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, hyalinated villi, atherosis 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 loose endothelium, smooth muscle and inner elastic lamina. Due to this, they loose the power to constrict with the response to the neural signals ². 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 walls ³. Increase in cytotrophoblastic cellular proliferation and syncytial knot formation adversely affects the weight, thickness, surface area and volume of the placenta ⁴.

Vasculosyntial 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).

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 vasculosyncitial membrane was 16% in normal and 92% in hypertensive.

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

According to Thomson et al in 1969, placental weight both were below average in hypertensive patients.

In our study, there were 8% syncytial knots found in hypertensive patients while 17.4% in normal patients. 

There was a 427 gm in mild PIH and 374 gm in severe PIH as found by Vissiellekki kressie.

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 Londhe.

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 increased.

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

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 group. Our findings were nearly similar with the above findings. With significant p value, Pushpa Goswami 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 27.

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

Acc to K. Devishankar and Sreechitrakarta, 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 patients. 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 patients. Pooja Dhabai found 296 areas of fibrinoid necrosis in normal patients and 696 in hypertensive patients.

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

Maham Aklaq found such areas 40% in normal patiets and 84% in hypertensive patients. Zhang P. et al in 2006 observed the mean of 2.96 + .352 in normal and 6.26 + -2.87 in PIH which was significant.
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 patients. Maham Aklaq found 2% in normal and 14% in hypertensive patients. Aparna Narasimha found 44% of placentas in hypertensive patients with decidual thrombosis and none were in normal patients. We also found 5% in normal patients and 37% placentas with decidual thrombosis in hypertensive patients.

Sreechitrakartha found 5% hyallinised villi in normal patients and were in 5, 7 hypertensive patients according to Pooja Dhabai. Sreechitrakartha found 5% hyallinised villi in normal patients and 27 in hypertensive patients.

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. 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. Increased fibrin deposits were due to poor circulation in the intervillous space which was found statistically highly significant.

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

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

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

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