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STUDY OF PATHOLOGICAL CHANGES IN PLACENTAS OF GESTATIONAL DIABETES MELLITUS AND ITS ASSOCIATION WITH FETAL OUTCOME

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GDM is associated with an adverse fetal and neonatal outcome that often presents with macrosomia, birth trauma, neonatal hypoglycemia, and respiratory distress syndrome. The inclusion of GDM into 'the great obstetrical syndromes' emphasizes the role of the placenta in interactions between the maternal and fetal unit.

The aim: To study pathological changes in the placentas of gestational diabetes mellitus and its association with fetal outcome.

Materials and methods: *The Prospective study was conducted among pregnant women above the age of 18 years diagnosed with gestational diabetes attending the hospital. All patients are subjected to complete physical examination along with obstetric examination. All the routine investigations, including the complete blood counts, blood picture, RBS, RFT, LFT, OGTT, CUE and, ultrasonography with doppler, histopathological examination of the placenta after delivery.*

Results*: The weight of the babies born to GDM mothers and normal mothers were compared, and the GDM mother's baby weighed higher, meaning diabetes has an effect on the baby's weight which was statistically significant. Comparison of the placenta was made for cases and controls; the result suggested all the morphological parameters placenta - weight ($p<0.001$), diameter ($p<0.001$), area ($p<0.002$) and thickness ($p<0.001$) were statistically significant. The complications reported were respiratory complications, hypoglycemia, hyperbilirubinemia, meconium staining, polycythemia, sepsis and hypocalcemia. Babies of gestational diabetic mothers have a higher risk of developing neonatal complications than non-diabetic mothers. Villous oedema, villous fibrosis, syncytial knots, and fibrinoid necrosis is seen on histopathological examination was <0.05, and hence there was a significant difference between these findings in both the groups.*

Conclusions: *GDM is associated with the adverse fetal and neonatal outcome that often presents with respiratory complications, hypoglycemia, hyperbilirubinemia, meconium staining, polycythemia, sepsis and hypocalcemia. Including GDM into 'the great obstetrical syndromes' emphasizes the role of the placenta in interactions between the maternal and fetal unit*

Keywords: *Gestational diabetes mellitus (GDM), neonatal hypoglycaemia, hypoglycemia, hyperbilirubinemia, meconium staining*

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1. Introduction

The human placenta is the organ responsible for facilitating nutrient uptake, waste elimination, and gas exchange between mother and fetus. The placenta is also a vital source of hormone production, such as progesterone and human chorionic gonadotropin, that maintain the pregnancy.

Consequently, placental dysfunction can lead to several adverse fetal outcomes. Moreover, because the placenta reflects the metabolic milieu of both mother and fetus, it is a valuable tool for studying the metabolic perturbations that may occur during pregnancy, such as diabetes mellitus [1, 2].

The extent to which maternal glycemic control contributes to placental abnormalities remains unclear. However, the literature demonstrates that when maternal glucose levels are well-controlled, the placentas of women affected by diabetes are normal as evaluated by routine light microscopy [3].

However, several studies have identified histopathologic placental abnormalities among women even with well-controlled pre-gestational and gestational diabetes. Moreover, placental abnormalities associated with maternal diabetes have been inconsistently reported in the literature, perhaps reflecting population differences in sample size, glycemic control, study methodology, prenatal care quality or diabetes types [4]. Gestational diabetes mellitus (GDM) is a metabolic disease defined as progressively impaired glucose intolerance with the onset or first recognition during pregnancy (WHO 2013). The prevalence of GDM varies between populations, ranging from 1.7 % to 11.6 % [5].

Numerous studies established that GDM is associated with a significantly higher risk of short- and long-term maternal and fetal complications. Fetuses with intrauterine exposure to hyperglycemia more often present with macrosomia, birth trauma, neonatal hypoglycemia, and respiratory distress syndrome.

Adverse long-term outcomes of hyperglycemia are caused by intrauterine fetal programming and consist of a higher prevalence of metabolic-related diseases. In addition, the development of subsequent type 2 diabetes mellitus and cardiovascular diseases are among widely discussed maternal complications [6].

