Macroscopical and microscopical study of placenta in normal and in pregnancy induced hypertension

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الخلاصه

المشيمه هي مؤشر للاضطرابات التي تؤثر على الأم والطفل. العديد من هذه الاضطرابات ترافقها تغيرات نسجيه في المشيمه. الدراسه الحاليه هي تحليل للتغيرات المظهريه والنسيجيه للمشيمه في حالات ارتفاع ضغط الدم المتعلق بالحمل. تمت دراسه 50 مشيمه (25 من الحوامل التي تعاني ضغط مرتفع و 25 من الامهات ذوات الضغط الطبيعي . اظهرت معايير القياس الشكليه في مجموعة الضغط المرتفع بان هنالك زيادة في معدلات كل من : وزن المشيمه ، قطر المشيمه ووزن المواليد بالمقارنه مع المجموعه الطبيعيه. تحليل التغيرات العيانيه بين نقص في معدل ا لفلقات وزياده في المواقع المكلسه اضافه الى زيادة الاندغام الجانبي للحبل السري مع المشيمه.

الشكل النسيجي للمشيمه في الامهات التي تعاني من ضغط مرتفع اظهر زياده في بقع التنكس الزجاجي وزياده في مناطق التكلس كذلك زياده تشعب الاوعيه الدمويه ذات الحجم المتوسط.

يمكن الاستنتاج ان ارتفاع ضغط الدم المرافق للحمل يولد انعكاسا واضحا على المشيمه من الناحيه المظهريه والنسيجيه.

Abstract

Placenta was an indicator of maternal and foetal disorders. Many of the disorders of pregnancy which are associated with high prenatal morbidity and mortality are accompanied by changes in placental histology. The current works was an analysis of gross and histological changes of placenta in hypertensive disorders of pregnancy. A total of 50 placentas are studied 25 placenta from hypertensive pregnant mothers and 25 from non hypertensive pregnant mothers. The morph metric parameters in hypertensive group revealed that there is an increase in the mean placental weights, mean placental diameter and mean birth weights in comparison with normal group.

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The analysis of macroscopic placental changes showed decrease in the mean number of cotyledons and increase in mean number of calcified areas as well as an increase in the number of marginal insertion of umbilical cord.The histological appearance of placenta in hypertensive pregnant mothers revealed increase in hyalinized spots and areas of calcification also a medial coat proliferation of medium sized blood vessels are observed . In conclusion the hypertensive disorder of pregnancy well reflected on mother placenta.

Introduction

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 consist of mesenchymal villi after this period subsequently stem or anchoring villi are formed. (1) 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 looks like a cuboidal epithelium. Most of the cells in the connective tissue core of the villi are fibroblast (2). In the first weeks of development the whole placenta consists of mesenchymal villi and after approximately 12 weeks immature intermediate villi are formed. Immature intermediate villi are no longer present after 24 weeks of pregnancy.

The terminal villi can be recognized by the presence of syncytio-vascular membranes .(3) Under normal conditions terminal villi can be recognized from 30-32 weeks onwards and around term 40% of the placental villi consist of these terminal villi.(4)

Many of the disorders of pregnancy which are associated with high perinatal morbidity & mortality are accompanied by gross pathological changes in placenta. Abnormal maturation can be seen in several different con ditions.Accelerated maturation i.e. premature formation of terminal villi can be

seen as a reaction or adaptation of the placenta to a decreased materno- placental perfusion.(5)

Histologically it can be recognized by a decrease of villous diameter and by accelerated formation of syncytio- vascular membranes failure of the second phase of trophoblast invasion of the spiral arteries is generally believed to give rise to several pregnancy induced hypertensive. Disorders of pregnancy e.g. pre- eclampsia delayed maturation can be seen in several different clinical situations it is well known in association with maternal diabetes but it can be seen also in macrosomic placentas in mothers without diabetes (6) .It can be observed in association with congenital and / or chromosomal anomalies. Another abnormality known under several different names associated with late intra-uterine fetal death is delayed maturation of the terminal villi, defective placental maturation or probably also terminal villi deficiency (7). The aim of the present study was to correlate the morphometry and histology of placenta from mothers with normal and pregnancy induced hypertension.

Material and Methods

Twenty five placentas from normal pregnant woman and twenty five placentas from pregnancy induced hypertension mothers were studied

Selected from Al-Hilla Teaching hospital. Mothers with hypertensions had their blood pressure ranging from 140\90 mm to 160\110 mm of Hg and above .After delivery placenta were collected for cross and histological studied .The size, surface area weight of placenta were noted along with the inspection of marginal vein for any thrombosis, the number of cotyledon, condition of membrane, pressure of infarction, calcification, and site of insertion of umbilical cord were noted.

The new born baby's birth weights were noted and the foeto-placental weight ratio was calculated in each case tissues were taken from different sites of placenta for histological studies.

Results

The placenta morphometric study (table-1) revealed that the mean placental weight was 487.50 ± 39.13 in the normal pregnant group and it was 400.75±60.31in the hypertensive group. The mean placental diameter 17.82±6.93 in normal and 16.00± 5.89 in hypertensive mothers. The mean birth weights for babies were 3.0±0.23 in the first group and 2.9±0.56 in hypertensive group.

