

## Investigation into the Morphology and Histology of the Placenta in Mothers with Hypertension

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### Abstract

**Background and Objectives:** Hypertensive pregnancy may be responsible for vascular damage, enhanced systemic inflammation and insulinresistance in the placenta as oxygen and nutrient transfer is impaired and oxidative stress is generated affecting the placental growth and development. Placental growth pattern in hypertensive pregnancies shows a variable pattern owing to placental insufficiency.

**Method:** The study was carried out on 80 sets placenta, mothers and their babies. The placenta were collected from Obstetrics & Gynecology Department of NMCH, Patna. Out of 80 placenta, 40 were from normal pregnant mothers and 40 from pregnancies complicated by PIH in previously normotensive women, serial number of placenta, mother and baby were same. placenta were collected soon after the normal vaginal delivery or caesarian section along with 10 cm long stump of umbilical cord. The aim of this study was to determine whether maternal PIH would affect the morphology and histology of placenta and compare this with that of normotensive mothers. It was a cross-sectional comparative study carried out at the maternity ward and anatomy department of NMCH, Patna. Histological findings were cytotrophoblastic cellular proliferation, syncytial knot formation, fibrin plaque formation. In 20 percent cases, villi were hypovascular. The hypovascular villi have abundance of syncytial knots, usually lack vasculosyncytial membrane and increased stromal collagen.

**Conclusion:** PIH adversely affect both morphology and histology of placenta.

**Keywords:** morphology, histology, placenta, hypersensitive & mothers.

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### Introduction

Pregnancy-induced hypertension (PIH) is the leading cause of maternal mortality is an important factor in fetal wastage. Pregnancy complications like hypertension are reflected in placenta in a significant way both macroscopically and microscopically. Several studies have shown that utero-placental blood flow is decreased in PIH due to maternal vasospasm. This leads to constriction of fetal stem arteries and has been associated with the changes seen in the placenta of PIH mother. The fetal pole of placenta consists of chorionic plate, composed of an outer layer of trophoblast and inner layer of vascularised EE mesodermal connective tissue. The bulk of the placenta fetalis consists of outgrowth of villi from the surface of chorionic plate. In the mature placenta the villi are about 40 micrometers in diameter [1]. Their trophoblastic covering layer is irregularly thinned and attenuated. Cytotrophoblastic cells though still present are fewer in number and less prominent. The syncytial nuclei are often aggregated to form multinucleated protrusions from the villous surface, these being known as syncytial knots. These are different from syncytial sprouts and syncytial buds.

Syncytial sprouts are present from the early stages of pregnancy and represent the initial stages in the development of lateral villi those that do not form villi tend to become pedunculated and latter break off from the villous surface to enter the inter villous space and the maternal circulations. Syncytial buds are an invagination of the trophoblast into the underlying stroma. Syncytial knots in contrast are very infrequently seen before the last two months of gestation, but thereafter they appear in rapidly increasing numbers become unduly numerous in prolonged pregnancies [2]. In some areas of many villi the syncytium between the knots in anuclear and attenuated, if such an area overlies a dilated fetal capillary it may on light microscopy, appear to fuse with the vessel wall to form what is known as the vasculo syncytial membrane, these are specialized zones of the trophoblast for the facilitation of gas transfer across the placenta. A deficiency of vasculo syncytial membranes in the mature placenta is associated with a high incidence of fetal hypoxia [3]. The villous stromal tissue is usually reduced to a thin compressed layer between the dilated villous capillaries

and contains fibroblasts and little collagen. In the normal full term placenta, each villus usually contains between one and six fetal capillary vessels, which are situated towards the villous periphery in close approximation to the villous trophoblast. They are characteristically sinusoidally dilated and occupy most of the cross-sectional area of the villus [4].

### Material and Method

The study was carried out on 80 sets placentae, mothers and their babies. The placentae were collected from Obstetrics & Gynecology Department of Nalanda Medical College and Hospital Patna, Bihar.

### Material

1. 80 placentae taken for the study, will be collected soon after delivery along with umbilical cord.
2. Weighing machine.
3. Inch tap.
4. Magnifying glass.
5. Dissection instruments.
6. Histological lab, reagents, etc

