



Comparative Study of Histology of Placenta in Normotensive and Hypertensive Cases

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Abstract

Aim: Placenta is an organ of the vital importance for the continuation of a pregnancy and fetal nutrition. the present study was undertaken to find out the morbid changes of the placenta of hypertensive mothers in comparison to normotensive mothers.

Material and Methods: One hundred and fifty placentae of hypertensive and normotensive mothers were obtained from the department of obstetrics and gynecology. The placentae were fixed in 10% formalin and sectioned at 1 cm interval keeping the foetal surface intact. Blocks prepared were then stained with haemotoxylin and eosin.

Results: 100 placentae were from normotensive subjects (mean age 25.6 ± 4.0 years) and 50 belonged to hypertensive patients (mean age 25.12 ± 3 years). The infarction was observed in 12% cases of normotensive to 70% cases in severe hypertension. 70% cases in severe hypertension had syncytial knots while 90% had calcification. Stromal fibrosis and hypovascularity also increases with the severity of hypertension and was seen 50% and 30% cases respectively in severe hypertension.

Conclusion: Hypertension during pregnancy induces syncytial knots, calcification, Stromal fibrosis, hypovascularity which compromise utero-placental blood flow and may significantly reduce the neonatal birth weight.

Keywords: Placenta; hypertension; birth weight.

Introduction

Placenta, the only point of contact between maternal and foetal tissues, is an organ of the vital importance for the continuation of a pregnancy and fetal nutrition. The information provided from the pathological assessment of the placenta may provide important clinical information which interests the anatomists, pathologists and the

obstetricians.^{1,2,3} Hypertensive disorders in pregnancy are the second most common obstetric cause of stillbirths and early neonatal deaths in the developing countries which may be due to maternal genetic predisposition like specific patterns of genetic variant of angiotensinogen gene and quantitative trait loci on some chromosomes including 5q, 10q, and 13q.^{4,5} Both

maternal blood flow and foetal circulation maintains placental perfusion. Pregnancies complicated with hypertension leads to decrease in utero-placental blood flow due to maternal vasospasm.⁶ This leads to constriction of foetal stem arteries, foetal hypoxia and accordingly it may lead to foetal distress and foetal death.^{7,8} This has also been associated with the changes seen in the placenta of preeclamptic women. So, the present study was undertaken to find out the morbid changes of the placenta of hypertensive mothers in comparison to normotensive mothers with a view to assess the significance of villous abnormalities by histopathological methods under light microscopy because these changes may serve as a guide to the duration and severity of disease.

Material and Methods

One hundred and fifty placentae of hypertensive and normotensive mothers were obtained from the department of obstetrics and gynecology, IGMC, Shimla. The placentae were grouped depending on the degree of hypertension as described by Derek.⁹

Type of cases Blood Pressure (mm Hg)

- Normal 100/80 to 119/89
- Mild hypertension 120/90 to 139/99
- Moderate hypertension 140/100 to 169/119
- Severe hypertension >170/120

Placentae with cord and membranes were collected immediately after delivery. Any abnormality of cord and membrane was noted. In all cases, the amnion and chorion were trimmed from the placenta. The placentae were collected in 10% formalin and kept for fixation for 1-2 days.

For microscopic examinations, the placentae were sectioned at 1 cm interval keeping the foetal surface intact.

Each surface of each slice was examined for any obvious lesions.

The 3-5 mm thick section were taken as described by Carolyn M Salfia and Edwina J Popek in Anderson's Pathology¹⁰ as follows:

- a) Recommended minimum sections include
- b) One section from the membrane roll
- c) Two sections from the umbilical cord
- d) Four sections from the placental tissues were taken from the central part of placenta. The maternal and foetal surfaces were included in separate sections.

Additional sections were taken whenever macroscopic lesions were detected.

Enface Block Technique

In special case, enface block of the basal plate of the placenta were taken to assess the uteroplacental vasculature. This method consists of shaving the basal plate and embedding the block on its face. These blocks were stained by routine haematoxylin and eosin stain.

The processing of these sections was done as follows:

1. Fixation of sections was done in 10% buffered formal saline.
2. Dehydration was carried out in ascending concentration of ethanol.
3. Clearing was done in xylene, followed by impregnation in paraffin.
4. Tissue blocks were prepared using Leukhart's 'L' pieces.
5. Three to five micrometer sections were cut using rotary microtome and these stained with haematoxylin and eosin as follows:
 - i. Deparaffinization was done in xylene and the sections were brought to water.
 - ii. Staining with Harris haematoxylin was done for 10-40 minutes and bluing was done in running tap water.
 - iii. Sections were differentiated in one percent acid alcohol followed by washing in water.
 - iv. Counter staining was done with one percent aqueous eosin for 2-4 minutes followed by dehydration in ascending concentration of alcohol.
 - v. Sections were cleared in xylene and finally mounted in DPX and were examined under light microscopes in different magnifications.

