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Original Article

Histology of Placenta in Normotensive and Hypertensive Cases in Khyber Pakhtunkhwa: A Comparative Study

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ABSTRACT

One of the most common pregnancy complications is Hypertension. It has a major effect on maternal and neonatal morbidities and fatalities. The placenta is an important organ for the maintenance of pregnancy and the nourishment of the fetus. **Objective:** To compare the morbid variations in the placenta of hypertensive and normotensive mothers. **Methods:** From the obstetrics and gynecology department, a hundred placentae of hypertension and normotensive females were collected. The placentae were preserved in formalin and sectioned. Blocks prepared were stained with hematoxylin and eosin. **Results:** The placentae from 50 normotensive mothers with 26±3.1 years (mean age) and 50 hypertensive mothers with 26± 2 years (mean age). In normotensive patients 12% Infarction was seen and 70% of severe hypertensive patients while syncytial knots were found in 70% of cases of severe hypertension, while calcification was observed in 90%. In severe hypertension, stroma fibrosis and hypervascularity increase in proportion to the degree of hypertension, with 50% and 30% cases seen in severe hypertension, respectively. **Conclusions:** In pregnancy hypertension causes, Stromal fibrosis syncytial knots, calcified, hypervascularity, all of which impair the uteroplacental flow of blood and lower newborn weight.

INTRODUCTION

Hypertension complicates roughly 10% of all pregnancies worldwide, and it is responsible for a significant burden of maternal and perinatal illness and mortality [1-3]. Hypertension is a frequent pregnancy condition that can have major consequences for both the mother and the fetus [4,5].In underdeveloped countries, hypertensive abnormalities during pregnancy are the 2nd most frequent obstetric reason for child deaths and early neonatal deaths, which may be attributable to the mother's genetic components such as specific patterns of angiotensinogen gene genetic variants and quantitative trait loci on some chromosomes such as 5q, 10q, and 13q [6]. Preeclampsia (PE) is one of the most dangerous hypertension illnesses, affecting 5–7% of all pregnancies globally and resulting in 70,000 maternal and 500,000 fetal fatalities each year [7-10]. The placenta is an important organ for the maintenance of pregnancies and embryonic nourishment as it is the only way to interact between maternal and fetal

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tissues. Anatomists, pathologists, and obstetrics may be interested in the information provided by the pathologist, that evaluation of the placenta as this could provide valuable clinical information [11]. Placental permeability is maintained by both maternal and fetal blood supply. Due to maternal vasospasm, pregnancy caused by hypertension results in a reduction in uteroplacental blood circulation [12]. This causes stenosis of fetal stem arteries and hypoxia and it results in fetal discomfort and death [13]. The goal of this study was to examine the morbid alterations in hypertensive and normotensive women's placentas to assess the importance of histologic abnormalities utilizing histological methodologies under microscopic observation in order to establish the disease's length and severity.

METHODS

The cross-sectional study was carried out in lady reading hospital Peshawar, a total of 100 placentae of both groups were obtained from the department of obstetrics and gynecology. Written informed consent was taken. The placenta, along with the cord and membranes, were obtained after delivery. If any cord and membrane abnormalities were identified. The amnion and chorion were taken from the placenta in all cases. According to Derek, both groups were divided on the basis of the degree of hypertension. "Normal blood pressure ranges from 100/80 to 119/89. Mild hypertension ranges from 120/90 to 139/99. Moderate hypertension ranges from 140/100 to 169/119. Severe hypertension ranges from >170/120" [14]. The placentas were collected in 10% formaldehyde solution and preserved for 1-2 days for fixation. The placentas were sectioned for microscopic investigations. Each slice's surface was checked for any visible lesions. The three sections were taken as follows: Four sections of placental tissues were obtained from the core region of the placenta, one from the membrane roll, and two from the umbilical cord. Separate sections were included for the maternal and fetal surfaces. In some cases, the Enface blocking technique of the placenta basal plate was taken to evaluate uteroplacental vasculature. hematoxylin and eosin stain were used to stain these blocks. Sections were fixed in 10 percent buffered formal saline. Dehydration was performed in increasing ethanol concentration. Clearing in xylene, followed by impregnation in paraffin. Tissue blocks were prepared. These specimens were cut by microtome and stained with hematoxylin and eosin as follows: Deparaffinization was done in xylene. Staining with hematoxylin and bluing. Sections were isolated in 1% acid alcohol before being washed in water. Counterstaining with eosin was done followed by dehydration. Sections were washed in xylene before being mounted in DPX and examined under light microscopes at various magnifications. Data was analyzed using Statistical Package for Social Sciences (SPSS) version 21. Chi-square test was done and 'p' value less than 0.05 indicated a statistically significant association.

