

A Comparative Study of Placentas in Normal and Hypertensive Pregnancies

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Abstract: Placenta is an important vital organ upon which the intrauterine existence of foetus is dependent. Pregnancy complication like hypertension(PIH) is reflected in placenta and is the major cause of maternal & foetal mortality & morbidity. **Objectives:** 1) To study morphology and histology of placenta in normal and hypertensive pregnancies. 2) Comparison of two groups and correlation of their changes with the foetal outcome. **Material & Methods:** 100 placentas of patients both from normal & hypertensive group were taken for study from the labour room of Dept. of Obstetric & Gynaecology & operation theatre of Dr. PDMMH, Amravati, Maharashtra. Gross morphological features of placentas like size, surface area, weight and insertion of umbilical cord were noted. For histological studies, biopsies from each placenta of size 5 mm were taken. This was followed by fixation in 10% formal saline and further histological processing of the tissue was carried out. **Observations & Results:** The morphological parameters like weight, size, surface area, number of cotyledons were reduced and areas of infarction, retroplacental clot, calcification were more in hypertensive placenta than normal placenta. ($p < 0.005$) Similarly, the histological features like increased syncytial knots, intravillous and intervillous fibrin deposition, cytotrophoblastic proliferation, hyalinised villi, atherosclerosis were also observed in hypertensive placentas. The observed differences were statistically significant.

Keywords: PIH (Pregnancy Induced Hypertension), Preeclampsia, placenta, syncytial knots, fibrin, villi, cytotrophoblasts

1. Introduction

Reproduction is a feature exhibited by both unicellular and multicellular organisms 'Mother and foetus' are the two important ends of reproduction. The mere intrauterine existence of foetus is dependent on one vital organ the 'Placenta'. The hypertensive disorder in pregnancy also known as preeclampsia is a multisystem disorder having symptom complex of raised blood pressure, oedema and / or proteinuria⁽¹⁾ and affects 5-7% of all pregnancies worldwide.

Generally we study the hypertensive disorders in pregnancy as five types :

- 1) Gestational hypertension
- 2) Preeclampsia
- 3) Eclampsia
- 4) Preeclampsia superimposed on chronic hypertension
- 5) Chronic hypertension

In normal pregnancy the spiral arteries which are initially small muscular arteries dilate at the decidual end where they lose endothelium, smooth muscle and inner elastic lamina. Due to this, they lose the power to constrict with the response to the neural signals². In preeclampsia, the spiral arterioles are more thick walled, tortuous, narrow and they retain smooth muscle and elastic lamina. The reduced remodelling and reduced invasion of the trophoblasts is supposed to be the core factor in the development of intrauterine growth retardation and preeclampsia. The preeclampsia is associated with widespread apoptosis of these invasive cytotrophoblasts and some maternal cells in the uterine walls³. Increase in cytotrophoblastic cellular proliferation and syncytial knot formation adversely affects the weight, thickness, surface area and volume of the placenta⁴.

Vasculosyncytial membrane becomes deficient in the placentas of hypertensive pregnancies resulting in foetal hypoxia leading to perinatal morbidity and mortality⁵.

It is believed that the preeclampsia is associated with a generalized impairment of trophoblastic invasion which leads to maladaptation of uteroplacental arteries. This ultimately results into unfavorable outcome of the pregnancy in the form of many maternal as well as foetal complications thus increasing the maternal and perinatal morbidity and mortality.

2. Materials and Methods

The present study was conducted in 100 pregnant patients with normal blood pressure and 100 patients with pregnancy induced hypertension (PIH) after taking the written informed consent of the patient.

Inclusion Criteria

Control Group:

The patients having blood pressure systolic between 100-120 mm of Hg and diastolic between 70-90 mm of Hg.

Experimental Group

The patients having blood pressure systolic of 140 mm of Hg and above and diastolic BP of 100 mm of Hg and above.

Exclusion Criteria- The patients suffering from PIH along with any other disease were excluded.

Histological Techniques

For histological study, biopsies were taken as from the centre as well as from the peripheral part of each placenta

Fixation

Tissues of 5mm size were fixed in 10% formal saline for 7 days. The following steps were followed,

- Dehydration
- Clearing
- Wax Impregnation
- Section Cutting

The back surface of the block was slightly heated and then mounted on microtome chuck. 5-7 micron thick sections were cut.

Staining: Following stains were used for the present study:

Haematoxylin and Eosin (H&E Stain) & Masson's Trichrome Stain

3. Observations and results

Two sets having 100 placentas each were taken for the study.

Group I – Normal Pregnancy

Group II – Pregnancy with history of PIH (Pregnancy Induced Hypertension).

