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MACROSCOPIC AND MICROSCOPIC STUDY OF PLACENTA IN HYPERTENSIVE AND NORMAL PREGNANCIES

Anatomy

Dr. Somavathi — 3rd Year PG, Department of Anatomy, Shadan Institute of Medical Sciences Teaching Hospital and Research Centre.

Potuganti Mithil Kumar* — Assistant Professor, Department of Anatomy, Shadan Institute of Medical Sciences Teaching Hospital and Research Centre. *Corresponding Author

ABSTRACT

Introduction: Pregnancies with hypertensive disorders are prone to a higher risk of preterm deliveries and low birth weights compared to healthy pregnancies. The risk of hypertensive disorders of pregnancy occurs mostly among mothers affected with severe chronic hypertension as well as those with superimposed preeclampsia on chronic hypertension.

MATERIALS AND METHODS: This is prospective and observational and case-control study. The study was performed in the Department of Anatomy, Pathology, Gynaecology and Obstetrics of Shadan Institute of Medical Sciences Teaching Hospital and Research Centre, during the period two years. Inclusion criteria: The pregnant mothers in the age group 20 years to 40 years attended for antenatal check-up. Patients having detailed history, clinical data, consent & cooperation of patients, blood and urine report and specimen of placenta available for examination.

Result: Birth Weight, Gestational age, Appearance, Pulse, Grimace, Activity, and Respiration (APGAR) score was decreased in hypertensive cases than normal. In the progress of hypertensive condition from gestational hypertension (GH) the birth weight of baby and gestational age was reduced significantly, but the reduction was not significant between GH and control group. Two Intra uterine death cases were reported in severe preeclampsia group.

Conclusion: The present study showed extensive changes in the structure of placenta in hypertensive pregnancy. The abnormalities in the angiogenic factors are responsible for this alteration.

KEYWORDS

Macroscopic, Microscopic, Placenta, Hypertensive, Normal Pregnancies

INTRODUCTION

Pregnancies with hypertensive disorders are prone to a higher risk of preterm deliveries and low birth weights compared to healthy pregnancies. [1] The risk of hypertensive disorders of pregnancy occurs mostly among mothers affected with severe chronic hypertension as well as those with superimposed preeclampsia on chronic hypertension. [2] Hypertensive disorders are common complications of pregnancy. Most adverse events like maternal and neonatal mortality and morbidity are attributed directly to the preeclampsia syndrome, characterized by new-onset hypertension with proteinuria during pregnancy. [3] Hypertensive disorders during pregnancy are classified into 4 categories, as recommended by the National High Blood Pressure Education Program Working: Chronic hypertension, Preeclampsia- eclampsia, Preeclampsia superimposed on chronic hypertension, Gestational hypertension (transient hypertension of pregnancy or chronic hypertension identified in the latter half of pregnancy). [4] A pregnant woman with a blood pressure of less than 140/90mmHg, throughout the pregnancy, is considered as normotensive. [5]

Hypertension is one of the medical problems that mostly affect pregnant women and it remains an important cause of both maternal and foetal morbidity/ mortality. Studies show that 10–15% of pregnancies will be complicated by high blood pressure. [6] Up to about one-quarter of all antenatal admissions will be hypertensive related cases. Over the last century, maternal mortality rates in high-income countries have steadily declined. [7] Every year about 70,000 women die and there are half a million stillbirths or neonatal deaths owing to hypertensive disorders of pregnancy (HDP)–the vast majority being in the developing world. [8]

The identification of the Hypertensive disorder and its effective treatment play a beneficial role in pregnancy outcomes for the mother and the foetus, and hence a reduction in both maternal and perinatal mortality. Hypertensive disorders are associated with low birth weight, fetal growth restriction and prematurity which greatly contribute to perinatal morbidity and mortality. [9] Many pregnancy complications that are associated with high foetal morbidity and mortality have shown gross deviations from the normal placental morphology and anatomy. [10] With the placenta serving as the image for the health status of the mother and foetus, complications like hypertension in pregnancy has reflected in the placenta in a significant way, either microscopically or macroscopically. [11]

The placenta is the growing organ of the human body. The normal placenta parenchyma is divided into 10-40 lobes or lobules separated by grooves or septa. During the first twelve weeks of development, the placenta consists of mesenchymal villi after this period, subsequently stem or anchoring villi are formed. [12] Microscopically the bulk of the villi consist of connective tissue in which blood vessels are found. The outer part of villus is surrounded by the syncytiotrophoblast which resembles like a cuboidal epithelium. Most of the cells in the connective tissue core of the villi are fibroblast. [13]

MATERIALS AND METHODS

This is prospective and observational and case-control study. The study was performed in the Department of Anatomy, Pathology, Gynaecology and Obstetrics of Shadan Institute of Medical Sciences Teaching Hospital and Research Centre, during the period two years.

