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Original Research Article

A Comparative Study on Placental Changes in Uncomplicated Pregnancies with Pre-Eclamptic Women

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Abstract
Context: *Preeclampsia is regarded as a risk factor in pregnancy and it leads to placental insufficiency. Placenta is a functional unit between mother and fetus. Thus preeclampsia causes both maternal and fetal morbidity and mortality. The present study intends to compare morphological and histological changes of placentae from uncomplicated pregnancies (the Control Group) and placentae from Pregnancy Induced Hypertension (the PIH group) .*
Study Design: *A prospective type of study.*
Place and period of study: *Department of Pathology, Anaesthesiology and Gynecology, S.S. Medical College, Gandhi Memorial Hospital, Rewa(M.P.) from 1st Dec 2015 to 30th June 2017.*
Materials: *200 cases were taken, 100 placentae from uncomplicated pregnancies (the Control Group) and 100 placentae from Pregnancy Induced Hypertension (the PIH group.*
Method: *Samples were grouped in two groups on the basis of presence or absence of Preeclampsia. All samples were studied morphologically and histologically.*
Result: *To summarise the results of this study there was statistically significant ($p<0.001$) decrease in weight, thickness and mean diameter of the placentae in the PIH group along with decreased mean birth weight of the baby in the PIH group as compared to the control group. There was increase in calcification and infarction in placentae of the PIH group. Histopathological examination of placentae in the PIH group showed increased syncytial knots, basement membrane thickening, fibrinoid necrosis and villus stromal fibrosis along with decrease in the vasculosyncytial membrane, as compared to the control group. The above mentioned microscopic findings were statistically significant ($p<0.001$).*
Conclusion: *In the present study all the morphological and histopathological variants of placenta are affected significantly in preeclampsia. From the findings in the present study, it is concluded that Pregnancy Induced Hypertension (PIH) had definite adverse influence on the histomorphology of placentae as compared to placentae of uncomplicated pregnancies. Usually antihypertensives are started when B.P. >160/110 mmhg, means from severe preeclampsia but early initiation of antihypertensive treatment will be helpful in improving the perinatal outcome.*
Keywords: *Placenta, Preeclampsia, Morphology, Histopathology.*

Introduction

Placenta is an unique and wonderful organ that arise de novo, directly related to the growth and development of the fetus in the uterus.[1] It does nutrient transfer, waste excretion, respiratory, endocrinal, barrier and immunologic function.[2,3] Pregnancy induced hypertension is associated with macroscopic and microscopic changes in the placenta. The maternal utero-placental blood flow is decreased in pre-eclampsia.[4,5] Maternal vasospasm leads to fetal hypoxia.[6,7] These changes are directly proportional to the severity of the disease and perinatal outcome become worse with advancing grades of pregnancy induced hypertension and utero-placental insufficiency[8,9] .

This is a prospective study to compare the histomorphological findings in placentae of pregnancy induced hypertension (PIH) and uncomplicated pregnancies.

Materials and Methods

Sample size was of 200, 100 placentae from uncomplicated pregnancies (the Control Group) and 100 placentae from Pregnancy Induced Hypertension (the PIH group).

Pregnant women with gestational age 28 to 40 week with B.P. less than 140/90 mmhg, no proteinuria or edema were included in control group.

Pregnant women with gestational age 28 to 40 week with B.P. more than 140/90 mmhg, with proteinuria more than 300 mg in 24 hour urine included in the study as cases of pregnancy induced hypertension. Cases with hypertension prior to this pregnancy, hypertension secondary to other causes, associated Cardiac diseases, Renal disorder, Diabetes mellitus, Multiple gestations, Anemia, Pregnancy ≤ 28 weeks of gestation, Postdated pregnancies and Placenta praevia / abnormalities of placental insertion were excluded from the study.

After delivery weight of newborn baby were recorded. The placental weight, diameter and thickness was assessed. Later the placental was cut into vertical strips (bread loaf manner) of 0.5 cms thickness and the gross lesions were re-examined. Evaluation of histological features as Syncytial knots, Vasculosyncytial membrane, Fibrinoid necrosis, villus stromal fibrosis and Basement membrane thickening were assessed

All the gross and histological features were compared between placentae obtained from Pregnancy Induced Hypertension (PIH) pregnancies with placentae from uncomplicated pregnancies which were control group.

Results

In this study we evaluated the histomorphological features in placentae obtained from Pregnancy Induced Hypertension (PIH) pregnancies and compared with placentae from uncomplicated pregnancies which were control group.

