EFFECT OF GESTATIONAL DIABETES AND MATERNAL HYPERTENSION ON GROSS MORPHOLOGY OF PLACENTA

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Background: Gestational diabetes is much more common than pre-existing diabetes i.e. it complicates 2% to 5% of pregnancies. When metabolic control is good, perinatal mortality should be no higher than in general population. However, macrosomia continuous to be a problem in higher than average proportions of such cases. Macrosomia also involves placenta within the chronic hypertensive disease, the most common diagnosis is essential vascular hypertension. **Methods:** Total 60 full term placenta, 20 from normal and 20 each from gestational diabetics and chronic hypertensive mothers were studied grossly. Shape, attachment of umbilical cord, weight, diameter and central thickness of all placentas were noted. **Results:** The study demonstrates that there is change of shape i.e. two lobes in one placenta from diabetic group. All other placentae were singly lobed and discoidal shape with central attachment of umbilical cord to the foetal surface of placenta. Weight central thickness and diameter were significantly greater in diabetic group as compared to normal and hypertensive group. Hypertensive group shows non significant decrease in weight of placentae while there was no change in central thickness and diameter of placenta in hypertensive than the normal group. **Conclusions:** On the basis of results of present study, it is concluded that diabetic's placentae showed increase in weight, central thickness and diameter. One out of 20 placentae in diabetic group also showed change of shape and attachment of umbilical cord to one love. Hypertensive's placentae showed no significant change in weight, shape central thickness and attachment of umbilical cord when compared with normal group.

Key words: Placenta, Gestational diabetes, Maternal Hypertension.

INTRODUCTION

At term human placenta is flattened mass with approximately circular or oval outline, but the shape is determined by the form of patch of villi finally left on chorionic sac.[4] Metabolic diseases associated with pregnancy, like hypertension and diabetes are highly common in low socioeconomic groups.[5] These diseases can affect tissue components of placenta like connective tissue in the core of chorionic villi and basement membrane of trophobastic epithelium lining the chorionic villi. During the first half of pregnancy, the placenta not only increases its surface area but reaches its maximum.[6] This accompanies increase in size, length and complexity of branching of villous stems. In the later half of pregnancy the placenta further increases its surface area, doubling its diameter, the overall thickness remaining static.[7] In gestational diabetes, when metabolic control is good, perinatal mortality should be no higher than in the general population.[2] However, macrosomia continues to be a problem in higher than average proportion of such cases. Foetal hyper-insulinemia is the cause of macrosomia. Even mild disturbances of maternal carbohydrate metabolism can lead to foetal hyperinsulinemia. Within the chronic hypertensive disease, the most common diagnosis is essential vascular hypertension[3] for which there is no known cause. There are other causes for chronic hypertension, however, these are less prevalent in young women.[1] Chronic hypertension in pregnancy is diagnosed if there is sustained elevation of blood pressure greater than 140/90 mm Hg prior to the twelfth week of gestation. If the patient has a superimposed pre-eclampsia, this is one of the most severe problems that mother and fetus will encounter.

This study was carried out to look for the morphological changes of placenta in the mothers suffering with gestational diabetes or hypertension.

MATERIAL AND METHODS

In this study 60 term placentae of male babies divided into three groups (detail follows) were studied. The samples were collected during a 6 months period from the department of obstetrics and gynecology unit-1, Jinnah postgraduate medical centre, Karachi. The placentae were preserved in 10% formalin. All subjects included in this study were

healthy looking multiparous mothers aged between 25–35 years. There were no racial, cultural or environmental differences among the subjects. Heights and weights of all the subjects were comparable. Subjects with abruptio placentae, twins and jaundice were excluded from this study. All placentae obtained were of normal vaginal deliveries.

Group A: In this group those 20 placentae from pregnancies, which were not complicated by any disease, were included.

Group B: In this group, 20 placentae from mothers suffering from mild to moderate chronic hypertension were used.

Group C: In this group 20 full term placentae from mothers suffering from gestational diabetes were used.

Placentae of each group A, B and C were studied macroscopically. Gross features of placentae were noted and compared statistically where possible. These features included, shape, site of attachment of umbilical cord, central thickness (in centimeter, diameters) in centimeters and weights (in grams) of placentae included in this study.

RESULTS

A. Shape of Placentae: The placentae were roughly oval or rounded in shape in all groups except one placenta in group C, which was bilobed in which large lobe was rounded and small conical in shape as show in figure-1.

Fig-1 Normal Placenta

B. Attachment of umbilical cord: All the placentae showed central attachment of umbilical cord on the fetal surface of placentae as shown in figure-2.

Fig-2: Diabetics Placenta

C. Central Thickness: Central thickness of all the placentae was measured in centimeters. The values for all the groups are given in table-1. Mean central thickness of group B when compared with group-A (control) the difference was not statistically significant, however in case of comparison of Group C with group A the thickness was significantly more (P<0.001).

D. Diameter of placentae: Mean diameter of placentae in all groups is given in table-2. There was non significant difference between group A and B, while mean diameter of placentae in group C was significantly more in group C as compared with both the other groups (P<0.001).

E. Weights of placentae: The mean weight of placentae in each group is given in table-3. the difference between groups A and B was statistically non significant, while the weight of group C placentae was significantly more than both the other groups (P<0.001).

