ORIGINAL RESEARCH PAPER

Anatomy

MORPHOLOGICAL AND HISTOPATHOLOGICAL CHANGES OF PLACENTA IN PREGNANCY INDUCED HYPERTENSION

KEY WORDS: Placenta, Histopathological Changes, Pre-eclampsia, Eclampsia.

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ABSTRACT

Introduction: In pregnancy induced hypertension (PIH), pathological changes in the placenta occurs which may result in reduced blood flow across placenta. The present study was done to assess the morphology & histopathological changes of placenta from preeclamptic mothers and to correlate their findings with those of normal pregnancies. **Materials and Methods:** The present study was a case-control study conducted during 2023-24 in ESIC Medical College and Hospital, Faridabad, Haryana in Insured persons under ESIC scheme. Placentae of normal pregnancies & those complicated by pre-eclampsia/ eclampsia with period of gestation 36-40 weeks were included. 30 placentae from mother having PIH and 30 placentae from normal pregnancies were examined for gross morphology and histopathological changes. **Results:** The mean weight of placentae was (410 ± 60 grams) lower among placentae of hypertensive pregnancies in comparison to placentae of normal pregnancies (478 ± 38 grams). This difference was statistically significant. (p 0.001). There was significant association between presence of gross infarction, calcification, Hyalinized area, fibrinoid necrosis, Syncytial Knot formation, medial coat proliferation, intervillous hemorrhage, decreased villous vascularity in PIH and control cases. **Conclusion:** In present study, mean placental weight in hypertensive pregnancies was lower than mean placental weight in normal pregnancies. It also revealed lesions like increased syncytial knots, cytotrophoblast cell proliferation, villous stromal fibrosis and fibrinoid necrosis in placenta from preeclampsia cases.

INTRODUCTION:

The placenta is an organ responsible for circulation, respiration and nutrition from mother to fetus. Information on placental size, shape, consistency, presence of accessory lobes, placental infarcts, hemorrhage are important factor for normal growth and development of fetus[1,2].

The mature placenta is disklike in shape, 3 cm thick, and about 20 cm in diameter. A typical placenta weighs about 500 g. The fetal side of the placenta is shiny because of the apposed amniotic membrane. The maternal side of the placenta is subdivided into as many as 35 lobes. The grooves between lobes are occupied by placental septa, which arise from the decidua basalis and extend toward the basal plate. Within a placental lobe are several cotyledons, each of which consists of a main stem villus and all its branches. The intervillous space in each lobe represents a nearly isolated compartment of the maternal circulation to the placenta.

In normal pregnancy the spiral arteries invade the trophoblast layer, where there is drastic structural change in the endothelium, smooth muscle and inner elastic lamina.

In hypertensive pregnancies, the spiral arteries remodeling is prevented[5]. Hypertensive disorders of pregnancy are a leading global cause of maternal, fetal, and neonatal morbidity and mortality. Preeclampsia/eclampsia is a multiorgan spectrum of diseases characterized by onset of hypertension (systolic ≥140 mmHg and diastolic ≥90 mmHg) after 20-week gestation and significant proteinuria (>0.3 g per 24 h or ≥2+ proteinuria, detected by urine dipstick).

It is believed that the preeclampsia is associated with a generalized impairment of trophoblastic invasion which leads to maladaptation of uteroplacental spiral arteries. The reduced invasion of the trophoblasts is supposed to be the core factor in intrauterine growth retardation. Hypoxia and reduction in blood flow could be responsible for morphological alterations of placenta in preeclampsia[10].

Placenta tries to compensate for reduced supply by regulatory patho-physiologic mechanism however these compensatory changes are insufficient and thus fails to develop adequate placental mass.

Hypertensive disorders in pregnancy, and in particular preeclampsia (PE) and eclampsia, are main causes of maternal mortality and morbidity globally, and an important cause of fetal and perinatal complications, such as increased risk of stillbirth, neonatal death, intrauterine growth retardation (IUGR), and preterm delivery[4].

MATERIAL AND METHODS:

The present study was done in ESIC Medical College and Hospital, Faridabad. All the subjects were insured person (IP's) covered under ESIC scheme.