The gross morphometric parameters viz. foetal weight, placental diameter and placental thickness were reduced in placentae of the PIH group as compared to the controls group and were found to be statistically significant ($p<0.001$). In the control group both the mean birth weight of the baby and mean placental weight was 2819.02 gms & 480.50 gms, while in the PIH group was 2083.40 gms & 384.48 gms respectively. The mean diameter of placentae of the control group was 16.60±1.78 cms and that of the PIH group was 14.12±1.83 cms. The mean thickness of placentae of control group was 2.54±0.35 cms and that of PIH group was 2.15±0.39 cms.

Calcification (72%) and infarction (26%) was more common in the PIH group as compared to the control group which showed 23(46%) placentae with calcification and 0% placenta with >5% infarction.

Significant microscopic changes were seen as increased syncytial knots, fibrinoid necrosis, villous stromal fibrosis and basement membrane thickening along with decrease in the vasculosyncytial membrane were observed in placentae of PIH group and compared with the control group.

The mean syncytial knots in the PIH group were (37.42±13.84) more than the control group

(16.46±7.47). The difference between the two was statistically significant (p<0.001).

The mean vasculosyncytial membrane in the PIH group (4.96±2.41) was significantly reduced (p<0.001) as compared to the control group (22.62±5.13).

The mean number of villi showing increased stromal fibrosis, in the PIH group (11.6±4.12) was significantly higher (p<0.001) as compared to the control group (4.74±1.64).

The mean number of villi with increased basement membrane thickening, in the PIH group (13.14±6.55) was significantly higher (p<0.001) as compared to control group (1.36±1.14).

The mean number of villi showing fibrinoid necrosis, in the PIH group (13.96±3.48) was significantly higher (p<0.001) as compared to the control group (5.62±2.02).

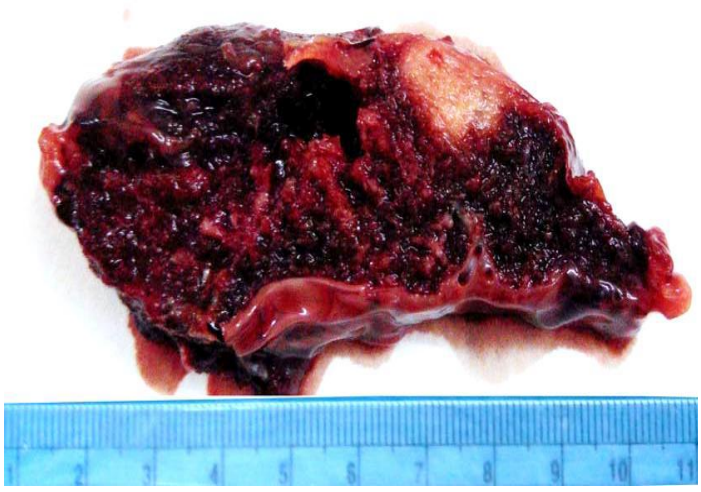

Photo.01 Photograph of Placenta Showing Calcification

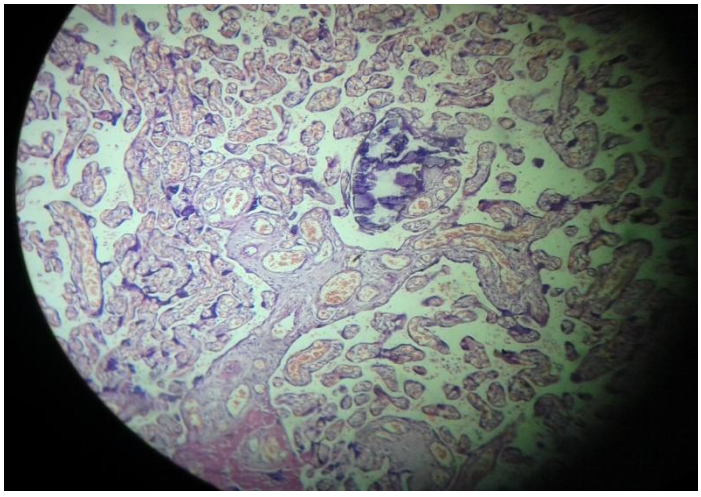

Photo.02 Microphotograph of Placenta Showing Calcification in PIH Group (H&E, 2X)