Results

In the present study, a total of 150 placentae were subjected to microscopic examination. Out of these, 100 placentae were from normotensive subjects (mean age 25.6 ± 4.0 years) and 50 belonged to hypertensive patients (mean age 25.12 ± 3 years). In normotensive group, 52% patients were multiparous and 42% were primigravida whereas in the hypertensive group 54% were primigravidae and 46% were multiparous. The hypertensive patients were further divided into three groups i.e. mild, moderate and severe hypertension. The mean birth weight observed in normotensive group was 2752.5 ± 417.26 gm while it ranges from 2386.84 ± 340.69 gm in mild hypertension to 2205.0 ± 225.40 gm in cases of severe hypertension.

The infarction was seen as greyish patches and confirmed microscopically. It was observed in 12 (12%) cases of normotensive, 14 (66.6%) cases in

mild hypertension, 16 (84%) cases in moderate hypertension and 7 (70%) cases in severe hypertension (Table 1).

Increased syncytial knots were seen in 10 (10%) cases in normotensive, 6 (29%) cases in mild hypertensive, 12 (63%) cases in moderate hypertension, 7 (70%) cases in severe hypertension.

Calcification was seen in 7 (7%) cases in normotensive, 7 (33%) cases in mild hypertensive, 8 (42%) cases in moderate hypertension and 9 (90%) cases in severe hypertension.

Stromal fibrosis was seen in 15 (15%) normotensive cases, 6 (29%) cases in mild hypertension, 9 (47%) cases in moderate hypertension and 5 (50%) cases in severe hypertension.

Hypovascular villi are seen in 2% of normotensive, 4 (19%) cases in mild hypertensive, 5 (26%) cases in moderate hypertension and 3 (30%) cases in severe hypertension (Fig. 1).

