## **Original Research Article**

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# Morphological study of placenta in pregnancy induced hypertension

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## ABSTRACT

**Background:** The intrauterine existence of fetus is dependent on one vital organ 'the placenta'. The placenta reflects the status of maternal hypertension as it is the mirror of maternal and fetal health. The hypertensive disorders complicate 5-10% of all pregnancies and form a dangerous triad with haemorrhage and infection that contributes greatly to maternal morbidity and mortality. The fetus is dependent on placenta for growth and development. Many disorders of pregnancy like hypertension are accompanied by gross and histological changes in placenta. Aim of the study was to study the various morphological lesions of placenta in pregnancy induced hypertension and compare them with normal pregnancies

**Methods:** Gross and microscopic examination was conducted on 70 placentas. These included 15 normal placentas and 55 placentas from pregnancy induced hypertension.

**Results:** In PIH, on gross the placenta showed areas of infarction, perivillous fibrin deposition and basal decidual haematoma, while microscopically showed increased syncytial knotting, cytotrophoblasitc proliferation, basement membrane thickening, vasculosyncytial membrane deficiency, infarction and fibrinoid necrosis.

**Conclusions:** Maternal disorders affect the placental histology and can be detected by morphological examination of such placentae. The placenta from hypertensive pregnant women show significant morphological changes as compared to control, which may alter the perinatal outcome.

Keywords: Hypertension, Infarction, Placenta, Pregnancy

#### **INTRODUCTION**

The placenta is an organ that connects the developing fetus to uterine wall to allow material uptake, waste elimination and gas exchange via the mother's blood supply.<sup>1</sup> Placenta is essentially a fetal organ, which functions to support the growth of fetus. Normal fetal growth and survival depends on the proper development and function of the placenta.

During its development throughout the gestation placenta undergoes different changes in weight, structure, shape and function continuously.<sup>2</sup> Placenta in latin means cake, that is floppy mass. When pregnancy is complicated by a medical problem like hypertension or diabetes which effects the maternal health, it also affects the architecture and functions of placenta. Thus, examination of placenta gives valuable information about the perinatal health of the baby and mother.<sup>3</sup> Hypertension is one of the common complications met with in pregnancy and diagnosed if maternal blood pressure is more than 140/90 mm of Hg. Hypertension in pregnancy may be chronic hypertension (onset before pregnancy) or may be induced due to pregnancy, preeclampsia and eclampsia. These disorders form one of the deadly triad along with hemorrhage and infection resulting in substantial number of maternal deaths and thereby fetal deaths.

Any disease adversely affecting the mother or fetus brings about morphological changes in the placenta. These morphological changes in the placenta can be studied by its gross and microscopic examination. The information obtained will be of excellent value in understanding the cause of adverse maternal, fetal and neonatal outcomes.

In the present study, detail macroscopic and microscopic changes in placenta in pregnancy induced hypertension (PIH) were studied as the hypertension has adverse effects on morphology of placenta.

#### **METHODS**

This study was carried out in the department of pathology at a tertiary care hospital. The study was done in 70 placentas, which were collected from the obstetrics and gynaecology department. Amongst the 70 placentas collected,55 placentae belonged to women with pregnancy induced hypertension and 15 placentas from women who were normal having uncomplicated full-term deliveries, all the placentas with attached membrane and umbilical cord were collected soon after delivery, washed in running tap water, to clean all the blood and then fixed in 10% formalin. Gross examination of placenta was carried out. The size, the shape, the weight and the site of insertion of umbilical cord were noted down. Four sections were taken from each placenta. Additional sections were taken whenever microscopic lesions were detected. Sections also were taken from the umbilical cord. Tissues were processed for routine paraffin embedding. Sections are cut at 5-micron thickness and finally stained with H and E. Whenever needed, special stains like Masson's trichrome, PAS, Von Kossa were done to confirm the villous pathology. Histopathological study of placenta was done and the slide were studied under the light microscope.

#### RESULTS

The study was done on 55 placentae of pregnancy induced hypertension in a tertiary care hospital. Total 70 placentae were studied out of which 15 were control study group. All the patients who were admitted in the maternity ward in the tertiary care centre were taken for the study, the patients were divided into 3 groups according to severity of toxemia as eclampsia 13 cases (23.6%), severe preeclampsia 15 cases (27.2%), and mild preeclampsia 27 cases (49%). Most of the cases (29) of PIH (52.8%) belonged to age group of 11 to 20 years and were primigravida (56.3%) while 26 cases (47.2%) belonged to age group of 21-30 years.

#### Table 1: Histological changes of placenta.

Microscopic examination	Degree of PIH								Control	
	Ι		II		III		IV		Control	
	No	%	No	%	No	%	No	%	No	%
Syncytial knots	13	100	9	60	12	45	34	62	3	20
Cytotrophoblasitc cell proliferation	10	76	10	66	18	65	38	69	2	13
Basement membrane thickening	10	76	10	66	18	67	31	56	2	13
VSMD	11	84	10	66	16	60	37	67	1	7
Fibrinoid necrosis	6	46	6	40	6	23	18	33	1	7
Calcification	8	61	3	20	9	33	20	36	1	7
Perivillous fibrin deposition	4	31	4	27	8	30	16	29	2	13
Infarction	7	53	7	47	3	11	17	31	-	-



Figure 1: Gross photograph cut surface of placenta showing infarcted area.

