Gestational Diabetes: Effect on gross Morphology of Human Placenta and Birth Weight

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ABSTRACT

Aim: To evaluate the effect of gestational diabetes on normal morphology of human placenta and birth weight.

Study design: Case control prospective study

Place of study: Department of Anatomy, Bahria Medical and Dental College, Karachi.

Methods: Total of 50 placentae were collected, 25 were normal placentae and 25 were gestational diabetic placentae.

Results: In this study gestational diabetes showed no effect on shape in normal and gestational placentae where as changes in the weight of placentae, diameter of placentae, thickness of placentae, number of cotyledons and birth weight was observed in gestational diabetic placentae.

Conclusion: The results in our study revealed that gestational diabetic placentae showed increase in diameter, weight, mean thickness and number of cotyledons. There was also increase in birth weight in gestational diabetes that revealed that good glycemic control might be a better option for reducing the diabetic induced changes.

Keywords: Human placenta, gestational diabetes, birth weight

INTRODUCTION

Gestational Diabetes, the term first applied in 1950’s was thought to be a transient condition that affected fetal outcomes adversely and then disappear after delivery. According to International Association of Diabetes, Gestational Diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset or first recognition during pregnancy.

Racial differences in population influence the disease prevalence and perinatal outcome in GDM. The overall prevalence of GDM in U.S.A was 3.5 out of 100 cases. The risk % of GDM of Pakistani population from 1995-2005 was 16.2.

Congenital anomalies are the most important cause of perinatal deaths in pregnancies complicated by Diabetes mellitus. Central nervous system anomalies and cardiovascular diseases are the most common. The pathogenesis of these anomalies is still unclear. Increased risk of spontaneous abortions and malformations correlates with diabetes-complicated pregnancies.

Maternal clinical complications associated with diabetic pregnancies include hypoglycaemia, nephropathy, diabetic ketoacidosis, preeclampsia, recurrent urinary infection, premature labour and polyhydramnios. Silva et al in 2003 demonstrated that gestational diabetes correlates with increased risk of future diabetes type 2. Placenta is a fetal organ, essential for growth and development of fetus. It is located at the interface between the maternal and fetal circulation, which is in contact through different circulation, i.e., syncytiotrophoblast exposes the placenta to the maternal circulation and the endothelium in contact with fetal blood. Owing to its position placenta is exposed to regulatory influence of hormones, cytokines, growth factors and substrate present in both circulation and, hence, may be affected by changes in any of these factors.

Placenta acts as fetomaternal organ and it has 2 components. A fetal part: that develops from chorionic sac. A maternal part: that develops from the endometrium also known as Decidua basalis. Chorionic plate is present on the fetal side. Decidua refers to that part of endometrium in a gravid uterus that separates from the remainder of the uterus after parturition. Maternal surface of human placenta is opaque, with 15-20 slightly elevated areas called cotyledons. These cotyledons have a covering of Decidua basalis. The septa extend and form grooves between the cotyledons. The fetal surface (amniotic surface) has the glossy appearance because of the surface covered by Amnion. The umbilical cord is usually attached near the centre of the fetal surface. Umbilical cord consists of two arteries and one vein. Umbilical vessels radiate out at the point of umbilical attachment, followed by veins which are deeper and larger.
MATERIAL AND METHODS

This study was approved by the ethical committee of JPMC, Karachi and was conducted in the Department of Anatomy Bahria University Medical and Dental College, Karachi. Total numbers of 50 full term human placentae were used in this study. Inclusion criteria for the subjects included registered cases of Gestational diabetes, parity 0 – 4, maternal age (25 – 35) years, gestational age (36 – 42) weeks and all cases were through cesarean Sections. Exclusion criteria included unregistered cases, known diabetes (Type 1 diabetes), smokers, renal disease, pre-eclampsia, cardiovascular disease, multiple pregnancies.

Placentae in this study were divided into two groups. Group-A (Normal full term placentae) included 25 placentae from pregnancies which were not complicated by any disease. Group –B (Gestational Diabetes full term placentae) in which 25 placentae from mothers suffering from gestational diabetes were included. Placentae of group-A and group-B were studied for macroscopic changes along with birth weight of newborn. All the data was noted and compared statistically, where possible.