The gross anatomy of the placenta (table-2) revealed the mean number of cotyledons was 18±2 in normal and 16±2 in hypertensive group. The mean calcified area was 5.24±0.15 in first group and 21.3±0.32 in hypertensive mother's .The mean marginal insertion of umbilical cord (fig.1) was 2.12±0.02 in normal group and 7.3±3.15 in pregnancy induced hypertension group.

Histological study of placental villi (fig.2) on examination under microscope it had been noticed that hyalinised villous spot (fig.2B) and calcified (fig.2D) also a medial coat proliferation of medium sized blood vessels (fig.2C) were observed per low power in the hypertensive group in comparison with placenta of normal pregnant group (fig.2A).

Table-1: Placenta morphometric study in normal and in pregnancy induced hypertension.

Type	Normal group	Hypertensive group
Mean placental wt. in grams	487.5±39.13	400.75±60.31
Mean placental diameter	17.82±6.93	16.00±5.89
Mean birth wt. of babies in kg.	3.0±0.23	2.9±0.56
Mean foeto-placental wt. ratio	4.89±0,63	5.48±0.24

Table-2 : Gross anatomy of placenta in normal and in hypertensive groups.

Type	Normal group	Hypertensive group
Mean number of cotyledons per placenta	18+2	16+2
Mean calcified area in placenta	5.24±0.15	21.3±0.32
Marginal insertion of umbilical cord	2.12±0.02	7.3±3.15

Discussion

The placenta regarded as valuable indicator of maternal and foetal disease. Placental examination can lead to the identification of basic morphologic alteration which can be easily documented and useful for diagnosis of feto-neonatal pathology (8)

The current study reveals that the placental weights and placental diameter show lower value in hypertensive group than in normotensive group. These finding corroborate with studies of other workers (9, 10, 11).In this study it was also found that the birth weight of babies in hypertensive group was lower than normal pregnancy group and feoto-placental weight were directly proportional to birth weight of babies. These finding agreed with the finding of other workers (7, 12). Evers etal 2003 demonstrated that intrauterine foetal death and asphyxia were associated with a relative low placental/ fetal weight ratio again indicating that the decreased surface area for diffusion or increased diffusion distance can lead to late intrauterine foetal death.(3)

In this study the mean number of cotyledons, calcified area and marginal insertion of umbilical cord in the normal pregnant was differ in values when compared to the hypertensive group. The finding may indicate cause or the effect of pregnancy induced hypertension. This concurrent with the findings of Udainia etal 2004(13) who had observed a

similar finding in case of toxemia also Pretouris 1996 reported cases of marginal insertion of umbilical cord in about 42% of cases of pregnancy induced hypertension(14)

On histological observation of placenta the microscopic finding was endothelial proliferation of arteries, hyalinization and calcification were obvious in hypertensive group in comparison to the normal group. This also agreed with the previous studies conducted by DiSalvo 1998, Rath2000(15,16)

The placenta shows several histological abnormalities in maternal diabetes like immaturity and hydroid changes of the chorionic villi , increased fibrinoid necrosis and chorangiosis (3). The histological abnormalities can be found in association with cytomegalovirus, toxoplasmosis , rubella and syphilis. (17) Horn LC etal (2004) they studied the cause of death in 310 consecutive autopsies of intrauterine fetal death they concluded that placenta or umbilical cord was responsible for 62% of intra uterine fetal death (18).We can concluded that hypertensive disorders of pregnancy well reflected on placenta with remarkable changes both macroscopic and microscopic.

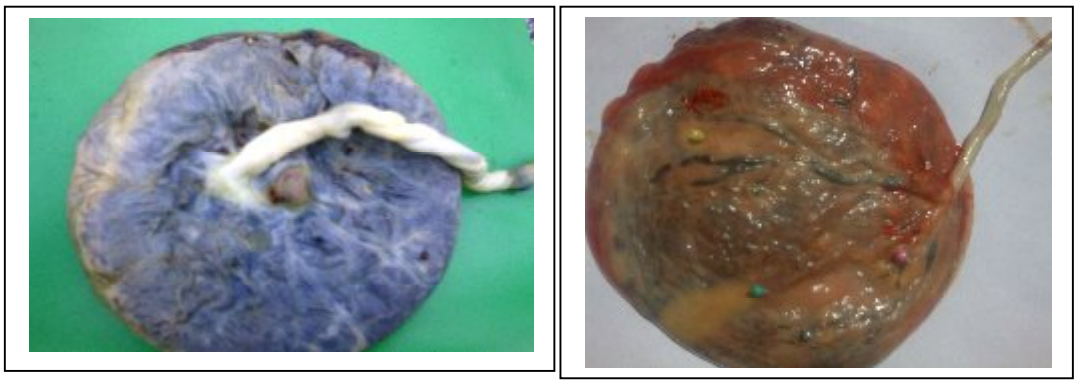

Figure-1: Gross anatomy of the placenta in :

A- normal pregnant mothers with normal umbilical cord insertion .(left)