Out of 55 placentae of PIH most of the placentae i.e. 25 (45.4%) were of 12-14 cm in size while in normal patients, 9 placentae (60%) were of 18-20 cm. Thus, average size of placenta in PIH was smaller than the control group. Out of 55 cases of PIH placentae from 18 cases (32.7%) were in the range of 300-400 gms and 37 cases (67.2%) were in the range of 400-500gms. The weight of the babies was also reduced in PIH (60%)as compared to the control group (26.7%). Thus, average fetal weight of the fetus in pregnancy induced hypertension was found to be lower than control group. 11% feta deaths were noted in PIH.23% were seen in eclampsia and 20% in preeclampsia. No death was

observed in the control group. Thus, outcome of the fetus worsens with the severity of toxemia.

The commonest lesion observed macroscopically in placenta from PIH were infarction (31%) Figure 1, followed by perivillous fibrin deposition (29%) and basal decidual hematoma (5.4%).

Table 1 shows the major histologic changes of placenta as compared to the normal placentas. These include syncytial knots (62%) Figure 2, cytotrophoblasitc cell proliferation (69%), basement membrane (B.M.) thickening (56%), vasculosyncytial membrane deficiency (VSMD) (67%), fibrinoid necrosis (33%) Figure 3, calcification (36%), perivillous fibrin deposition (29%) and infarction (31%) Figure 4.



Figure 2: Microphotograph of increased syncytial knots (H and E 40x).



Figure 3: Microphotograph of fibrinoid necrosis of some villi (H and E 40x).



Figure 4: Microphotograph of infarcted villi as well as normal villi (H and E 10x).

All these changes were present in a lower number in the control group. Microscopic changes of placenta in PIH and normal cases.

### DISCUSSION

Placenta is the only vital organ in the prenatal life, which can be examined without any difficulty to the mother and the baby. The placenta provides a paradox as it is one of the most readily available organ for examination, yet one of the least known. Placenta being a fetal organ shares the same stress and strain to which the fetus is imposed. Thus, any disease process affecting the mother and the fetus also has a significant impact on the placenta. Placenta has been described as the mirror of perinatal mortality. Normally the placental morphology varies considerably during its short life span. The present study was undertaken to study the various morphological lesions of placenta in pregnancy induced hypertension.

The study was done on 55 placentae from PIH cases and they were divided into three groups according to the severity of toxaemia as done by Fox (1978)4, Kher et al and Sodhi et al.<sup>5,6</sup> Out of 55 cases of PIH, 13 had eclampsia (23.6%), 15 cases had severe preeclampsia (27.2%) and 27 cases had mild preeclampsia (49%).

Pregnancy induced hypertension was most frequent in the second decade of life (52.7%) and in primigravida which was similar to the findings of Cheslay et al, who also noted that 31 cases (56.3%) were primigravida and were teenagers.<sup>7</sup> Datta et al also stated that the incidence is more common in primigravida.<sup>8</sup> The placental size was between 14-16 cm (25.4%) as compared to normal placenta where the size was between 18-20 cm (60%). Thus, the average size of the placenta was reduced in PIH than normal. These findings are in accordance to Novak et al, who also observed smaller size of placenta which was due to reduced maternal blood flow.<sup>9</sup>

There was significant reduction in the weight of placenta in PIH as compared to the placenta in control group. This was like the observation made by Harshmohan et al and Dutta.<sup>8,10</sup> The weight of the fetus was also low in cases of PIH than that of control group which were like the findings of Masodkar et al and the number of fetal deaths were also more (11%) in PIH.<sup>11</sup> No death occurred in control group. These findings were like the findings made by Dutta et al.<sup>8</sup>

The commonest lesions observed macroscopically were infarction (31%) followed by perivillous fibrin deposition (29%) and basal decidual hematoma (5.4%). These findings were similar with the findings of Wentworth et al, but were low as compared to Salvatore et al.<sup>12,13</sup> No significant gross lesions were observed in control group. Infarction occurs due to thrombotic occlusion of maternal uteroplacental blood vessels and is seen in pregnancies complicated by PIH. PIH worsens uteroplacental

ischemia resulting in high incidence of fetal hypoxia, intrauterine growth retardation and fetal death Fox et al.<sup>4</sup>

In the study, major histopathological changes observed were syncytial knots, Cytotrophoblasitc cell proliferation, basement membrane thickening, vasculosyncytial membrane deficiency, fibrinoid necrosis, infarction, calcification and perivillous fibrin deposition. All these findings were comparable with the findings Kher et al and Sodhi et al.<sup>5,6</sup>

All these above findings are seen in PIH.PIH worsens the uteroplacental ischemia. The villous abnormalities such as cytotrophoblasitc hyperplasia and basement membrane thickening are due to diminution of uteroplacental blood flow.

The proliferation of villous cytotrophoblasitc cells is thought to be in response to chronic placental ischemia and these cells perhaps secrete basement membrane substrate leading to high basement membrane thickness. The decreased fetal perfusion in turn produces increased syncytial knotting.

The vasculosyncytial membrane represents sites of closest approximation of maternal and fetal circulation. Deficiency of vasculosuncytial membrane is attributed to abnormalities of maturation and differentiation. Fibrinoid necrosis is a consequence of reduced perfusion and ischemia.

Thus, most of the changes in PIH represent changes due to placental ischemia secondary to reduced maternal uteroplacental blood flow which are responsible for exerting ill effects on the fetus.

#### CONCLUSION

The fetus, placenta and mother constitute a triad of contributors to pregnancy outcome. Maternal disorders affect the placental histology and can be detected by morphological examination of such placentae. Placental examination becomes important as it will help in understanding the specific etiologies of adverse outcome which will need specific treatment and preventive measures for those at risk for recurrence in subsequent pregnancies specifically in preeclampsia and eclampsia cases.

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