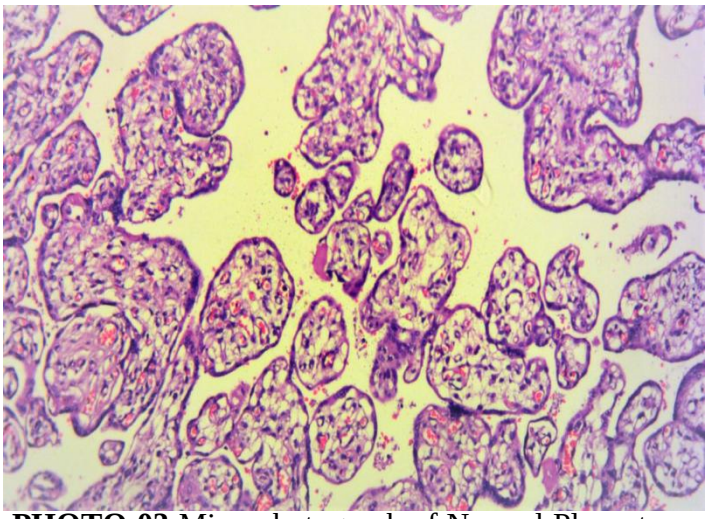

PHOTO 03 Microphotograph of Normal Placenta (H&E, 10X)

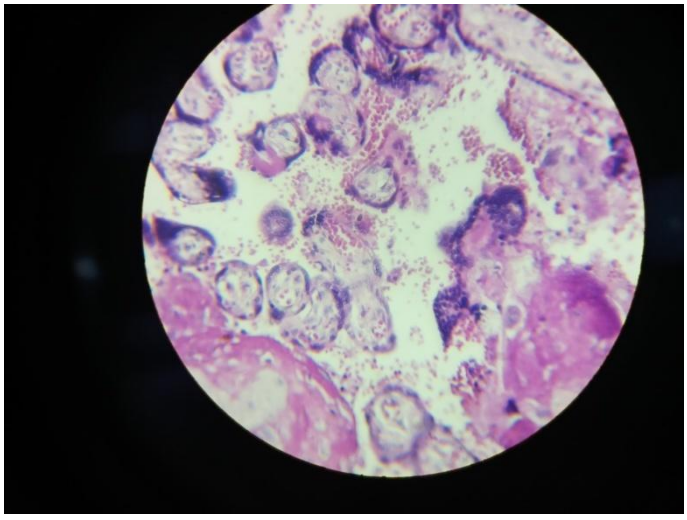

Photo 04 Microphotograph of Villi of Placenta of PIH Group. (H&E, 40x).

Note The Excessive Number of Syncytial Knots, Fibrinoid Necrosis, Stromal Fibrosis, Decrease In The Vasculosyncytial Membrane.

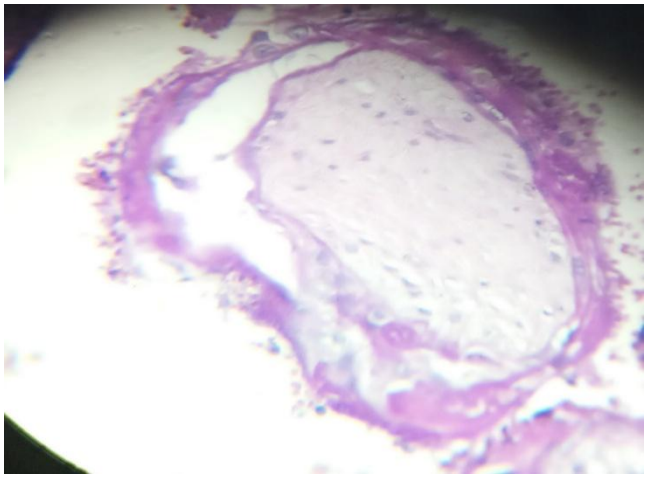

Photo 05 Photomicrograph of Villi of Placenta of PIH Group Showing Basement Membrane Thickening (H&E, 40X).

Discussion

Placenta being a fetal organ shares the same stress and strain, to which the fetus is exposed. It forms the morphological record of anatomical condition, intrauterine events and intrapartum events of the gestation.[10] Pregnancies complicated by PE are reflected in the placenta both macroscopically and microscopically. Although the placenta adapts well to the hypoxic condition in PE, the compensatory changes that occur are insufficient. These compensatory changes cause mal-development and inadequate placental mass, causing placental dysfunction that leads to oxidative stress and chronic fetal hypoxemia[11]. In preeclampsia, placenta tends to be smaller as compared to uncomplicated pregnancy[5]. Blood flow to placenta is reduced in hypertensive disorder of pregnancy and result in unduly small fetus. Moreover due to hypoxia placenta weight is also reduced. In the present study comparing preeclamptic placentas to control placentas, the mean placental weight, thickness and diameter were decreased and were found to be more significant. The placental weight is an important determinant of both birth weight and fetal growth. It is functionally significant as it is related to villous area and fetal metabolism.[12] The mean birth weight of the baby in control group was 2819.02 gms, whereas in the PIH group was 2083.40 gms. The mean weight of the placenta in the control group was 480 gms, whereas in the PIH group was 384.48 gms. Similar findings were statistically significant and simillar to others[13,14]. In our study, the mean diameter and mean thickness of placentae in the control group was 16.60 cms and 2.54 cms, whereas in the PIH group was 14.12 and 2.15 cms and mean thickness and mean diameter in the PIH group was significantly reduced ($p<0.001$), as compared to the control group which is in concordance with others[15]. The cause is uteroplacental ischemia leading to the inadequate maternal supply of oxygen and nutrients but also to placental ischemic damage.[16]