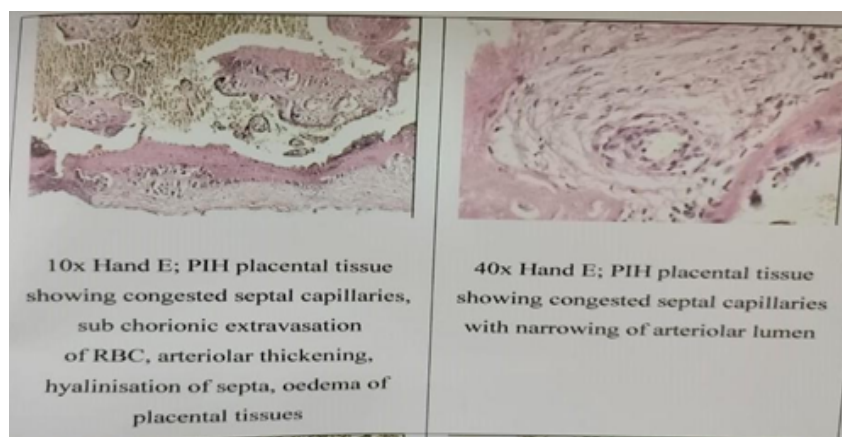
A general examination of the patients on admission into the labour room were noted from clinical records which included heights, weight builds. B.P. Routine laboratory investigations were noted - blood haemoglobin, blood sugar, urine examination USG report recorded from the clinical records. Grouping of the sample:- All the cases were divided into 2 groups : Group A (Control Group) - this group comprises pregnant women without any obstetric history of complications like anaemia pregnancy induced hypertension and gestational diabetes mellitus etc. Group B (PIH group) - this group comprises

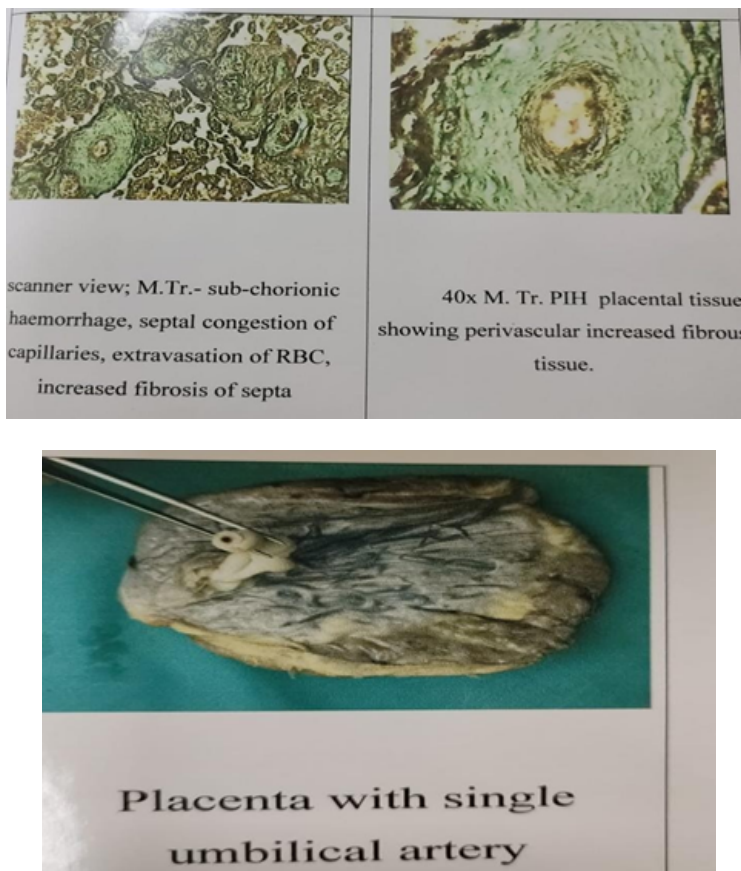
pregnant women with pregnancy induced hypertension. As soon as the placenta was delivered, the umbilical cord was cut it was put into formal saline. It was kept in a tray, to the membranes were trimmed off, cord was cut about 10 cm from the insertion. The blood clots adherent to maternal surface were picked up. The placenta was then washed in plain tap water naked Eye examination was done and following variables studied. Weight of placenta - Accurate weight of the placenta was recorded by weighing machine. Volume of placenta the volume of the placenta was measured by water displacement method. The placenta was immersed in water filled container. The volume of water poured out of the correction was measure is a graduated cylinder marked in millimeters (ml). Shape of placenta - The shape of the placenta and presence of accessory lobe were recorded after proper inspection. Each placenta was categorized as oval, circular or irregular in shape.

Diameter- The placenta was placed in a flat tray. At first the maximum diameter was measured with a metallic scale graduated in centimeter (cm). Then a second maximum diameter was taken at Right angles to the first time. The mean of two measurements was considered as the diameter of the placenta expressed in centimeters. Thickness of Placenta were measured. Number of Cotyledons: Any abnormality - presence of calcification, infarction etc were noted.

