

# A Study on Morphohistometric Changes of Placenta in Gestational Diabetes- A Comparative Analysis

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## Abstract

**Introduction:** The placenta is a complex fetal organ that fulfills pleiotrophic roles during fetal growth. Placenta is the most accurate record of the infant's prenatal experience. Gestational diabetes is much common than preexisting diabetes .i.e. it complicates 2% to 5% of pregnancies. It seems reasonable to expect that biochemical changes occurring in the pregnant women with diabetes should be reflected in the placental structure. Aim & objectives: In the present study an attempt is made to know the morphological changes of placenta in gestational diabetes mellitus. **Subjects and Methods:** In this study totally 60 placentae were studied, of which 30 were nondiabetic placentae and 30 from Gestational Diabetic mothers were studied macroscopically. Morphologically the shape, site of attachment of umbilical cord, central thickness of placenta was noted. Birth weight of the neonate and the placental ratio were also recorded. By using routine staining techniques and direct microscopy tissues of Gestational Diabetic placenta were studied qualitatively and compared them with normal placenta. **Results:** Our study demonstrates that there is a significant increase in weight and central thickness of placenta. Neonatal weight and placental ratio were also increased; there was no change in shape and site of attachment of umbilical cord in case of diabetic placenta when compared to normal. **Conclusion:** On the basis of results of present study it is concluded that diabetic placentae showed increase in weight and central thickness. Neonatal weight and placental ratio were also increased. These findings indicate that control of hyperglycemia only partially prevents the development of placental abnormalities.

**Keywords:** Placenta; Gestational Diabetes; Neonatal weight, Placental ratio.

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## Introduction

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.<sup>[1]</sup>

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 the chorionic sac. Metabolic diseases associated with pregnancy such as hypertension and diabetes can affect tissue components of placenta like connective tissue in the core of chorionic villi and the basement membrane of trophoblastic epithelium lining the chorionic villi.<sup>[2]</sup>

During first half of pregnancy, the placenta increases in surface area and reaches its maximum. This accompanies increase in size, length and complexity of branching of villous stems.<sup>[3]</sup> In the later half of pregnancy the placenta further increases in surface area, doubling its diameter, the overall thickness remaining static. Thus development of the

placenta precedes fetal development and growth, the latter being more pronounced in the third trimester.<sup>[4]</sup>

Diabetes is a major health concern in our society. Prevalence of diabetes among women of child bearing age is increasing due to more sedentary life styles, changes in diet and virtual epidemic of childhood and adolescent obesity.<sup>[5]</sup>

Gestational diabetes mellitus is defined as glucose intolerance of variable degree with onset during pregnancy. Abnormal maternal glucose tolerance occurs in 3-10% pregnancies.<sup>[6]</sup>

The placentae of the diabetic woman has attracted much interest largely because it is thought that placental damage may be partially responsible for the unduly high incidence of fetal complications that occur in pregnancies complicated by diabetes mellitus. Previous studies on functional morphology of placentae from diabetic mothers have produced inconsistent results and conclusions.<sup>[7]</sup> Studies of placenta from diabetic mothers may yield a moderately consistent pattern of abnormalities at microscopic level which are characteristic.

In this study an attempt was made to study the morphological changes of placenta in gestational diabetes which can explain the high fetal jeopardy.

Hence in the present study, to identify the morphological variations of placenta like shape, weight, attachment of umbilical cord and its correlations to the neonatal weight changes in normal pregnancy and in Gestational diabetes.

**Subjects and Methods**

Type of Study: - Cross sectional Study

The total number of specimens used in the present study was 60 placentae, 30 from mothers with uncomplicated pregnancies as control group and 30 from mothers with Gestational diabetes were considered as a study group. The study was conducted in the Department of Anatomy, Dr VRK and Shadan Institute Medical Sciences, Hyderabad, from January 2019 to December 2019.

Inclusion criteria: - mothers in the age group of 20 to 39 years.

Exclusion criteria:-Mothers with type 1 diabetes mellitus, with combined diabetes and hypertension, positive VDRL, with severe anemia were excluded from the study.

The gestational age and fetal weight were taken from the case sheets. Materials consisted of –



**Figure 1: Instruments used in the study.**

The cases were studied dividing into two experimental groups.

Group A – 30 placentae from pregnancies not complicated by Gestational Diabetes. Group B – 30 placentae from mothers with Gestational Diabetes.

**Collection and Examination of placenta:-**

The placenta with attached membranes and umbilical cord was collected soon after delivery washed in running tap water to clean all blood.