Gestational diabetes increases the risk for fetal macrosomia and stillbirths, along with the increased frequency of maternal hypertension and caesarean delivery. These complications have been attributed to abnormalities in the placenta. Therefore, the placenta of the diabetic woman has gained much interest, and several placental pathological changes have been described. Placentas from diabetic pregnancies present similar morphological abnormalities independent of the level of glycemic control. The incidence of intra-uterine fetal death and large-for-gestational-age (LGA) infants is still increased in these pregnancies despite the quality of blood glucose control.

Alteration of the placental development and subsequent vascular dysfunction are presented in 6 out of 7 women with all ranges of diabetic severity. The pivotal question is how placental lesions such as villous fibrinoid necrosis, villous immaturity and chorangiosis may affect fetal development. Maternal hyperglycemia directly stimulates metabolic and hormonal changes in the fetus. Increased level of insulin accelerates fetal metabolism and subsequently enhances fetal oxygen demands. Both, placental abnormalities and increased oxygen consumption often lead to chronic fetal hypoxia. In the vast majority of cases, oxygen saturation in the umbilical vein is significantly decreased as compared to non-diabetic pregnancies. Fetal hypoxia tends to increase erythropoiesis by induction of erythropoietin (EPO) secretion. A significantly elevated EPO level in cord blood correlates with enhanced nucleated red blood cell levels. Both of them are suggested as markers of chronic intrauterine fetal hypoxia. Hypoxia is one of the basic triggers for increased angiogenesis**.** This study was undertaken to test this hypothesis by investigating various macroscopic and microscopic features of the placenta and fetal outcomes in pregnancies complicated by gestational diabetes.

The aim: to study pathological changes in placentas of gestational diabetes mellitus and its association with fetal outcome.

2. Materials and Methods

The prospective study was conducted among patients attending the Modern government maternity hospital, Petlaburj, Hyderabad from November 2020 to October 2021**.** Institutional ethical committee approval was obtained prior to the initiation of the study (registration no-TSMC/FMR/00494 dated June 2020)

Ethical clearance and informed consent were obtained from patients. Pregnant women diagnosed with gestational diabetes attending Modern government maternity hospital, Petlaburj, Hyderbad.

Inclusion criteria: Above the age of 18 years **in** pregnant women with gestational diabetes

Exclusion criteria: Pregnant women with overt diabetes, gestational diabetes along with other pregnancy complications such as preeclampsia or hypertension.

All patients diagnosed with gestational diabetes during the study period were included in the study. A total of 150 subjects were taken into the study.

The patients were divided equally; that is 75 gestational diabetics, and 75 non-diabetics pre-designed pretested questionnaire was done.

The subjects were included in the study after their consent sociodemographic details were noted. History will be taken from patients and attendees. Personal history related to addictive habits will also be taken. The examination will be done according to the proforma. All patients are subjected to complete physical examination along with obstetric examination. All the routine investigations included the complete blood counts, blood picture, RBS, RFT, LFT, OGTT, CUE and ultrasonography with doppler. Histopathological examination of placenta after delivery

Data analysis. The collected data were collected, coded, entered into a Microsoft Excel worksheet and exported to SPSS. Data were analyzed using SPSS version 21. Data are presented as percentages in categories and then presented as tables and diagrams. The Chi-square test and paired t-test were used for the test of significance.

3. Results

The age distribution, among the total 150 patients, 128 of them were between 20–30 years, 12 were >30 years, and 10 were ≤19 years. The overall age of the patients was 25.2±4.19. Parity, among the 75 GDM 26 were primigravida and 49 were multigravida whereas the control group (non-diabetics) 39 were primigravidas and 36 were multigravidas. Of the modes of delivery among the gestational diabetics, 26 were elective, 23 the delivery was spontaneous, 14 had emergency LSCS, 11 the mode was induced, and one patient was vacuum assisted (Table 1).