Study material included 60 Placentae (30 samples were collected from normal deliveries and 30 samples were collected from cases having pregnancy induced hypertension, BP > 140/90 mmHg) from normal deliveries, caesarean sections or abortion of the age group 20-40 years and gestational age ranging from 36-40 weeks. Informed and written consent was taken.

Placentae from mother suffering from any disease like Cardiovascular, Diabetes, Respiratory and history of any previous genetic disorder, cases with gestational age less than 36 weeks and over 40 weeks, age of mother less than 20 years and more than 40 years, family monthly income more than Rs 21000 per month were not included.

Specimen were taken from maternal surface of placenta near centre specimen were be put in 10% formalin for gross morphology and were stained with H&E for microscopic study.

RESULTS:

The present study was performed on 30 placentae of women

with PIH and 30 placentae of normal pregnancy

Table 1: Maternal age group and gestational age wise distribution of study subjects

Age group	Control (n=30)		PIH (n=30)		Total (n=60)	
	No.	%	No.	%	No.	%
21-25	16	53.3	11	36.7	27	45
26-30	11	36.7	9	30	20	33.3
31-35	3	10	10	33.3	13	21.7
Total	30	100	30	100	60	100
Gestational age (weeks)	Control (n=30)		PIH (n=30)		Total (n=60)	
	No.	%	No.	%	No.	%
37	8	26.7	13	43.3	21	35
38	7	23.3	9	30	16	26.7
39	10	33.3	6	20	16	26.7
40	5	16.7	2	6.7	7	11.6
Total	30	100	30	100	60	100

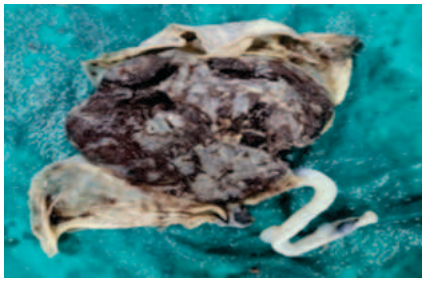

Fig. 1: Gross picture of PIH placenta showing hemorrhage and necrosis

Table 2: Comparison of Placental morphological and microscopic features in study group

Placental Morphology	Control (n=30)		PIH (n=30)		P value
	No.	%	No.	%	
Mean placental weight (gms)	478		410		0.001
Infarction (gross)	2	6.6	22	73.3	0.001
Calcification (gross)	09	30	24	80	0.003
Hyalinised area /100 magnification	4	13.3	12	40	0.003
Medial coat proliferation/100 magnification	3	10	26	86.6	0.001
Intervillous hemorrhage	6	20	20	66.6	0.003
Decreased Villous Vascularity	2	0	20	66.6	0.002

On analyzing gross and microscopic features of the placentae, there was significant association between presence of gross infarction, calcification among control group and cases. Gross and microscopic features also showed significant differences. (Table 2) Figure 2

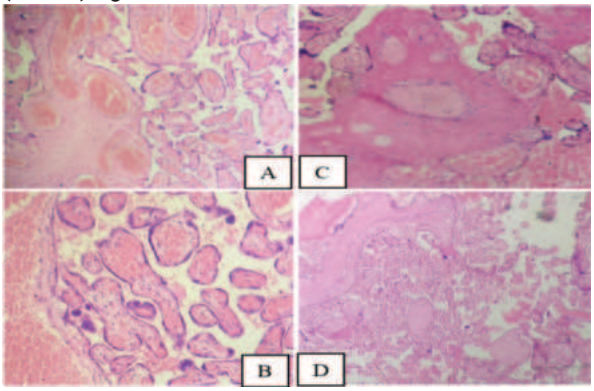

Fig. 2: Photomicrograph showing; A). Intervillous hemorrhage (H & E, 100X); B). Vascular medial coat proliferation (H & E, 100X); C). Calcification (H & E, 40X); D). Areas of infarction (H & E, 100X).