**Histology** with the help of knife 5-6 cm piece of placenta were taken from periphery and grossly abnormal areas and histological slides were prepared and stained with H and E STAIN and MASSON'S TRICHROME STAIN histological study; following features were specially noted for syncytial knot formation, calcification, thickening of basement membrane, cytotrophoblastic proliferation, and intervillous haemorrhage.

### Results





In present study 80 placenta were selected for histological and morphological study. This study is carried out on 40 placentae of PIH mothers and 40 of normal mothers, mother with B.P. >140/100 were included in PIH group and normal group as uncomplicated pregnancy. Placenta were examined in regard to weight, volume, shape, diameter, thickness, membranes, attachment of umbilical cord infarction, calcification in number of blood vessels in umbilical cord in morphological study and the mean weight of placenta is found to be 425.00 gm with S.D. of 69.921 in normal placentae. In PIH cases it is found to be 336.00 gm with S.D. of 47.88. The mean volume of placenta is found to be 352.50 ml. with S.D. of 58.88 in normal placentae. In PIH cases it is 236.50 ml with S.D. of 71.959. The mean diameter of placenta in normal subjects is found to be 17.050 cm S.D. of 1.8174 cm whereas in cases of PIH mothers it is found to be 15.61 with S.D. of 1.50. The mean thickness of placenta in normal subjects is found to be 2.16 with of .29 cm whereas in PIH cases it is found to be 1.9 cm with S.D. of .28 cms with S.D. of 2.35. Mean number of cotyledons in normal subjects are found to be 16.00 with SD of 2.35. The mean number of cotyledons in PIH cases are 14.70 with standard deviation of 3.36. The shape of placenta is found to be discoidal in 55% cases among normal placenta. In PIH cases it is discoidal in 65.0% cases. The attachment of umbilical cord is found to be central in 15% of normal subjects, 2.5% of PIH cases, where eccentric attachment is found to be 50% in normal, and 67.5%

in PIH subjects. Marginal attachment is found to be 35.0% in normal, and 30.05% in PIH subjects the mean birth weight of baby is found to be 2.638 kg with S.D. of S.D. 3312 is normal cases, and 2.084 with S.D. of 29 in PIH mother. In our study sub chorionic haematoma is found to be present in PIH mother. Calcification of placenta is found to in term placenta. Among the study group calcification is found to be more in PIH group present in umbilical cord all the placenta, three blood vessels i.e. 2 artery and one vein found to be it in umbilical cord except in one placenta. Placenta of anaemic group showed single umbilical artery. The mean weight of placenta is significantly lower in PIH subjects as compared to normal group ( $p < .05$ ). The mean volume of placenta is significantly lower in PIH subjects as compared to normal group ( $p < .05$ ). The shape of placenta is found to be discoidal in majority of cases of placentae in subjects of all group. Irregular shape of placenta is observed more among PIH cases. In Histological findings were cytotrophoblastic cellular proliferation, syncytial knot formation, fibrin plaque formation, congestion of septal capillaries, sub chorionic extravasation of RBC, arteriolar thickening, hyalinization of septa, oedema of placental tissue. In 20 percent cases, villi were hypovascular. The hypovascular villi have abundance of syncytial knots, usually lack vasculosyncytial membrane and increased stromal collagen.

**Table 1: PI volume[ml]**

| Group  | Mean   | STD Deviation | No. |
|--------|--------|---------------|-----|
| Normal | 352.50 | 58.88         | 40  |
| PIH    | 236.50 | 71.59         | 40  |

**Table 2: Weight PL[gm]**

| Group  | Mean | STD Deviation | No. |
|--------|------|---------------|-----|
| Normal | 425  | 69.921        | 40  |
| PIH    | 336  | 47.889        | 40  |

**Table 3: Mean Diameter[cm]**

| Group  | Mean   | STD Deviation | No. |
|--------|--------|---------------|-----|
| Normal | 17.050 | 1.817         | 40  |
| PIH    | 15.610 | 1.5092        | 40  |

**Table 4: Thickness**

| Group  | Mean  | STD Deviation | No. |
|--------|-------|---------------|-----|
| Normal | 2.075 | 0.2552        | 40  |
| PIH    | 1.960 | 0.2447        | 40  |
| Total  | 2.176 | 0.4858        | 80  |

**Table 5: COTYLEDONS**

| Group  | Mean  | STD Deviation | No. |
|--------|-------|---------------|-----|
| Normal | 14.50 | 2.856         | 40  |
| PIH    | 15.10 | 2.560         | 40  |
| Total  | 13.95 | 3.026         | 80  |