In this study the data was stored and analyzed using SPSS version 16.0. Mean and standard deviation was reported for following parameters. i.e. shape of placentae, shape of placentae, diameter of placentae, mean thickness of placentae, number of cotyledons and birth weight. To compare the differences of mean between two groups, independent sample t-test was used, and decision of significance was made if p-value found less than or equal 0.05.

RESULTS

The shape of the placentae was roughly oval or rounded in both group-A and group-B included in our study (as shown in photograph 1 and 3), except for one placenta which is bilobed (as shown in photograph 2). In this bilobed placenta large lobe was rounded and small lobe was conical in shape.

Weight of the placentae in both the groups was measured in grams. The mean value of the weight of placentae in group-A was 527.20±15.698 grams as shown in table 1. The mean value of the weight of placentae in group-B was 582.56 ± 28.61 grams as shown in table 1. When we compared the mean value of weight of placentae in both the groups, the mean weight of the placentae was increased in group-B which was highly significant (P< 0.001).

Mean thickness of placentae in both the groups was measured in millimetres. The mean value of thickness of placentae in group-A was 13.46±1.81 mms as shown in table 2, whereas the mean value of the thickness of placentae in group-B was 22.22± 0.80 mms as shown in table 2. When we compared the two groups that is group-A with group-B, the mean thickness of the placentae in group-B was increased which was highly significant (P<0.001).

Cotyledons were counted in both group A and B. Counting was started from left side of one end and going through right ward in spiral manner. In group-A the mean number of cotyledons was 17.88±1.66 as shown in table 2. In group- B, the mean number of cotyledons was 22.56±1.98 as shown in table 2. When we compared the two groups, mean value of number of cotyledons was increased in group-B which was highly significant (P< 0.001) as shown in table 2.

The mean birth rate was measured in kilograms. In group-A, the mean value of birth weight was 2.58 ± 0.30 kgs as shown in table 1, whereas when we measured the mean value of birth weight in group-B it was 3.26 ±0.29 kgs as shown in table 1. Birth weight was compared between group-A and group-B in which mean value of birth weight of group-B was increased and was highly significant (P < 0.001) as shown in table 1.

Photograph 1: A photograph of normal full term human placenta of group-A showing round shape placenta with amnion (A), chorionic vessels (C) and central insertion of cord (UC).
Photograph 2: A photograph of gestational diabetic full term human placenta of group-B showing two lobes (large rounded and small conical) showing eccentric attachment of cord (UC) with amnion (A) and chorionic vessels (C).

Table 1: Comparison of weight of placentae and birth weight in group-A and group-B

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group-A</th>
<th></th>
<th>Group-B</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>St. Dev.</td>
<td>Mean</td>
<td>St. Dev.</td>
</tr>
<tr>
<td>Weight of placentae (gm)</td>
<td>527.20</td>
<td>15.69</td>
<td>582.56</td>
<td>28.61</td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>2.58</td>
<td>0.30</td>
<td>3.26</td>
<td>0.29</td>
</tr>
</tbody>
</table>

P value in comparison of group A and group B

Table 2: Comparison of diameter of placentae, mean thickness of placentae and number of cotyledons in group-A and group-B

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group-A</th>
<th></th>
<th>Group-B</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>St. Dev.</td>
<td>Mean</td>
<td>St. Dev.</td>
</tr>
<tr>
<td>Diameter of Placenta (cm)</td>
<td>15.94</td>
<td>1.43</td>
<td>19.60</td>
<td>0.94</td>
</tr>
<tr>
<td>Mean Thickness of placenta (mm)</td>
<td>13.46</td>
<td>1.81</td>
<td>22.22</td>
<td>0.80</td>
</tr>
<tr>
<td>Number of Cotyledons</td>
<td>17.88</td>
<td>1.66</td>
<td>22.56</td>
<td>1.98</td>
</tr>
</tbody>
</table>

P value in comparison of group-A and group-B

DISCUSSION

The placenta is a complex fetal organ that fulfills pleiotropic roles during fetal growth. It separates the maternal and fetal circulation with which it is in contact through different surfaces i.e. the syncytiotrophoblast exposes the placenta to the maternal circulation and the endothelium is in contact with fetal blood. Because of this unique position, the placenta is exposed to regulatory influence of hormones, cytokines, growth factors and substrates present in both circulations and hence may be affected by changes in any of these. In turn, it can produce molecules that effect mother and fetus independently.

