Gruenwald (1961) hypothesized that a smaller placenta reflects poor foetal growth.

Nummi (1972) however reported contradictory findings, that there is a poor correlation between foeto-placental weight ratio and maternal or foetal complications. Fox (1997) noted that in many hypertensive gestations there is decreased foeto-placental weight ratio because of a compensatory hypertrophy of the placenta under the influence of unfavorable maternal environment.

Of the 73.33% hypertensive placentae associated with calcification, there were foetal complications like stillbirth, hypoxia and low birth weight. This is in contradiction to Fox (2007) who has specified that placental calcification does not have any pathological or clinical significance. So these results may be coincidental to the other placental lesions.

Conclusion

A comparative study between 30 placentae from women with various grades of hypertension during pregnancy and 30 from disease free gestation was conducted. Placental weight and volume were found to be much lower in higher proportion of cases of eclampsia and moderate to mild pre-eclampsia. Lower foeto-placental weight ratio was observed in cases of severe form of the disease than in cases with milder forms of toxaemia. It was thus concluded that lighter placentae usually accompanied a low birth weight of the foetus. The three main gross lesions which were observed were placental infarcts, retroplacental haematoma and calcification and their incidence in hypertensive pregnancies was higher as compared to placentae from the control group. Placental calcification was seen more commonly in the study group, while the control group also showed this feature, but to a lesser degree.

REFERENCES

Bartholomew RA and Kracke RR (1932). The relation of placental infarcts to eclamptic toxaemia; a clinical, pathological and experimental study. *American Journal of Obstetrics and Gynaecology* **24** 797.

Bhatia A, Sharma SD and Jalnawalla SF (1981). A comparative study of placental pathology and fetal outcome. *Indian Journal of Pathology and Microbiology* **24** 277

Brosens I and Ranaer M (1972). On the pathogenesis of placental infarcts in pre-eclampsia. *Journal of Obstetrics and Gynaecology of British Common Wealth* **79** 794

Brosens I, Robertson WB and Dixon HG (1977). Fetal growth retardation and the vasculature of the placental bed. *British Journal of Obstetrics and Gynaecology* **84** 656-664.

Brown HL, Miller JM, Kharoli D and Gabert HA (1988). Premature placental calcification in maternal cigarette smokers. *Obstetrics and Gynecology* **71** 914-917.

Browne JCM and Veall N (1953). Maternal blood flow in normotensive and hypertensive women. *Journal of Obstetrics and Gynaecology of the British Empire* **60** 141.

Chamberlain G, Phillip E, Howlett B and Masters K (1970). British births, Obstetric care, Heinemann London **2** 82-107.

Davey DA and Mac Gillivray I (1988). The classification and definition of the hypertensive disorders of pregnancy. *American Journal of Obstetrics and Gynaecology* **158** 892-898.

Earn AA (1950). Macroscopic examination of the placenta. *Canadian Medical Association Journal* **62** 602.
Fox H and Langley F (1973). The pathology of infarction in perinatal morbidity and mortality. *Biologica Neonatorum* **11** 87.
Fox H (1978). Pathology of the placenta. London: WB Saunders 477.
Fox H (1997). Pathology of the Placenta. 2nd edition London, United Kingdom: WB Saunders Company Ltd
Fox H (2007). Pathology of the placenta. 3rd edition Philadelphia: Saunders Elsevier 100-103.
Gruenwald P (1961). Abnormalities of placental vascularity in relation to intrauterine deprivation and retardation of fetal growth. *New York State Journal of Medicine* **61** 1508.
Hibbard BM and Jeffcoate TNA (1966). Abruptio placentae. *Obstetrics and Gynaecology* **27** 155.
Kalo J, Levin S and Rosin N (1979). Correlation between pathological findings of the placenta and clinical entities of the mother and fetus. *Journal of Obstetrics and Gynaecology of India* **29** 340.
Kher AV and Zawar AP (1981). Study of placental pathology in toxemia of pregnancy and its fetal complications, *Indian Journal of Pathology and Microbiology* **24** 245.
Kishwara S, Nurunnabi ASM, Begum M, Ahmed R and Ara S (2008). Study of proportional and absolute volume of placental parenchyma and non parenchyma between normal pregnant and preeclamptic women. *Journal of Dhaka Medical College* **17**(2) 78-82.
Londhe Pradeep S and Mane Abhay B (2011). Morphometric study of placenta and its correlation in normal and hypertensive pregnancies. *International Journal of Pharma and Biosciences* **4**(2) 429-437.
Little WA (1960). Placental infarction. *Obstetrics and Gynaecology* **15** 109.
Masodkar AR, Kalamkar LR and Patki PS (1985). Histopathology of placenta and its correlation with foetal outcome. *Journal of Obstetrics and Gynaecology of India* **35** 294.
Mathews R, Aikat M and Aikat BK (1973). Morphological studies of placenta in abnormal pregnancies. *Indian Journal of Pathlogy and Microbiology* **16** 15.
Maqueo M, Azuela JC and Manuel Dosal de la Vega (1964). Placental pathology in eclampsia and pre-eclampsia. *Obstetrics and Gynaecology* **24** 350.
Mehrotra VG, Mukherjee K, Pande M and Gurtu M (1972). The histological study of placenta in normal and abnormal pregnancy. *Journal of Obstetrics and Gynaecology of India* **22** 248.
Nummi S (1972). Relative weight of the placenta and perinatal mortality. A retrospective clinical and statistical analysis. *Acta of Obstetrics and Gynaecology of Scandinavia Supplement* **17** 1-69.
Park K (2009). Textbook of Preventive and Social Medicine, 20th edition, *Banarsidas Bhanot Publishers*, Jabalpur 482.
Salvatore CA (1968). The placenta in toxemia: a comparative study. *American Journal of Obstetrics and Gynaecology* **102** 347.
Shanklin DR (1959). The human placenta with special reference to infarction and toxaemia. *Obstetrics and Gynaecolgy* **13** 325.
Templeton A and Campbell D (1977). A retrospective study of eclampsia in the Caspian region. *Health Bulletin* **37** 55-59.
Tindall VR and Scott JS (1965). Placental calcification: a study of 3025 singleton and multiple pregnancies. *Journal of Obstetrics and Gynaecology of British Common Wealth* **72** 356.
Torpin R and Swain B (1966). Placental infarction in 1000 cases, correlated with clinical findings. *American Journal of Obstetrics and Gynaecology* **94** 284.
Udainia A and Jain ML (2001). Morphological study of placenta in pregnancy induced hypertension with its clinical relevance. *Journal of Anatomical Society of India* **50**(1) 24-27.
Wentworth P (1967). Placental infarction and toxaemia of pregnancy. *American Journal of Obstetrics and Gynaecology* **99** 318.
Wigglesworth JS (1962). The Langhans layer in late pregnancy. A histological study of normal and abnormal cases. *Journal of Obstetrics and Gynaecology of British Common Wealth* **69** 355.