Maternal surface was inspected for its completeness, calcification and infarction. The shape of placenta was ascertained by stretching it flat on the cutting board. Umbilical cord was examined for its site of attachment to the fetal surface. The membranes were cut off the edge of placenta with the knife. The placenta was then weighed in weighing machine graduated in grams. Thickness of placenta was measured by piercing a needle through the centre of placenta.

Placenta was cut into 2 cm vertical sections from maternal to fetal surface to ensure adequate fixation. Sections were collected in tagged containers with 10% formalin and taken to the department of Anatomy for histological examination.

**Results**

Present study included 60 placentas of which 30 were from Gestational Diabetic mothers and 30 were from non diabetic mothers. The study included women with an age group ranging from 20 to 40.

**Table 1: Total Number of Patients**

Groups	Total No of Patients
Normal (Group A)	30
GDM(GROUP B)	30
Total	60

Majority of normal cases were in the age group of 20-24 about 10 cases. About 8 cases were in the age group of 25-30 years of age. 8 cases were in the age group of 30- 35. only 4 cases were above the age of 35.

**Table 2: Age distribution of the Normal patients**

Age distribution	No. Of cases
20-24	10
25-30	8
30-35	8
>35	4

**Table 3: Age distribution of the Diabetic patients**

Age distribution	No. Of cases
20-24	2
25-30	14
30-35	12
>35	2

The age range for mothers with gestational diabetes was 25–39 years. The majority of mothers were 25–30 years. 14 cases were in this age group. 12 cases were in the age group of 30-35 years. Only 2 cases were in the age group of 20-24 years.

**Table 4: Placental weight in grams**

Groups	GDM	Normal	Total
<500	10	25	35
500	4	4	8
>500	16	1	17
Total	30	30	60

It is seen from [Table 4], that majority of placentae from gestational diabetes weighed more than 500 grams. The maximum weight recorded was 870 grams. Majority of normal placentae weighed less than 500 grams.

Overall incidence of shapes of placenta encountered in the study is shown in [Table 5].

**Table 5: Shape of the Placenta.**

Groups	Round	Oval
GDM	16	14
Normal	14	16
Total	30	30

The majority of placenta in the present study were oval in shape about 53.33% and 46.66% were round in shape. Majority of diabetic placentae were round in shape whereas majority of normal placenta were oval in shape.

The majority of placenta in the present study were oval in shape about 52.5% and 47.5% were round in shape. Majority

of diabetic placentae were round in shape whereas majority of normal placenta were oval in shape.

**Table 6: Mode of attachment of umbilical cord**

Groups	Central	Eccentric
GDM	20	10
Normal	22	08
Total	42	18

The above table shows that the most common mode of cord insertion is central accounting for about 84% followed by eccentric insertion of about 36% of total cases. Velamentous or battle dore insertion were not found in the present study.

**Table 7: Comparison of gross morphological parameters of diabetic placenta with reference to normal**

	Normal (Mean±SD)	DM (Mean±SD)	Significance P-Value
Baby weight	2.38 ± 0.54	3.02 ± 0.5	<0.001
Placental Weight	410.40 ± 57.45	537.28 ± 121.72	<0.001
Placental Central Thickness	1.79 ± 0.49	3.00 ± 0.33	<0.001
Placental ratio	165.03 ± 40.47	185.27 ± 36.64	<0.5

There was significant difference in baby weight among normal and Gestational diabetic group. The mean baby weight in normal pregnancies were 2.38kg whereas in gestational diabetic pregnancies were 3.02 kg. The difference between case and control group were statistically significant (p<0.001)

The mean placental weight in control group were 410.40grams whereas in diabetic placentae, mean placental weight was 537.28 grams. The difference between two groups were statically significant (p<0.001)

Placental Ratio is the ratio of placental weight to fetal weight. The mean placental ratio in control group was 165.03 compared to study group was 185.27.the difference between two values was found to be statistically significant (p<0.5).

**Table 8: Percentage variation of parameters**

	Normal	DM	Percentage variation
Baby weight	2.38	3.02	16.66
Placental Weight	410.50	537.28	22.09
Placental central thickness	1.79	3.00	35.05
Placental ratio	165.03	185.27	2.70

The above table shows the percentage variation between normal and Gestational Diabetic group. The baby weight shows a percentage variation of 16.66. Placental weight shows a variation of 22.09. Percentage variation between study and control group in case of placental central thickness and placental ratio was 35.05 and 2.70 respectively.