Table-1: Central Thickness (cm)

Parameter	Group A	Group B	Group C
Central thickness	2.15 ± 0.16	2.72 ± 0.16	3.98 ± 0.18

AVB=>0.05=N.S, AVC=<0.001=H.S, BVC=<0.001=H.s

Table-2: Diameter (cm)

Parameter	Group A	Group B	Group C
Diameter	14.26 ± 00.19	14.32 ± 00.32	18.95 ± 0.32

AVB=>0.05=N.S, AVC=<0.001=H.S, BVC=<0.001=H.s

Table-3: Weight of the placentae (gms)

Parameter	Group A	Group B	Group C
Weight	237 ± 10.08	532 ± 10.09	656 ± 19.14

AVB=>0.05=N.S, AVC=<0.001=H.S, BVC=<0.001=H.s

Statistical analysis shows highly significant increase in central thickness, diameter and weight of placentae in group C when compared with group A and B.

DISCUSSION

Placenta is an essential organ for exchange of nutrients and metabolites between mother and fetus.[8] Attachment of umbilical cord is normally at the centre of the fetal side of placenta. Thickness of placenta depends upon the length of stem villi.[3] The growth of placenta occurs in two phases, hyperplasia followed by hypertrophy.[9-11]

As all the subjects were apparently healthy and there was no evidence of maternal malnutrition. The haemoglobin level was about 10gm/dl in all the subjects included in this study. This may be the reason of normal shape and central attachment of umbilical cord in all placentae included in this study. Only in severe maternal malnutrition, abnormal shape and eccentric attachment of umbilical cord has been reported by previous workers.[12] These results are similar to the work of Laga and associates[13] who demonstrated that the placentae of malnourished mothers had anatomic, morphologic and histologic alterations. Regarding the size (weight, diameter, central thickness) a highly significant increase in weight (22%), diameter (33%) and ventral thickness (85%) was found in placentae of diabetic mothers when compared with normal placentae.

The weight gain in diabetic's placentae may be attributed to macrosomia and compensatory hyperplasia. Macrosomia affects the affects the fetus and fetal part off placenta, i.e. chorionic plate and all types of villi. This macrosmia may be attributed to fetal hyper insulenemia in response to hyperglycemia in fetuses of diabetic mothers.[7]

Due to macrosomia affecting the fetal part of placenta, the weight, diameter and central thickness of placentae in diabetic mothers increases as compared to normal placenta. Our results coincide with the work of Coustan[14] who reported that diabetic placentae were affected by generalized macrosomia of fetus observed in gestational diabetes.

The compensatory hyperplasia of terminal chorionic villi may be due to low pO_2 (partial pressure of oxygen) in chorionic villous blood. This low pO_2 resulted from reduced amount of maternal blood flow to the intervillous space as diabetics develop endartritis resulting in endothelial cells damage followed by proliferation which may lead to narrowing of the lumen of maternal blood vessels reducing the utero placental blood flow.[15] Another cause of low pO_2 in chorionic villous blood may be the excessive thickness of placental barrier.

Fetal hyperglycemia may so derange the osmotic environment that injury or cell death results.[6] This process involves the endothelial cells of capillaries. The damaged endothelial cells may be replaced without subsequent removal of old basal lamina.[16] New endothelial cells synthesize their own basal lamina leading to excessive thickness of basal lamina of fetal capillaries in chorionic villi. The basal lamina of chorionic capillaries is the part of placental barrier, so its thickness will increase the whole thickness of placental barrier which may lead to reduced transport of oxygen and other nutrients across the barrier. In response to this low pO_2 the terminal villi showed hyperplasia which may be partially responsible for increase in weight of placentae in diabetic group. Our observation is in agreement with the work of Salvatore[17] who observed continued branching of terminal villi in cases of prolonged ischemia.

In case of maternal hypertension, atherosclerosis affects the uterine blood vessels, narrowing their lumen which leads to reduced blood flow at the inter villous space.[17] Apoptosis, a physiological as well as pathological cell death increases in hypoxic conditions. In response to this hypoxia villi showed compensatory hyperplasia but more elimination of parenchymal cells by apoptosis. These parenchymal cells later on were replaced by fibrous tissue. These two processes, i.e. apoptosis and compensatory hyperplasia run side by side and balance each other in such a way that no significant difference in weight, diameter and central thickness of placentae in hypertensive group was found when compared with normal placentae.

Our results are in agreement with the results of Karlsson and associates[18] who reported similar observations in their study in albino vistar rats with established renal hypertension.

CONCLUSION

Clinically the adverse effects of diabetes and hypertension on the outcome of pregnancy are well established but we have seen their gross morphological impacts on placenta. Significant changes in gross morphology have been observed in hypertensive and diabetics. The shape of all placentae in the present study was found rounded except one placenta in group C which was bilobed in which large lobe was rounded and the smaller conical in shape. The observation reveals that maternal hypertension and diabetes have no effect on chorion frundosum which determines the shape of placenta. The villi on chorion frundosum survive due to adequate blood supply while on chorion leave they disappear due to ischemia produced by expansion of chorion. The attachment of umbilical cord was found at the centre of fetal side of placenta in all groups, which reflects that maternal hypertension and diabetes have no effect on the site of attachment of umbilical cord on placenta.

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