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ORIGINAL ARTICLE

Compare the Histological Features between the Normal and Hypertensive Placenta

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ABSTRACT

Objective: The purpose of the current research was to investigate the morphology and histology of placentas collected from moms who had PIH and to compare those results to placentas taken from women who had normal pregnancies.

Study Design: The study was conducted at Anatomy department of Women Medical and Dental College Hospital Abbottabad and Mohi-ud-Din Islamic Medical College, Mirpur, AJK during the period from February 2021 to July 2021.

Place and Duration: Observational/comparative study

Methods: In this study total 80 pregnancies, 40 normal placenta's and 40 hypertensive placenta's were presented. At first, the physical aspects of placentas, also known as their morphological traits, were noted. In order to investigate the histology, pieces of each placenta measuring 5 millimetres in thickness were removed. After this, the tissue underwent further histological processing, after which it was fixed in formal saline at a concentration of 10 percent. It was noted down how much newborns weighed at birth. SPSS 23.0 was used to analyze complete data.

Results: Placental weight, size, surface area and number of cotyledons were decreased in hypertensive placentas, whereas regions of infarction, retroplacental clot, and calcification were increased. Hyalinized villi and atherosclerosis have been reported in hypertensive placentas (p0.05). Foetal mortality and morbidity were closely linked to these alterations. We also found that among women with high blood pressure who had the aforementioned histological alterations in their placentas, the risk of foetal death and morbidity was higher.

Conclusion: We concluded that, PIH reduces placenta weight and size. Changes in utero-placental blood flow may induce placental insufficiency. It affects newborn birth weight. PIH impacts placenta shape, histology, and foetal development.

Keywords: Placenta, Pregnancy-induced hypertension, Histological, Morphological, Mortality, Morbidity

INTRODUCTION

The fetus's ability to survive in the womb is entirely reliant on the "Placenta." The fetus's regular growth and development depends on the presence of a healthy placenta.[1] It's the most accurate record of the baby's prenatal experience available to date[2]. A morphological record of the anatomical state, intrauterine events, and intrapartum events of gestation may be found in it. One of the most common causes of maternal death and a significant contributor to foetal loss is pregnancy-induced hypertension (PIH). Both macroscopically and microscopically, pregnancy problems such as hypertension are reflected in the placenta. In PIH, maternal vasospasm reduces utero-placental blood flow, according to a number of studies. [3] Preeclamptic women's placentas have been linked to foetal stem artery constriction as a result of this. [4] Fetal hypoxia, brought on by maternal vasospasm, may result in suffering or even death in the foetus. [5] Doppler ultrasonography may identify the decreased placental perfusion before to clinical signs of PE (US). The artery of the womb As a result of placental insufficiency, Doppler reflects downstream placental vascular resistance, which is linked to the multisystem consequences. Doppler waveform analysis of the umbilical artery may considerably reduce perinatal mortality in high-risk pregnancies, according to recent meta-analysis of randomised controlled studie[6]s. This has led to an increase in the use of Doppler ultrasonography findings in PE diagnosis. Histopathological changes in the hypertension-prone placenta include infarcts of the organ, increased syncytial knot formation, hypovascularity of the villi, accelerated cytotrophoblastic proliferation, thickening of the subtrophoblastic basement membrane, and atherosis of placental bed spiral vessels. These changes are all associated with an increased risk of preterm birth and preterm delivery. With increasing pre-eclamptic severity, intervillous and terminal villi volume decreases as well. [7,8]

Pregnancy-related hypertensive problems are prevalent, and they are part of a fatal trio that also includes hemmorage and infection, both of which increase the risk of mother and foetal death. According to several research [9,10], hypertension is to blame for anywhere from 2.6% to 7.6% of all maternal fatalities. It's common for preeclampsia-affected pregnancies to result in premature birth, low birth weight, placental abruption, Caesarean section, liver insufficiency, subcapsular liver hematoma, cerebral edoema, kidney failure, and intravascular coagulation.[11]

Placental anomalies have been implicated in the consequences of hypertensive diseases during pregnancy [12,13]. Hypertensive women's placenta has received a lot of attention, and pathological alterations have been documented [14]. Normal foetal development and survival may soon depend on placental villous angiogenesis [15].

We wanted to see whether the placenta's macroscopic and microscopic morphological traits differed between pregnancies with severe hypertension and those without.

MATERIAL AND METHODS

This observational study was conducted at Anatomy department of Women Medical and Dental College Hospital Abbottabad and Mohi-ud-Din Islamic Medical College, Mirpur, AJK during the period from February 2021 to July 2021 and comprised of 80 placentas. Women's gestational age, age and body mass index was recorded after taking informed written consent. The placentae of pregnancies complicated by other medical disorders, multiple pregnancies and preterm pregnancies were excluded.

Among 80 cases 40 placenta's were hypertensive (group I) and 40 were normal placenta's (group II). Singleton pregnancy, high blood pressure throughout pregnancy, preeclampsia, eclampsia, preeclampsia superimposed on chronic hypertension, and chronic hypertension were the inclusion criteria for this study. Shortly after birth, we removed the placenta and umbilical cord, cleaned them under running water, tagged them and stored them in 10% formalin for 4-6 weeks. The placenta was examined under a microscope and under a microscope. Each plant's dimensions and characteristics were meticulously documented, including the number of cotyledons and the exact location of its umbilical cord insertion. The feto-placental weight ratio was estimated based on

the birth weights of newborns. The placenta was investigated using a light microscope for histopathological analysis.

