

## Morphological changes in placenta of hypertensive pregnant women

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### Abstract

**Background:** The placenta reflects the status of maternal hypertension as it is mirror of the maternal and fetal health.

**Aim:** To find out the morbid changes in the placenta of hypertensive mothers in comparison to those mothers with normal pregnancies.

**Methods:** This study was carried out on fifty mothers with pregnancy induced hypertension (PIH), and the results are compared with the findings in 50 mothers with uncomplicated pregnancy. Statistical significance of difference between two groups was calculated by using Students "t" test. A difference between the two groups was considered to be significant when  $p < 0.05$ .

**Result:** Macroscopic study revealed that, compared to the controls there was trend of less placental diameter in eclamptic group ( $p = 0.0001$ ). Cotyledon number was found to be significantly less in eclampsia ( $p = 0.0001$ ). Cytotrophoblast invasion are limited to the superficial decidua, and few arterioles are breached due to abnormalities in adhesion molecule switching by invasive cytotrophoblasts, suggesting that this subpopulation of trophoblast cells fails to differentiate properly in hypertensive placenta.

**Conclusion:** Patients with pregnancy induced hypertension are found to have increased risk of low birth weight babies, which may be the result of smaller placental weight and area.

**Key Words:** hypertension, cytotrophoblast, placenta, histology.

### INTRODUCTION

Examination of placenta immediately after delivery provides much insight into the prenatal health of the baby and the mother.<sup>1</sup> Pregnancy complications like hypertension or gestational diabetes are reflected in placenta in a conspicuous way. Researches suggest that placental surface area is significantly less in PIH, but none have mentioned the exact surface area.<sup>2,3</sup> Reduced maternal utero-placental blood flow leading indirectly to constriction of foetal stem arteries has been attributed to these changes.<sup>4</sup> Maternal vasospasm leads to foetal hypoxia, fetal distress and death, which is fairly common towards the later part of pregnancy.<sup>5</sup>

In recent years, it has been revealed that there is a substantial relationship between confined placental mosaicism and foetal growth retardation.<sup>6</sup> An estimated 70% of the excess foetal deaths in PIH can be ascribed to large placental infarcts and markedly small placental size.<sup>7</sup>

Histopathological changes related to confined placental mosaicism may be associated with inadequate placentation and hence with

retroplacental ischaemia.<sup>8</sup> Present study has been undertaken to assess the morphology and histology of placenta from mothers with PIH and to correlate the findings with those from normal pregnancies.

### MATERIALS AND METHODS

Fifty mothers with uncomplicated pregnancies and 50 mothers with PIH were randomly selected. The age range of these mothers varied from 22 years to 34 years. Among the mothers with PIH, 24 were without proteinuria and oedema; whereas 26 had pre-eclampsia (hypertension was associated with oedema or proteinuria or both). All these mothers were normotensive before pregnancy. Mothers were examined clinically (for height, weight, blood pressure, pulse, anaemia, jaundice etc.) along with recording of their medical history (history of past illness, history of previous child birth etc). Mothers with PIH had their blood pressure more than 140/90 mm of Hg.

Placenta with cord and membranes were collected immediately after delivery for morbid and histopathological studies. Any abnormality of cord and membrane was noted. The amnion and

chorion were trimmed from the placenta. Umbilical cord was cut at a distance of 5 cm from the site of insertion. Placenta were washed in running tap water, dried with the help of blotting paper and weighed. Then surface area of maternal surface of placenta was calculated by taking its imprint on a graph paper.

The size, shape were noted along with the inspection of marginal veins for any thrombus; the number of cotyledons, condition of membranes, presence of infarction, calcification and site of insertion of umbilical cord. The newborn babies were inspected for congenital anomalies, Apgar score etc. Their birth weights were noted and the foeto-placental weight ratio was calculated in each case. Tissues for histopathological studies were taken from near the implantation of the umbilical cord; from margins at 12, 3, 6, 9 O'clock positions; centre of placenta; and fibrotic or infarcted area, if any.

## RESULTS

Mean birth weight of new born babies was 3.0Kg + 0.52 Kg in the control group and 2.3 + 0.4 Kg in the hypertensive group. Apgar score was below 7 in 15 babies of PIH group and in two such babies it was 2 and 3 only; whereas, in the control group it was within normal limit. The mean placental weight, mean placental area and mean placental volume were significantly less ( $p>0.01$ ) in the PIH group compared to normal. The mean foeto-placental weight ratio was significantly higher in the hypertensive group. The mean number of infarcted areas, calcified areas and marginal insertion of umbilical cord was significantly higher ( $p>0.01$ ) in PIH group to compared to controls. However, the number of cotyledons show lower values in hypertensives, but was not statistically significant. (Table-1)