Table 1

Distribution according to age

Age	GDM	Normal	Total
≤19 years	2	8	10
20–30 years	64	64	128
>30 years	9	3	12
Total	75	75	150
Mean age:	25.8±4.21	24.6±4.11	25.2±4.19
Primi	26	39	65
Multi	49	36	85
Mode of delivery			
Elective	26	15	41
Emergency LSCS	14	10	24
Induced	11	10	21
Spontaneous	23	38	61
Vacuum	1	2	3

Past history among the cases were majority had normal babies, and the remaining macrosomia (17 %), followed by the history of abortion (9.4 %), history of the intra-fetal uterine defect (8 %), previous gestational diabetes pregnancy (5.3 %) and 4 % had an anomalous baby (Fig. 1).

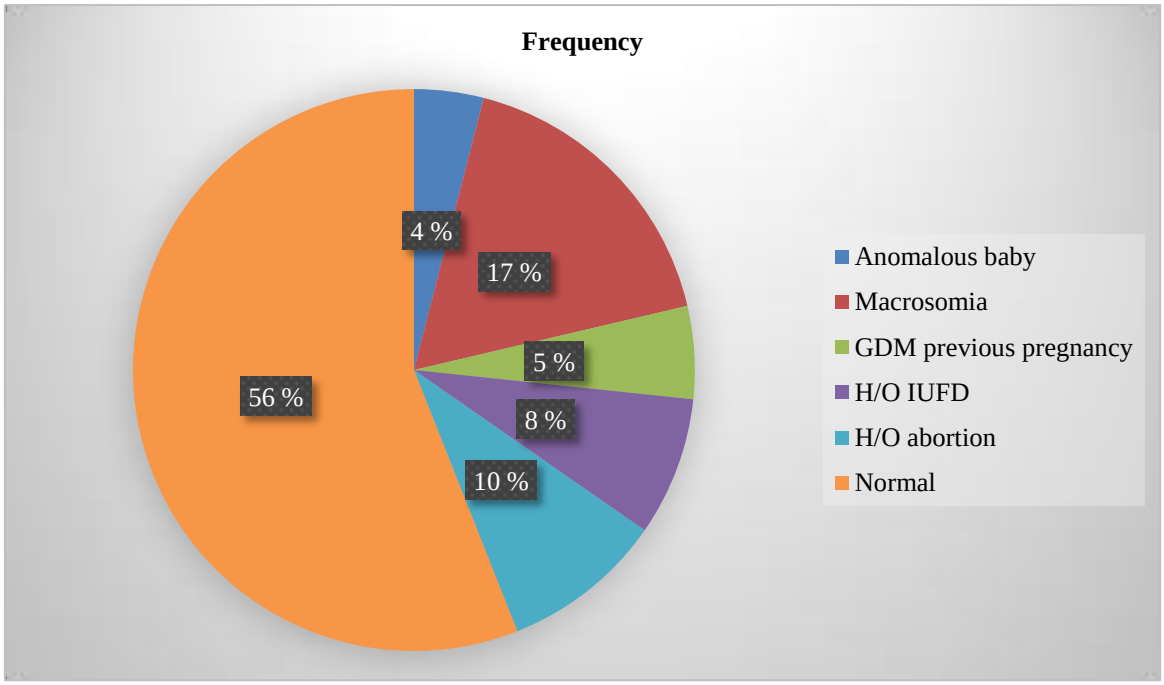

Fig. 1. Pie chart showing the previous history of diabetes

Of the placenta shape among GDM's, 38 the shape was round, 30 the shape was oval, and 7 the shape was irregular. In the normal patients, the shape was round (42) and oval (30), and none were irregular. Shows the site of cord insertion; among GDM 32 the insertion was central, in 20 the insertion was moderately eccentric, and insertion was highly eccentric in 23. In the normal group 51 of them, the insertion was central, in 13 the cord insertion was moderately eccentric; and in 11 the insertion was highly eccentric (Table 2).

The morphological characteristic among the cases were the mean placenta weight was 563.7±96.78 grams, the mean diameter was 18.15±1.59, the mean circumference was 56.98±5.02, area of the placenta was 258.8±44.7, and the central thickness was 2.51±0.37. Among the controls, the mean placenta weight was 496.6±88.39 grams, the mean diameter was 17.62±1.61, the mean circumference was 55.39±5.05, the area of the placenta was 243.4±45.93, and the central thickness was 1.79±0.37 (Table 3).