Observations – Gross Examination

- 1) When we studied the morphology of placenta, we recorded the size i.e. diameter (cm) and weight (gm) of placenta and we observed that in both the parameters, the values were lower in the hypertensive group as compared with the normal. With average diameter of 18.13 cm in normal and 16.23cm in hypertensive group.
- 2) Similarly weight of placenta was average 469.60gm for normal and 388.60gm for hypertensive patients. Both these parameters were having significant “p” value.
- 3) In normal patients 91% patients had central insertion of cord and eight% patients had eccentric insertion of cord and 1% only with marginal insertion. While in hypertensive patients there were 80% placenta with central insertion of cord, 2% with eccentric and 18% with marginal insertion.
- 4) It was found that mean number of calcified areas of placentas in PIH were more than those found in normal patients.
- 5) There was only one% patient with retroplacental clot in the normal group and in hypertensive group there were 14 % patients with retroplacental clot.



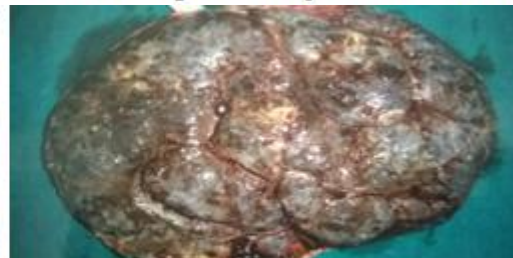
Small atrophic placenta of PIH



Placenta showing area of retroplacental hematoma



Cutsections of placenta with areas of infarction



PIH placenta showing areas of calcification

- 6) Only 4% patients in normal group had areas of infarction on placenta while there were 44% patients in hypertensive group who were having infarction on placenta.

Histological Findings

In control group of placentas, it was worth noting that the outer layer of the placental villi contained thinned out syncytiotrophoblastic lining. Their cytoplasm was distinctly basophilic. In T.S. there was thinning of syncytium with evident vasculosyncytial membrane. Intervillous fibrin deposit was seen and it was scanty. (photo4) The amount of collagen in the villi was variable and abundant in some areas and were well stained (blue) by Masson's Trichrome. Some of the villi showed intervillous fibrin deposits There was fibrosis of the villous stroma and sclerosis of the foetal vessels and the trapped villi appeared as isolated fibrous

blobs in a mass of fibrin and showed hyalinised type of villous stromal appearance. So there were increased number of avascular and hypovascular villi. Some of the villi were sclerosed with structural distortion and were referred to as 'Ghost villi'.

There were changes in the intimal or endothelial layer as degeneration and deposition of cholesterol laden macrophages (fatty infiltration). The change was called as atherosclerosis. Some villi showed abundant collagen deposition. The foetal blood vessels in these villi in hypertensive placentas showed thrombosis in lumen, fibrinoid infiltration in wall.

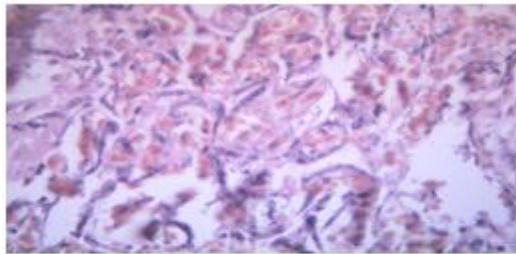
Syncytial knots mean in normal and hypertensive was 42.68 and 112.34 respectively.

Paucity of vasculosyncytial membrane was 16% in normal and 92% in hypertensive.

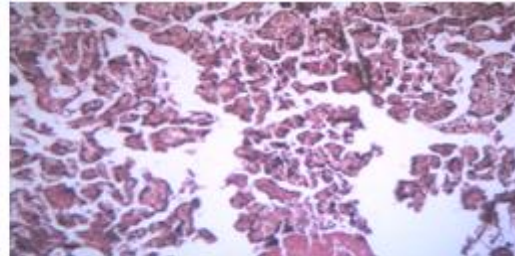
Excessive fibrin deposition 12% and 93% perivillous fibrin in normal and hypertensive.

Hyalinised villi 43% in hypertensive and 8% in normal. Some of the villi were sclerosed with structural distortion and were referred to as 'Ghost villi'.

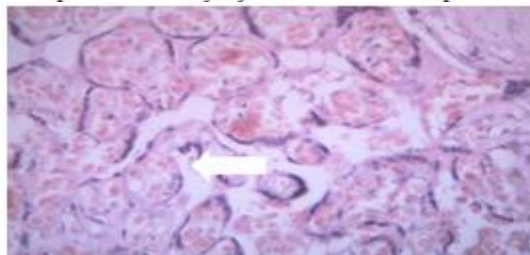
Atherosclerosis in 42% in hypertensive group with intimal or endothelial layer was observed as degeneration and deposition of macrophages.