Placenta of diabetic pregnancies showed several alterations including increased weight and size (Molteni et al., 1978). Shape of the placenta in our study was observed rounded to roughly oval. We found no difference in the shape of group-A controlled placentae and group-B gestational diabetes placentae; however, in Group B, we found 1 bilobed placenta. As pregnancy advances villi on the embryonic pole continues to grow and expand, giving rise to chorionic frondosum. Chorionic frondosum shaped like a disc which resulted in the term discoid placenta (Heide, 2007). Similar findings were observed by Ashfaq et al in 2005 in Pakistan which they found oval to rounded shape of placentae in normal and gestational diabetes. Panuganti et al in 2012 in their study, observed discoid shaped placentae in normal cases.
Placentae from diabetic mother tend to be heavier than the normal and complicated pregnancies at the same gestational age. Significant accumulation of nonparenchymal tissue and only moderate increase in parenchymal tissue results in the increase in the weight of placentae in gestational diabetes mellitus. In our study, we observed highly significant increase in the diameter and thickness of placentae of group-B gestational diabetes than in group-A normal placentae and significant increase in the weight of the placentae of group-B gestational diabetes as compared to group-A normal placentae. Our results coincided with the results of Akhter et al in 2010 in Bangladesh reported increased weight, thickness and diameter of placentae in gestational diabetic group as compared to the normal group.

Significantly increased diameter of the placentae in diabetic mothers may correlate with their increased volume and increased number of cotyledons as compared to the controlled group. In our study we observed highly significant increase in the number of cotyledons in group-B gestational diabetes when compared with the group-A controlled normal placentae. This could be reactive hyperglycaemia that occurs in the fetuses of diabetic mothers. This results in fetal macrosomia and compensatory hyperplasia in placentae which ended up in increase in the size of placentae in diabetic mothers. Our results coincided with the results of Boyd et al in 1986, Khaskhelli et al in 2013, and Mayhew et al in 1993 who also observed increase in the size of placentae (weight, diameter, central thickness and number of cotyledons) in diabetic mothers as compared to normal placentae. Our study also coincided with the study conducted by Sudha et al in India in 2012 who observed significant reduction in the placental weight of complicated pregnancies except for gestational diabetes mellitus, when compared with uncomplicated pregnancies.

It is necessary to have good metabolic control to prevent perinatal mortality. Its alteration results in higher rate of birth injuries and cesarean sections due to macrosomia. In our study we observed highly significant increase in the birth weight in group-B gestational diabetes as compared to group-A control placentae. This increase in birth weight explains the correlation of hyperinsulinism due to diabetes mellitus with macrosomia. Diabetic insults at the beginning of gestation as in many pregestational diabetic pregnancies may have long term effects on placental development. These adaptive responses of the placenta to the diabetic environment such as buffering excess maternal glucose or increased vascular resistance may help limit fetal growth within normal range. If duration or extent of the diabetic insult including maternal hyperglycemia, hyperinsulinemia or dyslipidemia exceeds the placental capacity to mount adequate response than excessive fetal growth may ensure. Our study correlated with the study of Jauniaux et al in 2006, Kucuk et al in 2009, and Khaskhelli et al in 2013 who all observed increase in the mean birth weight of gestational diabetes and mentioned that there was a direct proportion of placental weight with birth weight. In contrast to our findings Gillman et al in 2003 observed no significant difference in the birth weight of gestational diabetes and non gestational diabetes.

CONCLUSION

In this study significant changes in the gross features have been observed between normal and gestational diabetic placentae. This included increase in the diameter, weight, mean thickness and number of cotyledons in the placentae. There was also increase in the birth weight that revealed that good glycemic control is necessary to maintain the normal architecture of placentae.

REFERENCES