INCLUSION CRITERIA:

The pregnant mothers in the age group 20 years to 40 years attended for antenatal check-up. Patients having detailed history, clinical data, consent & cooperation of patients, blood and urine report and specimen of placenta available for examination.

Pregnant women having hypertension (blood pressure (BP) ≥ 140/90mm Hg). Normotensive (blood pressure of less than 140/90mmHg) pregnant mothers without any illness. Specimen of placenta collected from the full-term delivery cases (i.e, cases completed 37 weeks of gestation).

EXCLUSION CRITERIA:

Patients with age below 20 years and above 40 years. Other Medical or Surgical illness. Blood and urine report and consent not available and patients lost or missing in the follow-up period.

Examination of the specimen:

Examinations of placenta were conducted according to proforma adopted by Benirschke and later modified by Woodly et al [14].

Gross examination:

Following parameters of the placenta were determined: - dimensions, surface area, weight, volume, shape, examination of the fetal surface, insertion of the umbilical cord and amniotic membranes. Examination of the maternal surface, blood clots, infarcts, number of cotyledon and examination of cut sections.

Morphometry:

The collected placentae were washed under running tap water and the membranes were trimmed. The site of insertion of the cord was noticed. The umbilical cord length was measured by using measuring tape and then it was removed 4cm away from the placenta. Number of coiling was counted. The diameter of cord at three sites - foetal end, middle and maternal end- were also been measured with Vernier callipers before it separated from the placenta. Coiling index was calculated by dividing the number of coils with length of cord.

STATISTICALANALYSIS:

Statistical Analysis was done by using SPSS software Version 25th. Statistical Tests (Pearson Chi-square Test, Independent Samples Test) were applied whenever it was necessary. For significance p-value

RESULTS

In table 1, Birth Weight, Gestational age, Appearance, Pulse, Grimace, Activity, and Respiration (APGAR) score was decreased in hypertensive cases than normal. In the progress of hypertensive condition from gestational hypertension (GH) the birth weight of baby and gestational age was reduced significantly, but the reduction was not significant between GH and control group. Two Intra uterine death cases were reported in severe preeclampsia group. The details are given in table 1.

Table 1: Clinical Details

Parameters	Normal Pregnancy (Control)	Hypertensive Pregnancy (Case)
Baby weight in grams	2743.63 ± 343.21	2382 ± 312.43 *
Gestational age in Wk	39.23 ± 3.13	36.43 ± 3.63 *
Apgar score at 1 minute	7.73 ± 0.46	6.53 ± 0.57 *
Apgar score at 5 minutes	8.79 ± 0.31	7.43 ± 0.73 *

* = P<0.05 (Significant), **= P<0.001 (highly significant).

Table 2: Gross Morphometry - Placenta

Parameters	Normal Pregnancy (Control)	Hypertensive Pregnancy (Case)
Placental weight (grm)	443.72 ± 51.23	332.7 ± 38.21*
Fetoplacental (FP) Ratio	6.52 ± 0.63	7.12 ± 0.73*
Placental volume (cm3)	383.63 ± 41.73	263.63 ± 63.21*
Number of Cotyledon	14.3 ±1.37	10.41 ± 1.73*
Placental thickness (cm)	2.31 ± 0.42	2.13 ± 0.53
Placental diameter (cm)	17.89 ± 1.32	14.83 ± 1.43*
Site of insertion of cord	Central-13 (32%) Medial-16 (34%) Lateral-10 (30%) Marginal-1 (4%)	Central-16(36%) Medial-12 (30%) Lateral-11 (30%) Marginal-1 (4%)

Weight, volume and thickness were significantly reduced in hypertensive placenta. Among the hypertensive group, very small placentae were observed in severe preeclampsia groups. Feto-placental Ratio were increased in hypertensive group, highest ratio was found in severe pre-eclamptic group. Values are given in table 2.

Table 3: Histomorphometry – Placenta

Parameters	Normal Pregnancy (Control)	Hypertensive Pregnancy (Case)
Number of villi	9.12±2.48	12.43±3.53*
Volume of villi (mm3)	72.21±8.54	79.59±8.35*
Volume of IVS (mm3)	48.59±4.49	39.54±4.49*
Surface area of villi (mm2)	50.49±5.93	62.49±8.49*
Diameter- villi (mm)	19.48±3.30	17.49±3.49*
Capillary/ villi	5.49±0.39	3.49±0.48*
Vasculo syncytial membrane	9.39±2.39	5.49±0.39*
Thicknesssyncytium	1.49±0.49	1.83±0.31
Syncytial knots	8.59±1.49	13.4±3.39*

Number, volume and total surface area of villi were significantly increased in preeclampsia group, whereas inter villous space were reduced, which may be due to the proliferation of terminal villi. But no significant difference between control and hypertensive pregnancy. The above changes were more evident in severe group.