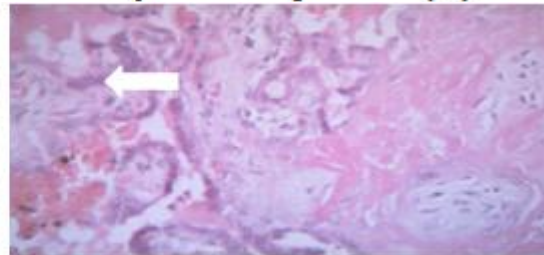
Hp view of few syncytial knots in normal placenta



10 X PIH placenta showing Excessive syncytial knots.



Normal Placenta Vasculosyncytial membrane



PIH Placenta absence of vasculosyncytial membrane

4. Discussion

Gross Findings

Figen Barut And Aikut Barut mentioned that placental weight in normal patients was 508 gm and 404gm in hypertensive patients⁶.

291gm in hypertensive patients and 573 gm in normal patients as was found by S. kotgiwar and N. Ambiyee⁷ and K. Devishankaran⁸.

It was 427 gm in mild PIH and 374 gm in severe PIH as was found by Vissiellekki kressie⁹

In our study, it was 388 gm for hypertensive patients and 469 gm for normal patients.

There were 3.5 % mean areas of infarction in normal patients while 17.4 % were mean areas of infarction in hypertensive patients as studied by Dr. Pradeep Londhe⁴. According to Thomson et al in 1969, birth weight of foetus and placental weight both were below average in preeclampsia but their ratios were slightly increased¹⁰.

There were 3.77 % 16.5% areas of infarction in normal patients and hypertensive patients respectively according to Mujumdar S. and H. Dasgupta¹¹.

In our study, 4% in normal patients and 44% in hypertensive patients were the areas of infarction. Fox in 1964 found 24.6% calcification in normal group¹². Our findings were nearly similar with the above findings. With significant "p value", Pushpa Goswami¹³ et al in 2012, found that mean number of calcified areas of placentas in PIH were more than those found in normal patients.

Histological Findings

According to study by H. Dasgupta, syncytial knots found in normal patients were 9 and in hypertensive patients were 27¹¹.

Acc to Pooja Dhabai, in normal patients were 6 and hypertensive patients were 23 almost three times syncytial knots¹⁴.

Acc to K. Devishankar⁸ and Sreechitrakarta¹⁵, there was 2 to 3 fold rise in syncytial knots in hypertensive as compared to normal patients.

Ambedkar Raj observed more than 30% syncytial knots in hypertensive than normal patients¹⁶. In our study, the syncytial knots were 42 in normal patients and 112 in hypertensive patients.

In our study, the intervillous fibrin deposition largely replaced degenerated trophoblastic lining.

Dasgupta H. found 33 % more areas of fibrinoid necrosis in hypertensive patients than in normal patients¹¹. Pooja Dhabai found 296 areas of fibrinoid necrosis in normal patients and 696 in hypertensive patients¹⁴.

Sreechitrakartha found areas of fibrinoid necrosis as 2% in normal patients and 19% in hypertensive patients¹⁵. Ambedkar Raj found 5% rise in such areas in every field¹⁶.

Maham Aklaq found such areas 40% in normal patients and 84% in hypertensive patients¹⁷. Zhang P. et al in 2006, observed the mean of 2.96 + -3.52 in normal and 6.26 + -2.8 in PIH which was significant¹⁸. Pandure and Ghosh

D.K. in 2011, reported 33.34% of placentas to have fibrinoid necrosis¹⁹.

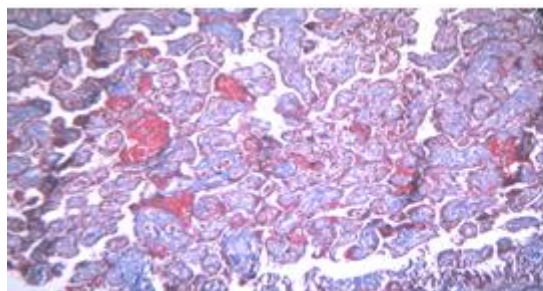
In our study, we got 12% areas of fibrinoid necrosis in normal patients and 93% in hypertensive.

When we studied decidual thrombosis, Sreechitrakartha found 4% in normal patients and 16% in hypertensive patients¹⁵. Maham Aklaq found 2% in normal and 14% in hypertensive patients¹⁷. Aparna Narsimha found 44% of placentas in hypertensive patients with decidual thrombosis and none were in normal patients²⁰. We also found 5% in normal patients and 37% placentas with decidual thrombosis in hypertensive patients.