Table 2

Distribution according to placenta shape

Placenta shape	GDM	Normal	Total
Round	38	45	83
Oval	30	30	60
Irregular	7	0	7
Site			
Central	32	51	53
Moderately eccentric	20	13	33
Highly eccentric	23	11	34
Marginal	-	-	-

The mean weight of the baby was 3.14±0.35 (kilograms) of GDM mothers, with the F:P ratio being 5.68±0.75. In the controls, the mean weight of the baby was 2.82±0.36 (kilograms) with an F:P ratio of 6.10±0.60. It was observed the babies of GDM mothers weighed more than the non-diabetic (Table 4).

Table 3

Gross morphology parameters of placenta

Parameters	GDM	Normal
Mean placenta weight	563.7±96.78 grams	496.6±88.39 grams
Mean diameter (cm)	18.15±1.59	17.62±1.61
Mean circumference (cm)	56.98±5.02	55.39±5.05
Area (Sq.cm)	258.8±44.7	243.4±45.93
Central thickness (cm)	2.51±0.37	1.79±0.37

Table 4

Morphological parameters of baby

Baby parameters	GDM	Normal
Mean baby weight (Kgs)	3.14±0.35	2.82±0.36
F:P ratio	5.68±0.75	6.10±0.60

The weight of the babies born to GDM mothers and normal mothers were compared, and the GDM mothers' babies weighed higher, meaning diabetes has an effect on the baby's weight which was statistically significant ($p<0.001$). The comparison of the placenta was done for cases and controlled. The result suggested all

the morphological parameters, i.e. placenta weight (p<0.001), diameter (p<0.001), area (p<0.002) and thickness (p<0.001), were statistically significant. When the circumferences were compared, it was not significant (p=0.49). All the parameters have increased among the cases (GDM) when compared with controls (Table 5).

Table 5

Comparisons of GDM placenta with normal placenta

Association	Mean	SD	95 % CI		p-value
			Lower	Upper	
GDM baby wt.	3.13	0.12	0.217	0.306	<0.001*
Normal wt.	2.87	0.11			
GDM placenta wt. * Normal placenta wt.	111.25	69.26	95.31	127.18	<0.001*
GDM placenta diameter * Normal placenta diameter	0.60	1.16	0.338	0.876	<0.001*
GDM placenta circumference * Normal placenta circumference	0.533	6.73	1.01	2.08	0.49
GDM placenta area* Normal placenta area	18.35	48.58	7.17	29.53	<0.002*
GDM placenta thickness * Normal placenta thickness	0.73	0.54	0.61	0.86	<0.001*

Neonatal complications were noted for the cases and controls.

Among the cases, 32 had complications, and among the normal group, only 9 had complications. The reported complications were respiratory ones, hypoglycemia, hyperbilirubinemia, meconium staining, polycythemia, sepsis and hypocalcemia.

The complications among the cases and controls were assessed, and a high statistical significance was obtained between the two, meaning babies of gestational diabetic mothers have a high risk of developing neonatal complications than babies of non-diabetic mothers (Table 6).

Shows the pathological changes among the cases and controls. Among the cases, chorangiosis was present in 11; most of the placenta was mild and moderate concerning PAS gravity. Vascular endothelial growth factor (VGEF) trophoblasts were moderate and strong among 53 cases. However, VGEF endothelial cells were negative in 50 cases (Table 7).

Table 6

Distribution of neonatal complications

Neonatal complications	GDM	Normal	Total
Respiratory complications	9	3	12
Hypoglycemia	6	2	8
Hyperbilirubinemia	8	3	11
Meconium staining	3	1	4
Polycythemia	1	–	1
Neonatal sepsis	3	–	3
Hypocalcemia	2	–	2
Total	32/75	9/75	150

Among the cases, 50 had increased villous oedema, 49 had increased villous fibrosis and fibrinoid necrosis, and 62 had increased syncytial knots (Table 8).

The p-value for all 4 parameters, i.e. villous oedema, villous fibrosis, syncytial knots, and fibrinoid necrosis, is seen on histopathological examination was <0.05, and hence there was a significant difference between these findings in both the groups (Table 9).