The histopathological examination of placenta revealed that number of syncytial knots, cytotrophoblastic cellular proliferation, fibrinoid necrosis, endothelial proliferation, calcified and hyalinised villous spots were significantly higher in the PIH group than the

**Table-1.** Placental and fetal morphometric study

Pathology	Control Mean±SD	Hypertensive Mean±SD	P'Value
<b>Placental and fetal morphometric study</b>			
Birth weight in kgs	3.0±0.52	2.3±0.4	p<0.05
Placental weight in gms	510± 64	404± 42	p<0.05
Placental area in sqcm	280±72	211±56	p<0.05
Placental volume in cc	640±184	402±126	p<0.05
Foeto-placental wt ratio	5.94± 0.86	6.02± 0.36	p<0.05
<b>Gross morphology Placenta</b>			
No of cotyledons per placenta	16±2	14±2	Not significant
No of infarcted area in placenta	3±1.5	11±1.5	p<0.05
No of marginal insertion of umbilical cord	7± 0.5	12± 0.5	p<0.05
No of calcified areas in placenta	5±1.5	18±1.5	p<0.05

normal. Study of stromal and villous histopathological changes revealed that stromal fibrosis, pseudo-infracts, medial coat proliferation of medium sized blood vessels and mean number of calcified and hyalinized areas per low power field were significantly higher ( $p>0.01$ ) in the hypertensive group. (Table-2)

**Table-2.** Microscopic study of placental villi

Morphology	Control Mean±S.D.	PIH. Mean±S.D.	p' Value
<b>Placental villi pathology</b>			
No of areas of syncytial knot formation	10±3.5	20±2.85	p<0.05
No of areas of cytotrophoblastic cellular proliferation	6±2	14±3.5	p<0.05
No of areas of fibrinoid necrosis	4±1.8	8±2.4	p<0.05
No of areas of hyalinised villi	3.5±1.2	13±2.7	p<0.05
<b>Stromal pathology</b>			
No of areas of stromal fibrosis/lpf	3.5±0.8	11.5±2.8	p<0.05
No of areas of medial coat proliferation of medium sized blood vessels/lpf	4.2±1.2	14.5±2.6	p<0.05
No of calcified areas/lpf	3.2±1.1	13.6±2.4	p<0.05
No of hyalinised areas/lpf	4.5±1.5	15.6±2.8	p<0.05

## DISCUSSION

Present study revealed that the fetal weight is significantly less in the hypertensive group than the control group. The morphometry of placenta i.e. weight, surface area and volume show significantly lower values in the PIH patients. These findings corroborate well with that of the other studies.<sup>7,8,9</sup>

The placental weight and size were directly proportional to the birth weight of babies.<sup>10</sup> This may be due to altered intracotyledonous vasculature seen in hypertension.<sup>9</sup> It was also established that the average weight of placenta is reduced proportionately with the degree of hypertension.<sup>11</sup>

The gross anatomic features of placentae i.e., the mean number of infarcted areas, calcified areas and marginal insertion of the umbilical cord are higher in PIH group than normal. Similar observations were made in previous studies.<sup>5,8</sup>

We also observed that in PIH patients, there is a significant increase in the number of syncytial knot formation, cytotrophoblastic cellular proliferation, proliferation of endothelial lining of capillaries, stromal fibrosis, calcification and hyalinisation of villi. It may be the results of overall reduction of foetal perfusion of the placenta.<sup>12</sup>

Histological findings of placentae, i.e., evidence of cytotrophoblastic cellular hyperplasia and patchy necrosis of the villous syncytiotrophoblastic cells are also obvious in the study group, which is in accordance to the previous studies conducted earlier.<sup>8</sup> It is established that the physiology of the uterine vascular pattern, notably of the spiral arteries in patients is remarkably modified following decidual implantation of blastocyst and during placentation in hypertensive patients.<sup>12</sup> A significant increase in the cytotrophoblastic cellular proliferation and syncytial knot formation in the placental villi may also indicate a disturbance

in the hormonal factors which may probably lead to altered morphometry of placenta resulting in PIH in the mother and low birth weight babies<sup>15</sup>. Role of marginal insertion of umbilical cord in the placenta has also been implicated in the induction of hypertension.<sup>9,13</sup>

Microscopic findings of localized fibrinoid necrosis, endothelial proliferation of arteries and hyalinization depict the mosaicism of placenta are probably the aftermath of hypertension.<sup>5</sup> Again the mosaicism of the placenta probably leads to placental insufficiency and ultimately to foetal growth retardation, thus creating a vicious cycle.<sup>14</sup>

## CONCLUSION

Patients with pregnancy induced hypertension have increased chance of ischemic damage to the placental tissue along with maldeveloped terminal villi. These findings may account for impaired gas and nutrient transfer in this disorder, thereby resulting in low birth weight babies.

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