## 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 the 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 will affect mother and fetus independently.<sup>[1]</sup>

Abnormalities of glucose metabolism, such as gestational diabetes and overt insulin dependent diabetes, are among the most common medical complications of pregnancy. Reports regarding the pathology of placenta in gestational diabetes are numerous and often contradictory. The longitudinal alteration in the maternal metabolism during pregnancy with normal glucose tolerance, provide both maternal and fetoplacental growth and energy requirements. There are major adaptations the maternal metabolism throughout pregnancy that results in increased availability of fat stores in early pregnancy and increased availability of carbohydrate and protein in late pregnancy. In female with diabetes, there is previous abnormality in carbohydrate metabolism and possibly protein and lipid metabolism. These biochemical changes occurring during pregnancy would reflect on placental structure.<sup>[8]</sup>

In the present study total of 60 placentas were studied which included 30 from gestational diabetic mothers considered as study group and 30 from nondiabetic mothers considered as control group. In the present study, complicating factors such as superimposed hypertension and other associated complications were excluded and the degree of control of diabetes was considered to be excellent.

### Age of the mother

Impaired glucose tolerance is usually more prevalent than diabetes in women of child bearing age. Maternal age, overweight and parity all predispose to gestational diabetes. Incidence of Gestational diabetes is low in the absence of risk factors.<sup>[9,10]</sup>

Emmanuel Odar studied that pregnancy is a diabetogenic state manifested by insulin resistance and hyperglycaemia. The age group at risk of getting gestational diabetes in this study was between 20–39 years.<sup>[11]</sup>

In the present study, the age range of mothers with gestational diabetes was 25 to 39 years with mean age of 32 years.

### Weight of the placenta

In the present study, the mean placental weight in control group was 410.40 grams whereas in Gestational diabetes the mean placental weight was 537.28 grams. The differences between two groups were statistically significant. Placental weight between two groups showed a percentage variation of 22.09.

Parameter	Present study	Muhammad Ashfaq (12)
Mean placental weight	537.28	656

Fetal hyperglycemia may so derange the osmotic

environment that injury or cell death results. This process involves endothelial cells of capillaries.

The damaged endothelial cells may be replaced with subsequent removal of old basal lamina. New endothelial cells synthesize their own basal lamina leading to excessive thickness of basal lamina of fetal capillaries in the 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 oxygen transport the terminal villi showed hyperplasia which may be partially responsible for increase in weight of placenta in diabetic group.<sup>[3]</sup>

Teasdale stated that a significant accumulation of non-parenchymal tissue and only a moderate increase in parenchymal tissue may be the cause of heavier placenta in gestational Diabetes.<sup>[7]</sup>

The weight gain in diabetic's placenta may be attributed to macrosomia and compensatory hyperplasia. Macrosomia affects the fetus and fetal part of placenta i.e. chorionic plate and all types of villi. This macrosomia may be attributed to fetal hyperinsulinemia in response to hyperglycemia in fetuses of diabetic mothers.<sup>[4]</sup>

**Neonatal Weight**

Variable	Present study(n=30)	Sanjoy kumar2010(n=35) (13)
Mean neonatal weight	3.02+_0.5	3.26+_0.40

The present study showed that neonatal weight increases with Gestational Diabetes Mellitus. The maximum recorded neonatal weight in normal pregnancy was 3200 grams whereas in Gestational Diabetic pregnancy, maximum neonatal weight recorded was 3900 grams. The neonatal weight in GDM with p value less than 0.001 is highly significant statistically.

According to Driscoll in 1965, Glucose passes the placenta readily and the fetus responds to hyperglycemia with hyperplasia of islet of Langerhans and increased insulin secretion, the primary reason for fetal overweight in maternal diabetes.<sup>[14]</sup>

**Placental Ratio**

The present study showed mean placental ratio in control group was 165.03 compared to study group was 185.27. the difference between two values was found to be statistically significant (p<0.5).

The study findings are in correlations with the study conducted by Lao. TT in 1997 which showed increased placental ratio in Gestational Diabetic groups compared to control group. He concluded that gestational diabetes mellitus is the production of placental hormones that leads to maternal insulin resistance, and the placental size has been shown to be increased both in gestational and pre-gestational diabetic pregnancies. But an increased placental size could be related to the concomitant increase in fetal size, and it is not clear if a disproportionally bigger placenta is found in gestational diabetes mellitus.<sup>[15]</sup>

An increased placental ratio represents an adaptive process

by the feto-placental unit in an unfavorable maternal environment. When there is a limitation imposed on fetal growth velocity due to nutritional deficiencies, the placenta may undergo hypertrophy in an attempt to compensate. An increased placental ratio would be a sign of fetal growth disturbance.<sup>[15]</sup>

**Shape of Placenta**

The present study showed that majority of placenta in both study and control group were oval in shape. As all subjects were apparently healthy and there was no evidence of malnutrition, may be the cause for normal shape of the placenta.