Table 7

Pathological changes

Pathological changes	Cases	**Controls**	**Total**
Chorangiosis			
Present	11	0	11
Absent	64	75	139
Total	75	75	150
PAS gravity			
Hazy	0	32	32
Trace	11	34	45
Mild	37	9	46
Moderate	22	0	22
Strong	5	0	5
Total	75	75	150
VGEF trophoblasts			
Week	22	6	28
Moderate	28	30	58
Strong	25	39	64
Total	75	75	150
VEGF endothelial cells			
Negative	50	2	52
Week intensity	15	2	17
Moderate intensity	2	30	32
Strong intensity	8	41	49
Total	75	75	150

Table 8

Histopathological parameters among the cases (n=75)

Pathological changes	Frequency	Per cent
Villous oedema		
Increased	50	75 %
Normal	25	25 %
Villous fibrosis		
Increased	49	65.4 %
Normal	26	34.6 %
Syncytial knots		
Increased	62	82.6 %
Normal	13	17.4 %
Fibrinoid necrosis		
Increased	49	65.4 %
Normal	26	34.6 %

Table 9

Comparison of histopathological parameters of diabetic and normal placenta

Parameters	Diabetic mean	Normal mean	p-value
Villous oedema	55.56	25.44	<0.001*
Villous fibrosis	52.43	28.58	<0.001*
Syncytial knots	56.85	24.15	<0.001*
Fibrinoid necrosis	58.48	22.53	<0.001*

4. Discussion

In the present study, 85.3 % were between 20–30 years, 8 % were >30 years, and 6.7 % were ≤19 years. The mean age of all the study patients was 25.2±4.19. The mean age of study subjects in the GDM group was 25.8±4.21 years, and in the control group, it was 24.6±4.11 years.

The present study findings differed from a study by Daskalakis G et al. in which the mean age of cases was 33.18 years, and the mean age of controls was 32.2 years [7]. The present study findings were similar to a study by Ana KMS et al. in which the mean age of study participants was 28.5 ±5.7 years [8]. The present study findings were similar to a study by Kalra P et al. in which the mean age of the patients was 25.33 ±3.17 years [9]. In this study, the parity, among the GDM group, 34.6 % were primigravida, and 65.4 % were multigravida, whereas in the control group (non-diabetics), 52 % were primigravidas and 48 % were multigravidas.

The present findings were similar to a study by Priyanka et al. in which multigravida status was more common among gestational diabetic women [10]. The present study findings were similar to a study by Shinde GR et al in which 64 % of Gestational diabetic women were multigravidas [11]. The present findings were comparable to a study by Mashkaria AM et al. in which 40.8 % were primi gravida, and 59.2 % were multigravida among women with Gestational diabetes [12]. In the present study, the past history revealed that among the cases/GDM group majority (56 %) had normal babies, and 17 % had macrosomia. The other important past history included a history of abortion (9.4 %), a history of the intrafetal uterine defect (8 %), previous gestational diabetes pregnancy (5.3 %) and 4 % had an anomalous baby.

The present findings were similar to a study by Karla P et al in which 15.1 % of GDM mothers had a previous history of fetal or early neonatal deaths, and 6.06 % had macrosomia babies [9].

The present findings were similar to a study by Hoseini S et al in which 12.3 % of GDM women had a history of previous fetal or early neonatal deaths [12].

The present findings were comparable to a study by Shinde GR et al in which 21.2 % had macrosomia. Other significant history included stillbirths, abortions and IUGR babies [11].

The present findings were similar to a study by Priyanka et al. in which macrosomia was found in 3.88 % of babies, 2.77 % had abortions, 7.22 % had intrauterine deaths, and 2.77 % were stillbirths [10].

In this study, the modes of delivery among the gestational diabetics were 34.6 % – elective, 30.6 % – spontaneous, 18.6 % had emergency LSCS, 14.6 % the mode was induced, and in one patient, it was vacuum assisted. The present study findings were similar to a study by Karla P et al in which among gestational diabetics, 79 % had a cesarean delivery, 18 % had a spontaneous vaginal delivery, and 3 % had assisted vaginal delivery [9]. The present study findings were similar to a study by Gajjar et al in which the Cesarean rate of 19.5 % in GDM patients [11]. The present study findings were similar to a study by Priyanka et al. in which 73.33 % had a vaginal delivery, and only 19.4 % had LSCS mode of delivery [13]. The present study findings concurred with a study by Shinde GR et al in which 36 % had elective LSCS, 16 % underwent emergency LSCS, and 30 % had spontaneous vaginal delivery [11].