Comparison of microscopic features of placenta among the PIH and control group were as shown in Table 3 and Table 4

Table 3: Microscopic features of placental villi among normal and PIH cases:

Microscopic features	Control (n=30)		PIH (n=30)	
	No.	%	No.	%
No. of Syncytial Knots formation per 100 villi				
0-30	20	66.7	0	0
31-60	09	30	7	23.3
61-90	01	3.3	20	66.7
91-120	00	00	03	10
No. of areas of fibrinoid necrosis / 100 villi				
01-05	25	83.4	0	0
06-10	3	10	16	53.4
11-15	1	3.3	9	30
16-20	1	3.3	4	13.3
21-25	0	0	1	3.3
No. of areas of cytotrophoblastic proliferation				
01-05	22	73.3	0	0
06-10	5	16.7	6	20
11-15	3	10	10	33.3
16-20	0	0	12	40
21-25	0	0	2	6.7
No. of calcified areas/100 magnification				
0	14	46.7	4	13.3
1	11	36.7	5	16.7
2	4	13.3	9	30
3	1	3.3	7	23.3
4	0	0	3	10
5	0	0	2	6.7

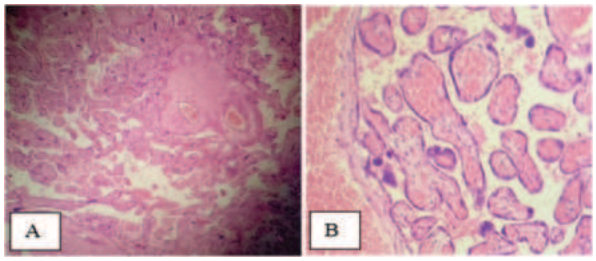

Fig. 3: Photomicrograph showing; A). Decreased villous vascularity in PIH (H & E, 100X); B). Syncitial knots in pre-eclamptic placenta (H & E, 40X)

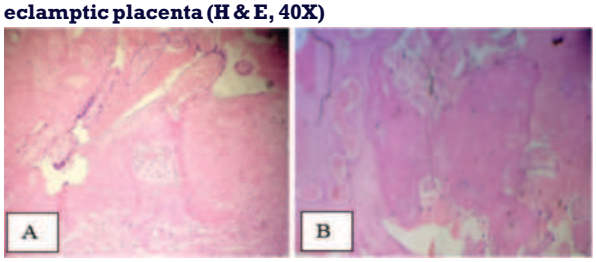

Fig. 4: Photomicrograph showing; A). Extensive fibrinoid necrosis (arrow) (H & E, 100X); B). Fibrinoid necrosis involving entire villous. (H & E, 40X)

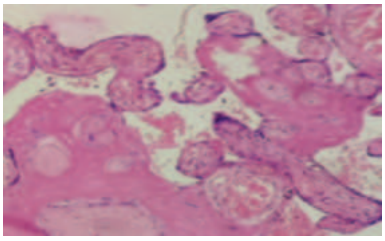

Fig. 5: Photomicrograph of intervillous fibrin deposition. (Magenta) (PAS, 40X)

Table 4: Comparison of parameters of placental findings among PIH and normal cases (n=60)

Placental parameter	Group	n	Mean	Std. deviation	P value
Syncitial knots /100 villi	Control	30	26.2	7.28594	0.001
	PIH	30	73.4253	14.2359	
Fibrinoid necrosis /100 villi	Control	30	3.5222	1.14596	0.003
	PIH	30	14.3333	5.39697	
Hyalinized villi / 100 magnification	Control	30	2	1.14686	0.0001
	PIH	30	5.9333	2.90425	
Cytotrophoblastic Proliferation /100 villi	Control	30	4.6667	3.27753	0.003
	PIH	30	18.2	4.39019	
Calcified areas / 100 magnification	Control	30	0.37	0.549	0.001
	PIH	30	2.8	1.442	

DISCUSSION:

In the present study, average weight of placentae was higher 478gms in control group compared to PIH group 410gms. This difference was found statistically significant. (p value 0.0001). In the study conducted by S Kishwara et al,[11] Boyd and Scott[12] (1985), Mayhew et al. (2003)[13] Barton et al. (2001)[14] observed that the mean weight of the placentae was significantly lower in the group with preeclampsia than that of control group. Odegard et al. (2000)[15]found that preeclampsia was associated with a 5% reduction in birth weight.

In present study, infarction was present in 73.3% placentae of hypertensive pregnancies. with significant P value (0.001) in control group and PIH group (Fig. 1, 2). Underlying pathophysiology seems to be defective remodelling of the spiral arteries, known as decidual arteriopathy, that could contribute to a hypoxic environment and thereby, placental insufficiency which is often found in pregnancies with PIH[8]. An incidence of mean infarct areas of 9.6 (19%) was found on the maternal surface of placenta in PIH cases in study conducted by Tallapalli Lakshmi Sri Gowri et al[8]. and Wentworth et al,[19].