B- hypertensive pregnant mothers with marginal umbilical cord insertion.(right)

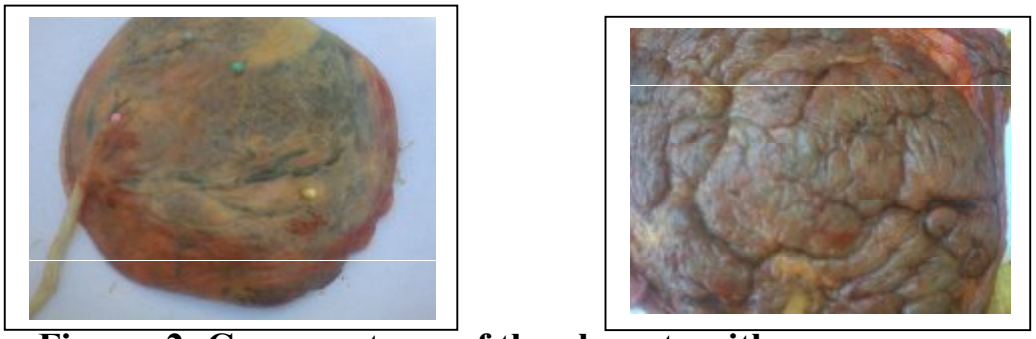

Figure -2: Gross anatomy of the placenta with:
A- Calcified area (left) . B- Cotyledons (right)

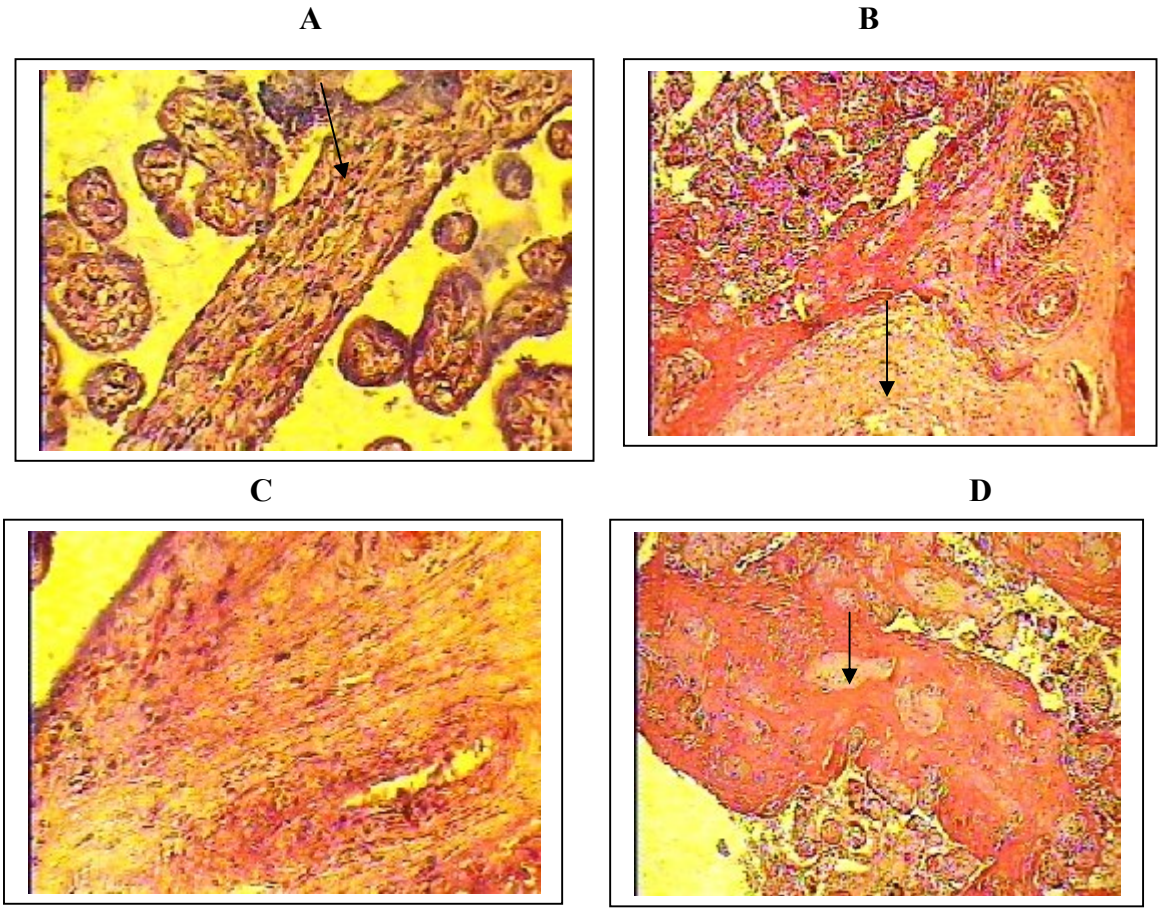

Figure-3: Histological study of placenta in normal and hypertensive group: A- Normal . B-Hayalinised areas. C-Areas of proliferation blood vessels. D- Calcified areas.
(H&E X40)

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