Microscopic Changes	Group	Hypertensive Group			p-value
		Mild Hypertension	Moderate hypertension	Severe	
INFARCTION	14(14%)	16(68%)	18 (85%)	10(100%)	P=0.001
INCREASED SYNCYTIAL KNOTS	12(12%)	8(30%)	14 (64%)	6 (60%)	P=0.02
CALCIFICATION	9 (9%)	6(29%)	10(43%)	8 (80%)	P=0.001
STROMAL FIBROSIS	18(18%)	5(28%)	12 (45%)	4(40%)	P=0.001
HYPOVASCULAR VILLI	44%)	3 (18%)	6 (27%)	3(30%)	P=0.001

Table1: Normotensive and Hypertensive Group Microscopic Findings

RESULTS

Under a light microscope, both group slides were examined, including one hundred placentae. Group A had normotensive fifty participants with an average age of 26±3.1 years while Group B had hypertensive fifty patients with a mean age of 26±2 years. According to the multiparous and primigravida in both normotensive and hypertensive groups, showed 62% of patients were multiparous and 45% were primigravida in normotensive while in Group B hypertensive group 64% were primigravidae and 50% were multiparous. The degrees of hypertension were divided into mild, moderate, and severe hypertension. The normotensive group A had a mean birth weight of 2864.6 315.2 gm, while mild hypertension had a mean birth weight of 2275.2 210.2 gm and severe hypertension had a mean birth weight of 2112.2 101.40 gm. Under the microscope, the infarction was observable as bluish spots. Table 1 shows the normotensive and hypertensives cases of participants. The infarction was noticed in 14 (14%) normotensive, 16 (68%) mild hypertensive, followed by 18 (85%) moderate hypertensive, and 10 (100%) severe hypertensive cases. While the increased syncytial knots were noticed in normotensive 12 (12%), mild hypertensive 8 (30%), moderate hypertension was14 (64%), in severe hypertension 6(60%) cases. Normotensive calcification was observed in 9(9%) cases, in mild hypertensive 6 (29%), moderate hypertension in 10 (43%), and in severe hypertension 8 (80%) cases. Stromal fibrosis was observed in 18 (18%) normotensive cases, mild hypertension in 5 (28%) cases, moderate hypertension in 12(45%), and 4 (540%) severe hypertension cases. Hypo vascular villi are seen in normotensive 4%, in mild hypertensive 3 (18%), in moderate hypertension 6 (27%), and 3(30%) cases in severe hypertension.

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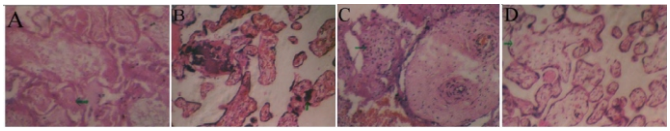

Figure 1: A) Infarction, B) Syncytial knots, C) Stromal fibrosis, and D) Hypovascular villi are histological abnormalities identified in placentae (labeled with arrow)

DISCUSSION

The calcification of the placenta near the end of pregnancy is a sign of age, but it could also be hypertension-related premature ageing, which limits the amount of nutrients and oxygen delivered to the infant and worsens perinatal output [15]. Due to thrombotic blockage of maternal uteroplacental arteries, placental hemorrhage is more likely in hypertension patients. In our research, placentas from hypertensive parents had a greater incidence of infarction and calcification, which was also noticed in other research findings [16,17]. In the severity of hypertension calcification and infarction of the placenta increase shown in (Table 1). Browne et al., observed that the histologic lesions could be contributed to significantly reduced maternal uteroplacental blood supply in hypertension associated with pregnancy vasospasm, as well as adversely affecting placental pathology with progressive raise [18] while in our study hypertension group had a wide range of villous lesions. Syncytial knots become more common as the pregnancy progresses and the uterus and placenta don't get enough blood. They are important to look for during a placental examination [19]. Rohini Marwani et al., in 2013 reported the syncytial knots in hypertensive group number of areas was 60% which was knowingly raised (26 %) than in the control group while in our study , 60% of cases with severe hypertension had increased syncytial knots in the placenta whereas the placenta of normotensive mothers had 12% of syncytial knots. Hypoxia is induced by exposure syncytial knots. the disturbance in the hormone, that lead to disrupted morphometry of placenta and PIH in the mother had significance in syncytial knot formation in placental villi [9]. In 2018, Jahsan et al., reported syncytial knots, stromal fibrosis, and fibrinoid necrosis in a significantly higher number of cases than controls [1]. Corresponding to our study, stromal fibrosis was also increased in the hypertensive group. These higher rates of stromal fibrosis could be linked to a reduction in uteroplacental blood flow caused by obliterative endarteritis in hypertensive placentas [20]. The lower number of vessels could be due to fibrosis or inadequate vascular development from the start. Each of these abnormalities in hypertensive women's placentas could be ascribed to a reduction in uteroplacental blood flow, which could lead to an increase in maternal and fetal mortality. This emphasizes the significance of a comprehensive placental inspection at the time of birth [21].

CONCLUSIONS

Histological alterations such as syncytial knot formation, stromal fibrosis, calcified areas, hyalinized areas, and hypovascular villi are all caused by hypertension during pregnancy. The uteroplacental blood flow is obstructed by these changes, which could reduce newborn weight. The placenta is affected by pregnancy-induced hypertension, which may be associated to poor perinatal outcomes. Gynecologists, embryologists, and pathologists will benefit from this research.

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