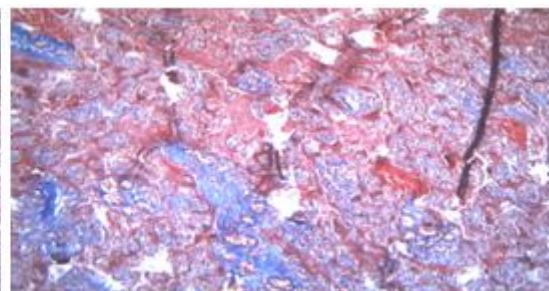
0.26 was hyallinised villi in normal patients and were in 5.7 hypertensive patients acc. to Pooja Dhabai¹⁴. Sreechitrakartha found 5% hyallinised villi in normal patients and 27 in hypertensive patients¹⁵.

Maham Aklaq found 6% hyallinised villi in normal patients while 40% in hypertensive patients¹⁷ and our findings were similar as 8% in normal patients and 43% in hypertensive patients. S. Kotgirwar and M. Ambiyee found that there was perivillous fibrin deposition in 16% of villi in PIH²¹. This perivillous fibrin deposition in intervillous space was the result of thrombosis of maternal blood as observed by Malik, et al and Mirchandani, et al²².

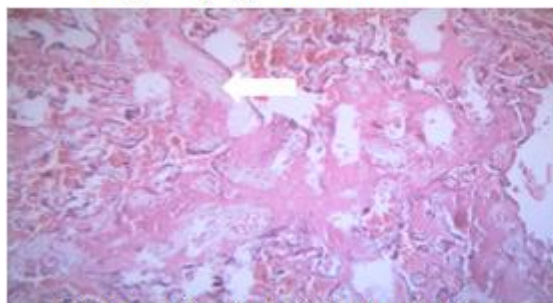
In our study, in cases of PIH placentas especially in cases of abruptio placenta (retroplacental clot) the blood vessels in the basal plate were congested and with infiltration of cytotrophoblasts. Reda Awadall Saleh 2008, observed the villi showing that endothelial degenerated atheromatous plaque formation were more common in PIH and were present in significantly more value²³.



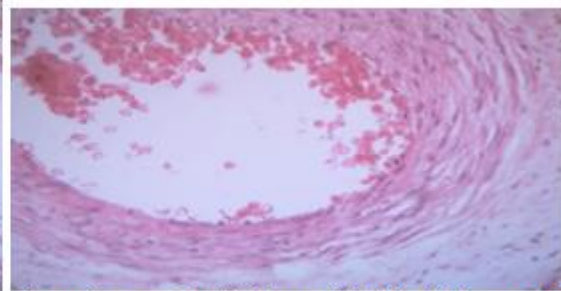
Normal placenta Masson Trichrome stain showing scanty deposition of fibrin



PIH placenta Increased amount of fibrin deposition



PIH placenta showing hyalinization of villi



atherosclerosis seen in decidual vessel Endothelial degeneration

5. Summary & Conclusion

We studied 100 placentas in normal and 100 placentas in hypertensive patients. Gross morphological examination of the placenta provided insights into the bad pregnancy outcome. Birth weight and placental morphology like weight, diameter, surface area, thickness, no of cotyledons and type of cord insertion were recorded. The weight, diameter, surface area and the thickness of the placenta in the control group were higher than that of the normal group. Retroplacental haematoma, areas of infarction and calcification were noted in more number in placentas with history of PIH than in normal group and in all these parameters observed difference was statistically highly significant.

In the histological study, of syncytial knots the observed difference between hypertensive and normal placenta was statistically highly significant. ($P < 0.0001$). There were significant variations in villous appearance in both the

groups. Cytotrophoblastic proliferation and X cells was a response to ischaemia to replace the damaged syncytiotrophoblast in ischaemic placentas. This was statistically highly significant. ($P < 0.0001$). Increased fibrin deposits were due to poor circulation in the intervillous space which was found statistically highly significant. ($P < 0.0001$). There were also increased avascular and hypovascular villi. Marked changes of atherosclerosis in decidual vessels and paucity of vasculosyncytial membrane were noted in PIH placentas.

These various histological changes leading to poor perfusion of villi was able to establish a relationship by morphological and by histological examination of placentas of patients with PIH with the adverse pregnancy outcome. Knowing that placenta is the mirror image providing a wealth of information on the antenatal events, it is very important to have examination of placenta adequately. This study may help to anticipate some prognosis for future pregnancy.

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