In the present study, it was found that among the GDM group, 50.6 % had a round-shaped placenta, 40 % had an oval placenta, and 9.4 % had an irregular placenta. In the normal patients, the shape was round (60 %) and oval (40 %); none were irregular. The site of cord insertion, among GDM patients was central (42.6 %), moderately eccentric (26.7 %) and highly eccentric (30.6 %). In the normal group, 68 % had central insertion, 17.3 % had moderately eccentric insertion, and 14.6 % had highly eccentric insertion.

The present study findings were similar to a study by Elshennawy TMA et al in which the placentas of the control group showed normal discoid (45 %) or oval (55 %) shape. Insertion of the cord was eccentric (70 %) or central (30 %). The placentas of the diabetic group showed an oval (60 %) shape. Diabetic placentas had eccentric cord attachment (80 %) [14].

In the present study, the morphological characteristic of the placenta among the cases included the mean weight being 563.7±96.78 grams, the mean diameter being 18.15±1.59 cm, the mean circumference being 56.98±5.02 cm, the area of the placenta was 258.8± ±44.7 cm^2, and the central thickness was 2.51±0.37 cm. Among the controls, the mean placenta weight was 496.6±88.39 grams, the mean diameter was 17.62± ±1.61 cm, the mean circumference was 55.39±5.05 cm, the area of the placenta was 243.4±45.93 cm^2, and the central thickness was 1.79±0.3 cm. The comparison of placental morphological parameters, i.e. placenta weight ($p<0.001$), diameter ($p<0.001$), area ($p<0.002$) and thickness ($p<0.001$), was statistically significant among the GDM group as compared to controls. When the circumferences were compared, it was not significant ($p=0.49$).

The present study findings were similar to a study by Ashfaq et al in which the placenta of women with GDM had a 22 % increase in weight, 33 % increase in diameter, and 85 % increase in central thickness compared to normal placentas [15].

The present study findings were similar to a study by Saha S et al. in which it was observed that the placentae of diabetic mothers were significantly bigger in size, weight, volume, area, thickness, diameter and circumference than those of normal mothers [16]. In this study, the mean weight of the baby was 3.14±0.35 (kilograms) of GDM mothers with the fetal weight: Placental weight ratio being 5.68±0.75. In the controls, the mean weight of the baby was 2.82±0.36 (kilograms) with a fetal weight: Placental weight ratio of 6.10±0.60. It was observed the babies of GDM mothers weighed more than the non-diabetic. The weight of the babies born to GDM mothers and normal mothers were compared and the GDM mother's baby weighed significantly higher. This implies that diabetes affects the baby's weight.

The present study findings were similar to a study by Huynh J et al. in which placental weight was significantly higher among the GDM group as compared to controls. Likewise, the present findings were comparable to a study by Taricco et al in which lower fetus-to-placenta weight ratios were observed in women with GDM.[18] The present study findings were comparable to a study by Daskalakis G et al in which Fetal/placental weight ratios among cases were 4.13 ±0.82 and among controls was 4.52 ±0.76. The mean fetal birth weight (g) among cases was 3,305.1 ±312, and among controls, it was 3,120.2 ±270 [7].

In the present study, neonatal complications were significantly higher among the GDM group as compared to controls. Among the cases, 42.6 % had complications, and among the controls, only 12 % had complications. The complications reported were respiratory complications, hypoglycemia, hyperbilirubinemia, meconium staining, polycythemia, sepsis and hypocalcemia.

The present study findings were similar to a study by Karla P et al. in which the prevalence of stillbirths, macrosomia, and neonatal intensive care unit (NICU) admissions was significantly higher in the GDM group than in the non-GDM group. In addition, the prevalence of hypoglycemia and hyperbilirubinemia was also higher in GDM than in the non-GDM group but not significant [9].

