Original Article

A comparative histological study of human placentae obtained from mothers with PIH and mothers with normal blood pressure

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Abstract

The present study was performed to evaluate the histological changes in placentae obtained from patients with history of pregnancy induced hypertension (PIH) and compare them with placentae obtained from patients who had normal blood pressure in pregnancy. 100 placentae were obtained from the labour room and operating theatres of Sanjay Gandhi Memorial Hospital, Rewa. 50 placentae were from patients who had pregnancy induced hypertension (study group) and 50 were from normotensive pregnant women (control group). On comparison of histological examination of placentae obtained from hypertensive mothers with normal placentae, it was noted that in the former there was three times more incidence of syncytial knot areas, two times more incidence of fibrinoid necrosis, and cytotrophoblastic proliferation. Presence of areas of hyalinzed villi was six times more common in study group. The difference in histological examination of placenta was more significant if the patients had hypertension ≥ 140/100 mm Hg. This also correlated with the birth weight of babies born. The mean weight of babies born to hypertensive mothers was 1.97 kg while it was 2.68 kg in normotensive mothers. The study has shown that in moderate to severe hypertension there are a number of histological changes in the placenta which may be responsible for the occurrence of complications associated with it.
Keywords: Pregnancy induced hypertension, placenta, histology.

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INTRODUCTION

The placenta is a vital organ for maintaining pregnancy and promoting normal foetal development[1]Survival and growth of foetus is essentially dependent on formation, full development and functions of the placenta[2]. The examination of the placenta in utero as well as postpartum, gives valuable information about the state of the foetal well being[3]. Careful examination of placenta can give information which can be useful in the management of complications in mother and the newborn. The Maternal mortality rate in India has declined from 570 in 1990 to 230 per one lakh population in 2008[4]. But, still it remains high in comparison to developed countries and is largely preventable. The hypertensive disorders are responsible for 5-8 % of all maternal deaths[5]. Pregnancy complications like hypertension or gestational diabetes are reflected macroscopically and microscopically in the placenta[6,7,8,9.] In pregnancy induced hypertension, there is increased resistance to utero-placental circulation which adversely affects the growth of placenta[10]. These abnormalities ultimately result in unfavourable outcome of pregnancy with reduction of fetal weight. Hence, this study was done to find and correlate the histopathological parameters associated with pregnancy induced hypertension.

MATERIAL AND METHODS

The present study of placenta with its clinical significance was conducted in the Department of Anatomy, Shyam

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Shah Medical College, Rewa, (M.P.). For present study, 100 placentae were studied 50 cases belonged to pregnancy induced hypertension (PIH) and 50 cases belonged to normal pregnancy (Control Group). Placentae were collected from labour room and gynaecology operation theatre of Sanjay Gandhi Memorial Hospital, Rewa. Each patient taken for study was initially examined by an obstetrician in antenatal clinic of the hospital. A total of 100 cases were studied. In pregnancy induced hypertension, only those cases having blood pressure 140/90mm of Hg or above, with or without oedema, and/or proteinuria were included[11]. Placenta with it's cord and membranes were collected immediately after delivery and were preserved in 10% formalin solution. Weight of the foetus and placenta were noted Gross examination of placentae for presence of infarction, calcification andthrombosis was done. For histological study, tissue of size 2x2mm were taken near the attachment of umbilical cord, margin and center of the placentae and processed for light microscopic studied. Slides were stained with haematoxylin and eosin (H and E). On histological examination, Syncytiotrophoblastic knot, cytotrophoblast proliferation, hyalinized villi, fibrinoid necrosis, stromal fibrosis, medial coat proliferation, and calcification were noted.

OBSERVATION AND RESULTS

In the present study, mean birth weight of the new born baby was 400.70 gm in the study group and493.8 gm in the control group. On light microscopic examination of placental villi showed significant changes in the study group. The mean number of areas of syncytial knots in pregnancy induced hypertensive group (28.57±9.56) was significantly increased than in control group (9.16±4.08). The mean number of areas of cytotrphoblastic proliferation in study group (19.07±6.77) was significantly increased than in control group (7.74±4.00). The mean number of areas of fibrinoid necrosis and hyalinised villi in pregnancy induced hypertensive group (2.8±1.26 and13.30±5.70) was significantly increased than in control group (1.46±5.33and3.02±1.67). The various observations and results are tabulated and depicted as below

Table 1: Study of placental villi in control and P.I.H. Cases

Sr. No	Parameter of placental villi (randomely selected area) in LPF	Control A n=50	PIH B n=50	Statistical significance
1	Mean no. of areas of syncytial knot formation	9.16±4.08	28.57±9.56	Significant
2	Mean no. of areas of cytotrophoblastic proliferation	7.74±4.00	19.07±6.77	Significant
3	Mean no. of fibrinoid necrosis	1.46±5.33	2.8±1.26	Insignificant
4	Mean no. of areas of hyalinized villi	3.02±1.67	13.30±5.70	Significant

Statistical analysis done by unpaired Student's 't' test, ns = not significant, */*** = significant

Table 2: Study of stromal pathology in control and P.I.H. Group

Sr. No.	Parameters of placental pathology p>0.01	Control Group	Hypertensive Group	Statistical Significance (per LPF)
1	Mean no. of areas of stromal fibrosis /LPF	0.58±0.70	7.04±3.70	Significant
2	Mean no. of areas of medial coat proliferation of medium sized blood Vs /LPF	1.93±0.70	8.61±3.81	Significant
3	Mean no. of calcified areas /LPF	4.96±2.84	11.71±5.57	Significant
4	Mean no. of hyalinized areas/LPF	0.94±1.78	9.71±4.86	Significant

Statistical analysis done by unpaired Student's 't' test, ns = not significant, */*** = significant.