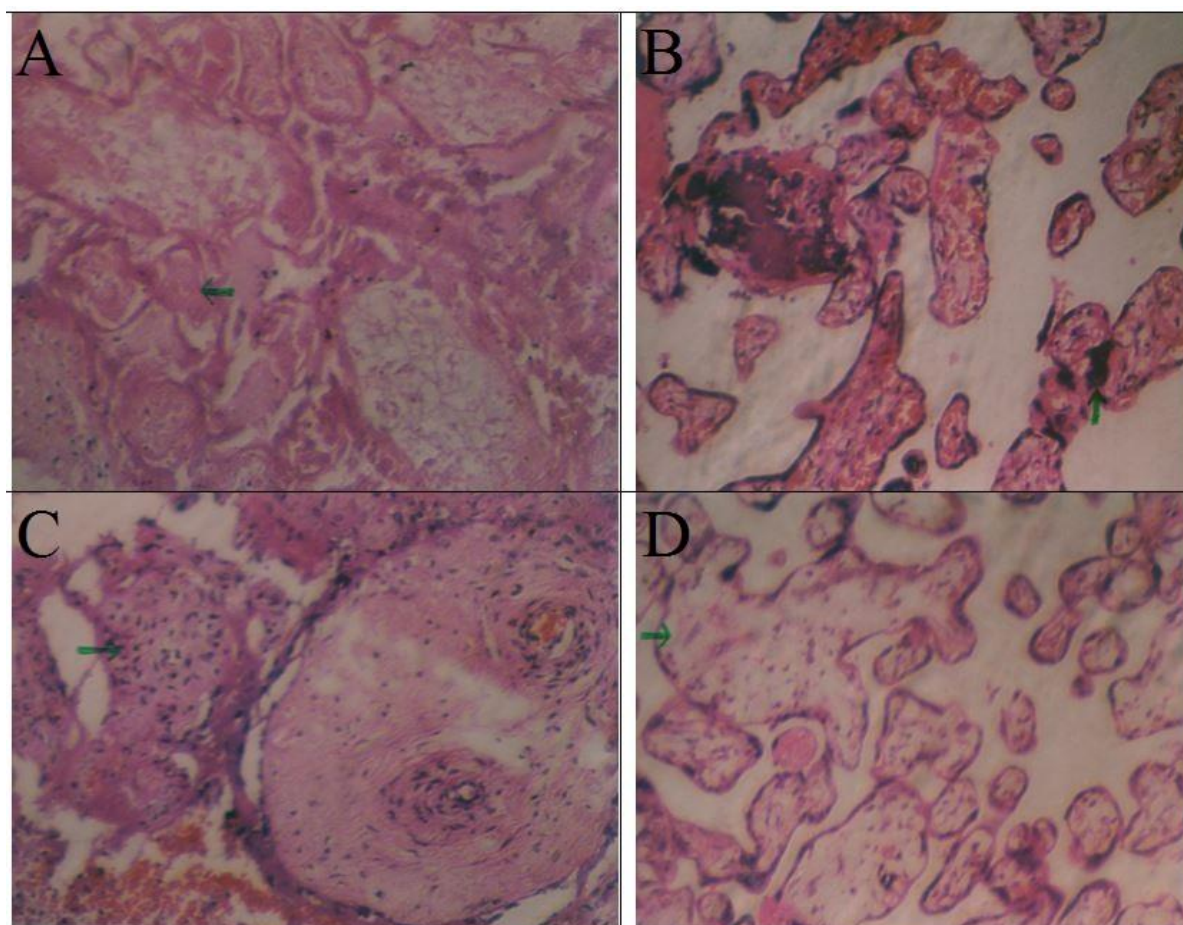


Fig. 1 Histological changes seen in placentae (marked with arrow) **A-** Infarction, **B-** Syncytial knots, **C-** Stromal fibrosis and **D-** Hypovascular villi.

Table 1 Microscopic findings observed in normotensive and hypertensive group

Microscopic Changes	Normotensive Group	Hypertensive Group		
		Mild Hypertension	Moderate hypertension	Severe hypertension
Infarction	12 (12%)	14 (66%)	16 (84%)	10 (100%)
Increased Syncytial knots	10 (10%)	6 (29%)	12 (63%)	7 (70%)
Calcification	7 (7%)	7 (33%)	8 (42%)	9 (90%)
Stromal Fibrosis	15 (15%)	6 (29%)	9 (47%)	5 (50%)
Hypovascular villi	2 (2%)	4 (19%)	5 (26%)	3 (30%)

Discussion

Wide spectrum of villous lesions were observed in hypertensive group in our study. These villous lesions could be attributed to the decreased maternal utero-placental blood flow in hypertension due to maternal vasospasm and worsening of placental pathology with progressive increase in hypertension.^{11,12}

Placentae from hypertensive mothers showed higher incidence of calcification and infarction in our study which is also observed in other studies.^{13,14} Also, the calcification and infarction of placenta increases with the increase in the severity of hypertension (Table 1). The calcification of placenta near the end of pregnancy indicates 'aging' of the placenta, but it may be the sign of the premature aging in hypertension, which will decrease the amount of nutrition and oxygen going to the baby and may worsen the postnatal outcome. Placental infarction is also more frequently observed in hypertensive cases due to thrombotic occlusion of maternal uteroplacental vessels.^{15,16}

Presence of syncytial knots increases with increasing gestational age, and with conditions of uteroplacental mal-perfusion and are important in placental examination.¹⁷ In our study, increased syncytial knots in placenta were observed in 70% cases with severe hypertension whereas placenta of only 10% of normotensive mothers showed syncytial knots which was also reported by Rohini Motwani et al¹³, Heazella AEP et al¹⁸ and Kristina L et al.¹⁹ They also hypothesized that the formation of syncytial knots may be induced by exposure to hypoxia. A significant increase in syncytial knot formation in placental villi

indicates the disturbance in the hormonal factors, which may probably lead to altered morphometry of placenta resulting in PIH in the mother.

In the present study, stromal fibrosis was also increased in hypertensive group which corresponds with the study of Rohini Motwani et al¹³, Heazella AEP et al¹⁸, Kristina L et al¹⁹ and Masodkar AR et al.²⁰ This increased incidence of stromal fibrosis may be related to reduce uteroplacental blood flow as a result of obliterative endarteritis which was found in placentae of hypertensive group.¹⁴

As reported in other studies as well as in our study villus vascularity was lower in PIH group.^{13,17,19,20}

The reduced number of vessels may be due to their de-novo poor formation or secondary to fibrosis.²¹

All the above changes in the placentae of hypertensive group may be due to reduced utero-placental blood flow, which may be accountable for increase in maternal and foetal mortality and morbidity. This signifies the need of thorough examination of placenta at the time of delivery.

Conclusion

Hypertension during pregnancy induces histological changes such as, areas of syncytial knot formation, stromal fibrosis, calcified areas, hyalinised areas, and hypovascular villi. These changes compromise utero-placental blood flow and may significantly reduce the neonatal birth weight. Further studies have to be undertaken to ascertain the statistical significance of microscopic villous abnormalities among hypertensive patients.

Conflict of Interest: Authors have nothing to declare.

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