Table 4: Histomorphometry - Placenta - Stem Villi Vessels

Parameters	Normal Pregnancy (Control)	Hypertensive Pregnancy (Case)
WL Ratio stem artery	0.64±0.18	1.8±0.94*
Wall Thick Stem Artery	7.26±1.83	7.49±2.43
Media Stem Artery	5.02±1.42	5.54±1.59
WL Ratio stem vein	0.19±0.07	0.53±0.08*
Wall Thick Stem Vein	3.39±1.24	5.43±2.63*
Media- Stem Vein	3.23±1.12	4.54±1.62*

Stem villi vessels of placenta also have a change in their structure. Proliferation of smooth muscles of media was observed in hypertension especially in severe cases. So, the wall lumen ratio also increased in hypertensive pregnancy. Given in table 4.

Table 5: Histopathology – Placenta

Parameters	Normal Pregnancy (Control)	Hypertensive Pregnancy (Case)
Stromal fibrosis	4.03±0.84	7.01±2.74*
Fibrinoid necrosis	3.54±0.21	5.94±1.49*
Hyalinised villi	6.01±1.49	9.03±2.46*
Endarteritis obliterance	1.93±0.74	3.02±0.81*
Cytotrophoblast: proliferation	3.98±0.84	8.01±1.09*
Trophoblast basement membrane thickness	3.98±0.74	7.03±1.11*

DISCUSSION

The present study found the reduction of the birth weight, placental weight and APGAR score were more in severe preeclamptic group. Udainia et al et al reported that the birth weight, placental weight and APGAR score were significantly reduced in pregnancy induced hypertension when compared with normal. [15] Gunapriya et al also observed a similar result in severe preeclampsia. [16]

The present study observed a weight reduction in placenta of hypertension which is inversely proportional to maternal blood pressure. So, the lowest placental weight has been noticed in hypertensive pregnancy along with reduced baby weight and APGAR score. Lo YF et al et al stated that the fetal growth capacity is determined by the efficiency of placenta and umbilical cord. So, insufficiency of the placenta and cord might be the reason for the reduction of birth weight of the baby. [17] According to Manop Janthanaphan et al the baby weight is increasing proportionately along with placental weight in normal pregnancy. [18]

Molteni et al suggested the baby weight and placental weight are increased along with increasing gestational age. He also observed that the preterm birth is more frequent in hypertensive pregnancy that is accordance with our study. [19] Study of Allen et al supports the present finding of small for gestational age in preeclampsia and the severe group reported still birth also. The low perfusion of placenta is associated with preterm birth and fetal mortality. Failure in the remodelling of spiral arteries by invasion of trophoblastic cells, as normal pregnancy is the reason for the insufficient blood supply of placenta in hypertension. [20] According to Barker et al baby with low birth weight and placental weight may have cardiovascular disease in their later life. [21]

The present study also showed there was a reduction in placental parameters especially lowest in severe preeclampsia. The reduction of all these parameters might contribute to the total decrease of placental weight and volume. According to Gill JS et al stated that the placental dimensions like its thickness, diameter, and number of cotyledons were reduced in pregnancy induced hypertension. [22]

In preeclampsia fetoplacental blood flow is severely impaired and transplacental gas and nutrition exchange is poor, placing the foetus at a risk of hypoxia, and low birth weight. When the rate of uteroplacental flow is chronically reduced, there is a tight direct linear correlation between the rate of mean uteroplacental blood flow and placental weight. The placenta has the ability to control the growth of the foetus. So, the mean birthweight and gestational age are lower, and preterm deliveries are higher in hypertension. Any kind of placental dysfunction may affect oxygen exchange and which may lead to a state of oxidative stress and chronicfoetal hypoxemia. [23]

Pre-eclampsia may be a factor that evokes an initiation of hypertension in utero and its amplification through childhood and adulthood. According to Barker et al it seems to be possible that similar alterations like umbilical arteries occur in other foetal blood vessels. It may result in an increase of peripheral resistance as well as an increase of the blood pressure in the foetal vascular system.[24] In-vitro experiments by Howard et al on perfused human placental cotyledons show that acute reduction in oxygen tension induces foeto placental vasoconstriction.[25]

CONCLUSION

The present study showed extensive changes in the structure of placenta in hypertensive pregnancy. These structural alterations affect the foetal outcome adversely. The abnormalities in the angiogenic factors are responsible for this alteration. In conclusion, the data demonstrated that placental tissue from pregnancies with hypertensive disorders showed a different expression of angiogenic factors according to different degrees of clinical severity.

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