Table 3: Study of baby weight, placental weight and umblical cord insertion between the two groups

Sr. No.	Placental Parameters	Control Group A	PIH Group B	Statistical Significance
1	Mean birth weight of babies in Kg	2.684 Kg	1.9764 Kg	Significant
2	Mean placenta weight in grams	493.8 gm	400 gm	Significant
3	Marginal insertion of umbilical cord (%)	26%	74%	Significant

Statistical analysis done by unpaired Student's 't' test, ns = not significant, */*** = significant

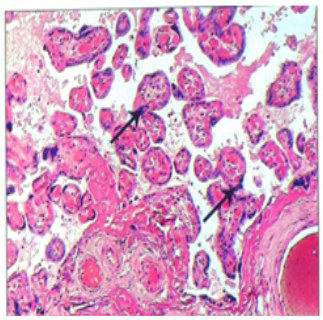
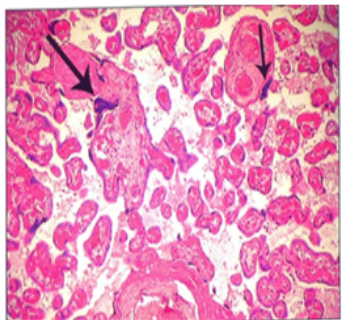


Figure 1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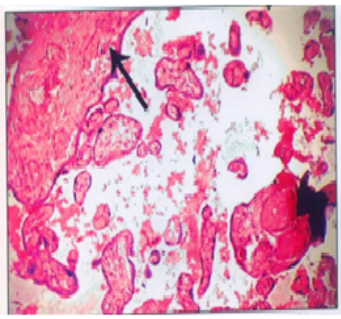
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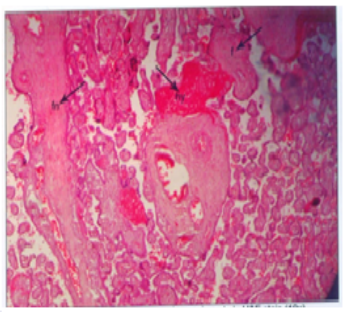
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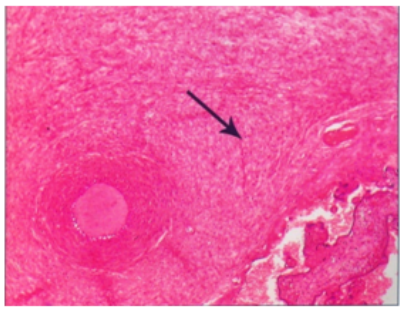
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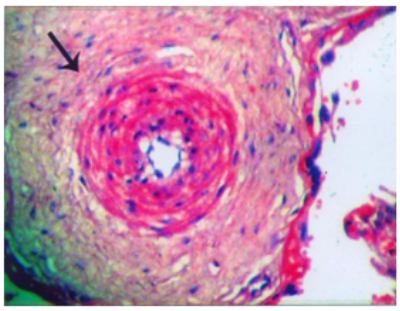
Figure 6

Legend

Figure 1: Photomicrograph of term placenta in H&E stain (10x) arrows shwoing syncytial knot.
Figure 2: Photomicrograph of term placenta in H & E stain (10x) arrowed arrows showing cytotrophoblast profliferation.
Figure 3: Photomicrograph of term plcenta in H & E stain (10x) arrow showing fibrinoid necrosis
Figure 4: Photomicrograph of term placenta in H & E stain (10 x) arrows showing hyalinised villi with fibrosis & fibrinoid necrosis.
Figure 5: Photomicrograph of term placenta arrows showing in H and E stain (10x) arrows showing
Figure 6: Photomicrograph of term placenta in HandE stain (10x) Stromal fibrosis Medial coat proliferation.

DISCUSSION

Placenta, is the most important link between the developing foetus and mother its histomorphometric characteristics has been widely studied in normal and diseased mothers. Much literature exist regarding the condition of placenta in hypertensive and normotensive mothers, so the present study was undertaken to analyze the histopathological changes in pregnancy induced hypertensive placentae with a view to assess the significance of villous abnormality. Maternal vasospasm leads to decreased utero-placental blood flow is proposed by Browne and Veall[11]. Damania, et[12] al found that as hypertension increases histopathological changes also increases. Rath[13] (1994) stated that the low birth weight of babies in hypertensive mother was due to alteration in the arrangement of the intracotyledons. Fox (1967)[14] and Udania *et al* (2004)[15] had also observed increase in incidence of placental infarction with the severity of toxaemia. Villous maturity can be indicated by syncytial knot, so increased in syncytial knots are associated with reduction in foetal perfusion Genset (1992)[16.] Fibrinoid necrosisis significantly higher in pregnancy induced hypertensive placentae and it is due increased in coagulative changes in endothelial damage. Increased incidence of Stromal fibrosis may be due to obliterative endarteritis which corresponds with the study of Masodkar AR and Narshimha *et al*[17,18]. In our study as well as in other study it was found that vascularity was decreased in pregnancy induced hypertensive placentae which leads to degenerative changes like calcification. Cytotrphoblastic proliferation was significantly higher in pregnancy induced hypertensive placentae leads to thickening of basement membrane. Most of the histopathological changes in placenta was due to hypoxia, which is responsible for maternal foetal mortality and morbidity.

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