The findings were concurrent with a study by Shinde GR et al. in which macrosomia, congenital anomalies and NICU admissions were needed in 50.54 % of babies born to GDM women [11].

The present study findings were comparable to a study by Mahalakshmi MM et al in which macrosomia was present in 17.9 % of the babies, hypoglycemia in 10.4 %, congenital anomalies in 4.3 %, and the neonatal mortality rate was 1.9 % among babies born to GDM women [17, 18].

In this study, the pathological changes among the cases and controls were compared, and it was observed that among the cases, chorangiosis was present in 14.6 %; the majority of the placentas were mild and moderate with respect to PAS gravity. Vascular endothelial growth factor (VGEF) trophoblasts were moderate and strong among 70.6 % of cases. VGEF endothelial cells were negative in 66.7 % of cases. Among the cases, 66.7 % had increased villous oedema, 65.3 % had increased villous fibrosis and fibrinoid necrosis, and 82.6 % had increased syncytial knots. There was a significant association between cases and the pathological changes of the placenta, i.e. villous oedema, villous fibrosis, syncytial knots and fibrinoid necrosis.

The present study findings were comparable to a systematic review by Huynh J et al in which GDM pregnancies had more immature villi, increased volume of parenchymal tissue, and increased incidence of fibrinoid necrosis and chorangiosis [19].

The present study findings were similar to a study by Daskalakis G et al. in which a significant increase in fibrinoid necrosis, chorangiosis and ischemia was noted among the placenta of GDM patients [7]. The present study findings concurred with a study by Saha S et al. in which among diabetic mothers, there was a significant increase in villous oedema, fibrin deposition, calcification and congestion of blood vessels [16]. The present study findings were similar to a study by Jarmuzek P et al in which the majority of placentas from GDM pregnancies showed histological findings like villous immaturity, villous fibrinoid necrosis, chorangiosis, and increased angiogenesis [20].

Limitation

1. Small sample size
2. There could be an element of information bias in this study.
3. Limited previously conducted studies.

Prospects for further research. The continuous rise in the rate of maternal obesity is followed by increased GDM incidence. A detailed sequence of events that leads from altered glucose metabolism to placental dysfunction and subsequent pregnancy complications may become an important issue for further studies. The concept of the ‘great obstetrical syndromes’ points to the underlying aetiology of adverse interactions between the materno-placental and fetal unit. Ways to modify or even

prevent this sequence of changes remain challenging for future research.

Recommendations

1. To identify and diagnose patients as early as possible

2. Identification of risk factors.

3. Regular, timely follow-up (special testing parameters).

4. Reduce further complications

5. Reduce the incidence

6. Early intervention is required to minimize complications, morbidity and mortality.

5. Conclusion

The mean placenta weight was 563.7± ±96.78 grams, the mean diameter was 18.15±1.59, the mean circumference was 56.98±5.02, the area of the placenta was 258.8±44.7, and the central thickness was 2.51±0.37. The mean weight of the baby was 3.14± ±0.35 (kilograms) of GDM mothers with the F:P ratio being 5.68±0.75. The weight of the babies born to GDM mothers and normal mothers were compared, and the GDM mothers' babies weighed higher, meaning diabetes has an effect on the baby's weight which was statistically significant. A comparison of the placenta was made for cases and controls. The result suggested all the morphological parameters, i.e. placenta weight ($p<0.001$), diameter ($p<0.001$), area ($p<0.002$) and thickness ($p<0.001$), were statistically significant. Babies of gestational diabetic mothers have a higher risk of developing neonatal complications than those of non-diabetic mothers.

Villous oedema, villous fibrosis, syncytial knots, and fibrinoid necrosis is seen on histopathological examination was <0.05, and hence there was a significant difference between these findings in both the groups. GDM is associated with the adverse fetal and neonatal outcome that often presents with respiratory complications, hypoglycemia, hyperbilirubinemia, meconium staining, polycythemia, sepsis and hypocalcemia. Including GDM into 'the great obstetrical syndromes' emphasizes the role of the placenta in interactions between the maternal and fetal unit.

Conflict of interest

The authors declare that they have no conflict of interest in relation to this research, whether financial, personal, authorship or otherwise, that could affect the research and its results presented in this article.

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