**Table 6: Shape Group Cross Tabulation**

|            | Group  |       |       |
|------------|--------|-------|-------|
|            | Normal | PIH   | Total |
| Irregular  | 02     | 08    | 10    |
|            | 5%     | 12.5% | 12.5% |
| Oval       | 22     | 26    | 46    |
|            | 55%    | 65%   | 57.5% |
| Round      | 16     | 06    | 32    |
|            | 40%    | 15%   | 40%   |
| Triangular | 00     | 00    | 00    |
|            | 00%    | 00%   | 1.4%  |
| Total      | 40     | 40    | 80    |

## Discussion

Present study reveals the weight, volume, diameter, thickness and no. of cotyledons of placenta of pregnancy induced hypertensive mother having lower values is comparison to normotensive mothers. Begum Nunnabi (2011) [5] calculated that 70% of the excess foetal deaths with hypertension are due to large placental infarcts, markedly in women with 1 placental size placental weil. Normal term pregnancy found the placental weight of 400- 1000 grams whereas in PIH group found placental weight to be 360-570 grams. Udainia et al (2004) [6] in his study found the placental weight ranging between 250-700 gm. In the present study the placental weight is found to range between 260-430 gms in PIH cases and 300-850 in normal pregnancy. Chakravorty (1975) [7] in his study of pregnancy induced hypertension found the means placental weight to be 410 gms. Mean placental weight in pregnancy induced hypertension as seen in present study is 318.50(±43.53)grams and is in conformity

with the findings of above author. The mean volume of placenta is found to be 2,36,50 ml (±71.95) patients, which is significantly lower in comparison to normal placentae. In cases of severe preeclampsia or long-standing cases of pregnancy aggravated hypertension placentae were grossly small in volume, irregular in shape. Majumdar (2005) [4]. In the present study the mean placental diameter of PIH mothers is found to be 15.61(1.50) which is significantly lower than the normotensive mothers. ( $p < 0.05$ ). Ashfaq, Janjua, Channa [8] found the mean placental diameter 14,32300.32 but did not found significant difference in placental diameter of normal and hypertensive mothers, The mean of thickness of placenta in PIH group is 1.900(±0.2821) which is similar significantly lower than ( $p < 0.05$ ) with normal study group. In the present study the mean no. of cotyledons found is 14.70(±3.36) which is lower than with the control group but not significant. These findings are in conformity with the study of Kishwara, Ara who found significantly less no. of cotyledons in PIH group (10.0) than control group (11.0). In the present

study marginal insertion of umbilical cord was found to be 30% cases in PIH, which is comparable with findings of the other two study groups. Fox (1967), and Udainia et al (2004) [6] had observed increase in the marginal attachment of umbilical cord in PIH cases. Also, reported cases of marginal insertion of placenta in about 42% cases of pregnancy induced hypertension. Role of marginal insertion of umbilical cord in the placenta has also been implicated in the induction of hypertension Rath G" (2000) [3]. In the present study, irregular shaped placenta found more in PIH mother i.e. 8% than normal study group i.e. 1%. Majumdar et al. (2005) [4] found that mothers with pregnancy induced hypertension had smaller irregular placenta with marginal insertion of umbilical cord. Kishwara, Ara" (2009) [9] found irregular placenta more in PIH group 8% as compared to normal study groups. It has been reported that the maternal utero-placental blood flow is decreased in pre-eclampsia, because there is maternal, vasospasm. Reduced maternal uteroplacental blood flow leading indirectly to constriction of foetal stem arteries, Stock et al (1980) [10] has been associated with the changes in the placenta of pre-eclamptic mothers. Naeye and Friedman (1979) [11] calculated that 70% of the excess foetal deaths in women with hypertension are due to large placental infarcts, markedly small placental size. Normal term pregnancy found the placental weight of 400-1000 grams Udainia et al (2004) [6] in his study found the placental weight ranging between 250-700 gm. In the present study the placental weight is found to range between 260-430 gms in PIH cases and 300-850 in normal pregnancy [12]. Histological findings were cytotrophoblastic cellular proliferation, syncytial knot formation, fibrin plaque formation. In 20 percent cases, villi were hypovascular. The hypovascular villi have abundance of syncytial knots, usually lack vasculosyncytial membrane and increased stromal collagen [13]. It is often stated that in this disease, placenta shows syncytial degeneration and premature ageing. [14]

### Conclusion

That it is concluded that the pregnancy induced hypertension, adversely affects both histology and morphology of placental. Prevention of hypertension in pregnancy may decrease the low birth weight. If these diseases are diagnosed at an early stage by frequent monitoring of blood pressure, blood and urine sugar tests, and clinical examinations, added precaution can be instituted during antenatal period and labour to reduce the further risk to mother and foetus.

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