It was necessary to document the results of several morphological and morphometric examinations. The data was analysed via descriptive statistics. They were shown as Mean Standard Deviation (SD) (standard deviation). Students unpaired "t" test was used to examine the statistical significance of the differences between the mean values of the control and study groups. A statistically significant difference was defined as one with a P value less than 0.05.

RESULTS

In group I mean age of the females was 28.6±3.54 years with mean gestational age was 34.13± 7.34 weeks while in group II mean age was 29.5±6.19 years and had mean gestational age was 35.7±3.32 weeks. Birth weight of fetus in group I was 2431.5±4.65 grams and in group II mean birth weight was 2912.7±2.42 grams. In group I mean weight of placenta was 412.8±3.18 grams and in group II weight was 423.7±11.32 grams.(table 1)

Table-1: Demographics details of mothers and morphological details of placenta

Variables	Group I	Group II
Mean Age (years)	28.6±3.54	29.5±6.19
Mean Gestational age (weeks)	34.13± 7.34	35.7±3.32
Mean birth weight of fetus (g)	2431.5±4.65	2912.7±2.42
Mean placenta weight (g)	412.8±3.18	423.7±11.32

We found that regions of infarction, retroplacental clot, syncytial knots, stromal fibrosis, fibrinoid necrosis and calcification were higher in hypertensive groups as compared to normal cases with p<0.005.(table 2)

Table-2: Comparison of histological findings among both groups

Variables	Group I	Group II	P value
infarction	0.3+1.5	1.3+1.4	0.03
retroplacental clot	5.2+2.16	3.1+4.29	0.04
syncytial knots	27.6+4.5	21.7+6.5	0.05
stromal fibrosis	2.7+2.8	0.9+4.12	0.01
fibrinoid necrosis	4.11+4.9	1.13+7.9	0.04
calcification	0.12+ 0.61	0.9+ 0.43	0.01

Foetal mortality and morbidity were closely linked to these alterations. We also found that among women with high blood pressure who had the aforementioned histological alterations in their placentas, the risk of foetal death and morbidity was higher.(table 3)

Table-3: Comparison of morbidity and mortality among both groups

Variables	Group I	Group II
Morbidity		
Yes	8 (20%)	1 (2.5%)
No	32 (80%)	39 (97.5)
Mortality		
Yes	7 (17.5%)	0
No	33 (82.5%)	40 (100)

DISCUSSION

Pregnancy-induced hypertension and its related mortality and morbidity have been linked to the weight of the placenta and the baby's birth weight in previous research. A stillbirth was shown to be a prevalent complication of hypertensive pregnancies, according to researchers. Researchers have found that in hypertensive pregnant women, there is an increased risk of intrauterine growth retardation (IUGR) and placental haemorrhage. Placental morphological features might explain why preeclampsia babies are smaller. [16] For example, abnormalities like fibrinoid necrosis, a lack of vascular membrane, and an increase in thickening of the trophoblastic membrane were linked to the severity of preeclampsia, which led to a poorer outcome for the baby. [17]

In this study we have taken 80 placentas in which 40 were hypertensive and 40 were normal. In group I mean age of the females/mothers was 28.6±3.54 years with mean gestational age was 34.13± 7.34 weeks while in group II mean age was 29.5±6.19 years and had mean gestational age was 35.7±3.32 weeks. These results were comparable to the previous studies.[18,19] Birth weight of fetus in group I was 2431.5±4.65 grams and in group II mean birth weight was 2912.7±2.42 grams. In group I mean weight of placenta was 412.8±3.18 grams and in group II weight was 423.7±11.32 grams. In the Shevade et al [20] study, the average foetal weight was 2.1 kilogrammes in the PIH group and 2.8 kilogrammes in the control group. Mayhew and colleagues, S. Kishwara et al. [21], Boyd and Scott10 (1985), (2003) [22] Barton et al. (2001)[23] found that the preeclampsia group's mean neonatal birth weight was lower than the control group's. Birth weight was 5 percent lower in women who had preeclampsia, according to Odegard et al.[24].

We found that regions of infarction, retroplacental clot, syncytial knots, stromal fibrosis, fibrinoid necrosis and calcification were higher in hypertensive groups as compared to normal cases with p<0.005. Normal patients had 40 percent fibrinoid necrosis, but hypertension patients had 84 percent.[25] In one previous study normal patients had 2.96 +-3.52 regions of necrosis, but those with PIH had 6.26 +-2.8 [26] areas of necrosis, which is significant. Fibrinoid necrosis was found in 33.34 percent of placentas in 2011 by Pandure and Ghosh D.K. [27]. According to Kantilal M et al, 69 percent of placentas with hypertension and 38 percent of placentas with normal blood pressure had syncytial knots [28]. For hypertensive placentae, the mean number of syncytial knot formation areas under low power field was 26.47 and 8.93, which is close to the study made by Pratishtha potdar, which was 28.57 9.56 for hypertensive placentae,29] and similarly 23.15 4.84 for normotensive placenta in a study made by Prathiba A[30].

These changes have a direct impact on foetal death and morbidity. We also discovered that the probability of foetal death and morbidity was increased in women with high blood pressure who had the aforementioned histological abnormalities in their placentas. When a pregnant woman has hypertension, it has a detrimental effect on both her and her unborn child, which is why it is so dangerous to the health of the entire population. There are several changes that occur in the spiral arteries of the uterus after conception and during placentation, which were normal before conception. [31]

CONCLUSION

We concluded that, PIH reduces placenta weight and size. Changes in utero-placental blood flow may induce placental insufficiency. It affects newborn birth weight. PIH impacts placenta shape, histology, and foetal development.

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