The calcification indicates an 'ageing' of the placenta that occur near the end of pregnancy but it may be a sign of premature ageing in the cases of PIH.[17] In our study, calcification was seen in 46% placentae of the control group, whereas in the PIH group, it was seen in 72% placentae. Similar findings were recorded by others[12]. In our study, placenta with more than 5% area of infarct was 0% in the control group, whereas 26% in the PIH group which is in concordance with others[12]. Thus, the present study reaffirms the findings in the above studies that only the placentae of the PIH group show >5% area of infarction.

The microscopic lesions were assessed as percentage of placenta in PIH and control group showing more than 30% of syncytial knots, villi with <5% of vasculosyncytial membrane, >3% of villi with fibrinoid necrosis, stromal fibrosis and basement membrane thickening more than 3% of villi as supported by most literature.

The mode of formation and function of syncytial knot has been considered as a degenerative process and ageing change.[18]The placental villous membrane (syncytioplasm) and the fetal capillaries remain separate but act as a single unit, called vasculosyncytial membrane.[19,21] The two factors responsible for the formation of stromal fibrosis are a normal aging process and a reduced uteroplacental blood flow[1]. Fibrinoid necrosis may be a manifestation of endothelial damage in placenta which may lead to increased coagulation tendencies.[20,21] The basement membrane thickening is the byproduct of cytotrophoblast cell hyperplasia, as the basement membrane protein is secreted by these cells[1] . Hence, cytotrophoblastic proliferation is seen as basement membrane thickening in placental ischemia of the PIH group[1,21].

In the present study, >30% of syncytial knots were seen in, 2% placenta of the control group and 66% placenta of the PIH group and similar findings are there in others[21] which is statistically significant increase ($p<0.001$).

In the present study villi showing ≤5% of vasculosyncytial membrane in the control group

was 0%, whereas in the PIH group was 84% which is statistically significant decrease ($p<0.001$) and in concordance with others[21].

Villous stromal fibrosis of >3% villi were seen in, 82% placentae of control group and 96% placentae of the PIH group in the study which is statistically significant increase ($p<0.001$). Similar findings were recorded by others[1,21].

In the present study, fibrinoid necrosis of >3% of villi was seen in 82% placentae of the control group, whereas 100% in the PIH group. These findings were statistically significant ($p<0.001$) and in concordance with other workers[21].

>3% of basement membrane thickening was seen in 4% of the control group and 82% of the PIH group in this study. Similar findings were recorded by others[1,21] which is statistically significant increase ($p<0.001$).

Conclusion

The intrauterine existence of foetus depends on one vital organ, the "placenta". Placenta maintains an accurate record of infant's prenatal growth. Placenta being a fetal organ shares the same stress and strain to which the fetus is exposed. Any disease process like Pregnancy Induced Hypertension (PIH) affecting the mother also has a great impact on placenta and fetus. The histomorphological feature of placenta is important determinant of both baby birth weight and fetal growth.

In the present study all the morphological and histopathological variants of placenta are affected significantly in preeclampsia. The results of this study there was statistically significant ($p<0.001$) decreased mean weight of the placentae in the PIH group along with decrease in thickness, mean diameter and mean birth weight of the baby in the PIH group as compared to the control group. There was increase in calcification and infarction in placentae of the PIH group. Histopathological examination of placentae in the PIH group showed increased syncytial knots, basement membrane thickening, fibrinoid necrosis and villus stromal fibrosis along with decrease in the vasculosyncytial membrane, as compared to the control group. The above mentioned microscopic findings were statistically significant ($p<0.001$).

From the findings in the present study, it is concluded that Pregnancy Induced Hypertension (PIH) had definite adverse influence on the histomorphology of placentae as compared to placentae of uncomplicated pregnancies. Usually antihypertensives are started when B.P. >160/110 mmhg, means from severe preeclampsia but early initiation of antihypertensive treatment will be helpful in improving the perinatal outcome.

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