According to study conducted by Muhammad Ashfaq in 2005, shape of placenta in diabetic and non diabetic groups were roughly oval or round in shape except one placenta which was bilobed.<sup>[12]</sup>

Laga and associates in 1972 demonstrated that the placenta of malnourished mothers had anatomic, morphologic and histologic alterations.<sup>[16]</sup>

Hamilton in 1951 showed that term placenta is circular or oval in outline and is determined by the form of villi finally left on chorionic sac.<sup>[17]</sup>

**Site of Attachment of Umbilical Cord**

In the present study majority of cases showed central insertion, 70% in both control and study group and eccentric insertion of cord was found in 30%

**Placental Central Thickness**

In the present study, the placental central thickness in GDM Group was 3.00±0.33 compared to normal cases which were 1.79±0.49 and the differences between two groups were statistically significant.

Central thickness	Present study	Muhammed Ashfaq (59)
GDM	3.00±0.33	3.98±0.18
Control Group	1.79±0.49	2.15±0.16

It was also reported that thickness of placenta depends on the length of stem villi.<sup>[18]</sup>

The results also coincide with the work of Couston who reported that Diabetic placenta were affected by generalized macrosomia of fetus observed in Gestational Diabetes.<sup>[19]</sup>

**Conclusion**

The present study reveals that the incidence of Gestational Diabetes mellitus is more common in the age group of 25 to 30 years. The shape and cord insertion did not show significant variation.

The morphometry of placental weight and central thickness was significantly higher in the study group compared to normal control group. The birth weight and placental ratio was also increased in GDM group.

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## References

1. Desoye G, Sylvie H. The human placenta in gestational diabetes. *Diabetes care* 2007; 30(2):120-26
2. Pinker GD, Robert DWT. A short textbook of gynecology and obstetrics. 8th edn. sydney: Hodder & Stoughton; 1976; 106-7.
3. Kumar V, Cotran S & Robin SL. Basic Pathology. 7th Ed; Pennsylvania: WB Saunders; 2000; 1082-4.
4. Queenan J T. Management of high risk pregnancy. 4th ed; England: Blackwell science; 1999; 261-70.
5. Ranjana V, Mishra KS, Mohini J. Cellular changes in the placenta in pregnancies complicated with diabetes. *Int J Morphol.* 2010; 28(1): 259-64.
6. Dutta DC. Medical and surgical illness complicating pregnancy. Text Book of Obstetrics.6th edition. Calcutta: New Central Book Agency; 2004.
7. Teasdale F. Histomorphometry of the placenta of the diabetic woman: Class A diabetes mellitus. *Placenta.*1981; 2: 241-52.
8. Feezko R, Kluber KM. Cytoarchitecture of muscle in genetic model of murine diabetes. *Am J Anat.* 1988;182:224-40.
9. King H. Epidemiology of glucose tolerance and GDM in woman of child bearing age. *Diabetes care* 1998; 21(2):9-13.
10. Amankwah KS, Prentice RL, Fluery FJ. Incidence of gestational diabetes. *Obstet Gynecol* 1977;49:497-498.
11. Odar E, Wandabwa J, Okiondo P. Maternal and fetal outcome of gestational diabetes mellitus in Mulago hospital, Uganda. *Obst J* 2004; 4: 893-9.
12. Ashfaq M, Janjua MZ, Channa MA. Effect of Gestational Diabetes and maternal Hypertension on gross morphology of placenta. *J Ayub Medical College* 2005; 17: 44-7.
13. Chakraborty SM, Yousuf A, Islam S, Banu LA. Impacts of Gestational Diabetes Mellitus on the Mother and the Neonate – a Descriptive Study. *Chittagong. Bangla J Anat* 2010; 8: 64-8.
14. Driscoll, S. The pathology of pregnancy complicated by diabetes mellitus. *Med Clin North Am* 1965; 49:1053-67..
15. Lao TT, Lee CP, Wong WM. Placental weight to birth weight ratio increased in mild gestational glucose tolerance placenta. *Placenta* 1997; 18: 227-30.
16. Laga M, Driscoll SG, Munro HN. Quantitative studies of human placenta. *Biol. Neonate* 1973; 23: 260-83.
17. Hamilton WJ, Boyd JD. Observation on the human placenta. *Proc R Soc Med* 1951; 44(6): 489-96.
18. Moore K.L. The developing Human. Philadelphia: WB Saunders, 1983.
19. Couston DR Gestational Diabetes. In: Management of high risk pregnancy. 4th ed. England: Blackwell science; 1999.

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