Syncitial knots are focal clumps of syncitial nuclei that protrude into the intervillous space from the surface of the villi. Formation of knots on more than a third of villi is considered excessive.

In present study, Mean number of syncitial knots was 73.4 ± 14.2 in PIH group while it was 26.2 ± 7.2 in control group which was found significant with P value 0.001 (Table 5) Fig. 3. Syncitial knots formation was found to increased by 66.7% in PIH cases. It was higher in PIH group compared to control group. S. Rajyalakshami et al[7] found in their study that 4 out of 6 (66.6%) cases of mild, 9 cases (56.2%) of moderate, 23 cases (82.1%) of severe pregnancy induced hypertension showed increased prominent syncitial knots. Similar results have been observed by Pasricha et al,[20] Dhabhai et al[21] and Nafees et al.[22] They found higher number of syncytial knots in preeclamptic placenta as compared to control placenta. Narasimha et al[23] and Tomas et al[24] have also reported the similar findings.

Fibrin has immunohistochemical features of blood clot product and accumulates in maternal vascular under perfusion associated with decidual vasculopathy and the syndrome of preeclampsia[7]. The fact that the intervillous space is involved would seem to implicate a maternal circulatory abnormality. The deposition of fibrinoid is diffuse without preferential basal concentration, although there may be patchy, sometimes extensive involvement of the maternal floor as part of the process (Fig.5).

In present study, the fibrinoid necrosis was found 14.3+5.3 in PIH cases and 3.5+1.1 in control cases which is significant with P value 0.003. Similar results have been found in studies conducted by S. Rajyalakshmi et al[7] and Narsimha et al[23].

Microscopically, the villi are widely separated by pink amorphous matrix-type fibrinoid containing numerous mononuclear trophoblast cells. There was statistically significant association between presence of hyalinized area in placentae of PIH cases and normal pregnancies (p value 0.0001). (Fig. 4,5) Various workers like Motwani et al[25] have previously demonstrated hyalinized villi in preeclamptic placenta.

In present study, the mean number of cytotrophoblastic proliferation was 18.2 ± 4.39 in PIH group while it was 4.6± 3.27 in control group which has been increased by 33.3% in hypertensive pregnancies. Saumya et al[9], Narasimha et al[23] Arnholdt et al[26] Motwani et al[25] also found similar results showing significant increase in number of cytotrophoblast cells in preeclamptic placenta.

In present study, we found calcification in 70% cases of PIH which is highly significant with P value of 0.001. The calcium deposits were mostly observed in the villi and basement membrane of the villi, strongly suggestive of uteroplacental insufficiency because of narrow lumen[7]. S. Rajyalakshami et al[7], found calcification in 31.2% and 42.8% of moderate and severe PIH cases in their study. The incidence of calcification was 26.9% and 60.5% in studies conducted by Narshimha et al[23] and Mohan SR et al[31].

There was statistically significant association between PIH and decreased villous vascularity. (Fig. 3). In present study, the villous vascularity was decreased by 66.6% (p value 0.002). Majumdar Set al,[16] Narsimha et al[23], Motwani et al[25] reported reduced villous vascularity in preeclamptic placenta as compared to control placenta. Saleh et al[27] observed that villi of preeclamptic placenta showed regression of villous capillaries upto complete disappearance.

CONCLUSION

The present study was conducted on 60 placentae (30 form Normal pregnancies and 30 from Hypertensive pregnancies) on Insured person under ESIC scheme.

From the present study, it can be concluded that the hypertensive disorders of pregnancy adversely influence the morphology of the placenta. Gross morphological examination of the placenta provided insights into the bad pregnancy outcome. The histopathological changes observed in the placentae of patients with hypertensive disorders of pregnancy such as infarction, cytotropho blasticproliferation, syncytialknots, basement membrane thickening, , intervillos haemorrahge, decresed villous vascularity and fibrinoid necrosis were compared with control group and all parameters were statistically significant, which can adversely influence the perinatal outcome.

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