SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Dharmalingam *et al.*, 2024
DOI: 10.21276/SSR-IIJLS.2024.10.5.5

Original Article

open access

Placental Abnormalities and Neonatal Outcomes in Gestational Diabetes: A Case-Control Study in Northeast Tamil Nadu

Mathivanan Dharmalingam[1*], Thenmozhi Murugan[2], Preethi Rajamani[3]

[1]Associate Professor, Department of Anatomy, Karpagam Faculty of Medical Sciences and Research, Coimbatore, India
[2]MBBS Graduate, Govt. Chengalpet Medical College, Chennai, Tamilnadu, India
[3]Senior Resident, Department of Pathology, Karpagam Faculty of Medical Sciences and Research, Coimbatore, India

***Address for Correspondence:** Dr. Mathivanan Dharmalingam, Associate Professor, Department of Anatomy, Karpagam Faculty of Medical Sciences and Research, Coimbatore, India
E-mail: maddiballack@gmail.com **& ORCID ID:** https://orcid.org/0009-0005-7386-0209

Received: 02 May 2024/ Revised: 03 Jun 2024/ Accepted: 24 Aug 2024

ABSTRACT

Background: Placenta is the chorio-deciduate organ for exchanging nutrients and gases between mother and foetus. Pathological changes in the placenta have been observed in Gestational Diabetes Mellitus and it also affected neonatal outcomes. Owing to the increase in the incidence of Gestational Diabetes Mellitus, the following study was framed to analyse the morphological, morphometrical and histopathological features of the placenta and the neonatal outcomes among the Gestational Diabetes mellitus-complicated mothers in our population.

Methods: 30 normal placenta (control), 30 Gestational Diabetes Mellitus complicated placenta (cases) and 30 neonates born to normal mothers(control), 30 neonates born to Gestational Diabetes Mellitus complicated mothers (cases) in our obstetric ward were included in the study. Parameters were framed to assess morphology, morphometry, histopathological changes of the placenta, biochemical profiles and neonatal outcomes between controls and cases.

Results: Morphologically, 100% discoid-shaped placenta were observed both in controls and cases. The central type of umbilical cord attachment was seen more in controls, whereas the eccentric type predominated in cases. Statistically, there was a significant ($p<0.5$) difference between controls and cases in placental diameter, total cholesterol, High-Density Lipoprotein, Low-Density Lipoprotein and HbA1C. Histopathological features showed high stromal calcification in controls and basement membrane thickening in some cases. Neonatal Intensive Care Unit admissions predominated as a neonatal outcome in controls and cases.

Conclusion: The results of our study reflected significant changes in several parameters among placenta of Gestational Diabetes Mellitus complicated mothers. The future scope of the survey shall be immunohistochemical and radiological substantiations.

Key-words: Diabetes, Mellitus, Gestational, Neonatal, Placenta, Placental Abnormalities, Neonatal Outcomes

INTRODUCTION

Gestational Diabetes Mellitus (GDM) is defined as "carbohydrate intolerance of variable severity with the onset of the first diagnosis during the present pregnancy" [1]. GDM is a transient insulin resistance state in which hormones in pregnancy, such as human placental lactogen (HPL), cortisol and progesterone, significantly affect the placenta's fetal and neonatal outcomes [2]. GDM complications may affect the placental features like its weight, diameter, thickness [3-9], and vasculature and may exhibit pathological features like syncytial knots, cytotrophoblastic hyperplasia, basement membrane thickening, villous oedema, fibrin deposits, villous fibrosis and chorangiosis [10-21].

Maternal complications due to insulin resistance [22,23] in GDM include perinatal loss, infections, polyhydramnios, preterm labour, preeclampsia, birth injury, metabolic syndrome, chances of recurrences in the next pregnancy (50%), conversion of GDM to type 2 diabetes, dyslipidaemia (elevation in 1% of HbA1c may elevate 18% in lipid profile) and cardiovascular complications [1]. The facilitated diffusion mechanism mainly transposes

How to cite this article
Dharmalingam M, Murugan T, Rajamani P. Placental Abnormalities and Neonatal Outcomes in Gestational Diabetes: A Case-Control Study in Northeast Tamil Nadu. SSR Inst Int J Life Sci., 2024; 10(5): 6145-6154.

Access this article online
https://iijls.com/

Copyright © 2024| SSR-IIJLS by Society for Scientific Research under a CC BY-NC 4.0 International License

DOI: 10.21276/SSR-IIJLS.2024.10.5.5

glucose from the placenta to the fetus, and the feto-maternal glucose concentration gradient determines the diffusion level. Fetal hyperglycaemia results from uncontrolled glycaemic levels in mothers, further elevating fetal insulin secretion and disarrayed growth [2,21-27]. Because of this, resulting neonatal complications in GDM are macrosomia, hypoglycaemia, respiratory distress syndrome, birth injury, shoulder dystocia, hyperbilirubinemia (jaundice), sepsis, Intrauterine growth retardation (IUGR), Neonatal intensive care unit (NICU admissions) and mortality [1,2,10,19,20,21,24-26]. The prevalence of GDM in India was around 0.53% in 2015-16, increased to 0.80% in 2019-21 and further increased to 4.2% in 2023-24.

The ephemeral life of the placenta is a significant period in the evolution of eutherian mammals. Functionally, Placenta is a feto-maternal organ which aids in the exchange of nutrients and gases between the mother and fetus [28,29]. Structurally, the placenta is a chorio-deciduate organ (hemo-chorial), discoid in shape measuring 15 to 25 centimetres (cm) in diameter, weighing around 500-600 grams (g) and with a thickness of around 3 cm at the centre. The placenta comprises maternal and fetal components [29].

Maternal component is derived from serotina or decidua basalis (endometrium). The maternal surface is rough because of cotyledons (15 to 30), each with a stem villus. Basal plate exhibited over the maternal surface constitutes stratum spongiosum of decidua basalis, cytotrophoblastic shell and syncytiotrophoblastic layers. Histological features of the basal plate include connective tissue, decidual plate and placental septa [29].

The fetal component is derived from extraembryonic mesoderm and trophoblast. The placenta's fetal surface is smooth and provides an area for the attachment of the umbilical cord. Based on the attachment of the Umbilical cord to the placenta, the placenta shall be classified into central, eccentric, marginal, velamentous and furcate types [3,24,29]. The chorionic plate covers the fetal surface and consists of syncytiotrophoblast, cytotrophoblast and extraembryonic mesoderm. Histological features of fetal surface include amnion cells (cuboidal epithelium), mesodermal tissue, fetal blood vessels and connective tissue [29].

Embryological development of the placenta includes primary villi constituting syncytiotrophoblast and cytotrophoblast, secondary villi comprising trophoblasts and extraembryonic mesoderm (EEM) and the final tertiary villi comprising trophoblasts, EEM and fetal capillaries. The area eroded by the syncytiotrophoblast is converted into lacunar (intervillous) spaces where maternal blood circulates [29]. Microscopic features of villi are multinucleated cytoplasmic mass (syncytiotrophoblast), syncytial knots (clusters of syncytiotrophoblast nuclei), cytotrophoblasts in early stages, mesodermal connective tissue, hofbauer cells (phagocytic cells), fetal blood vessels and intervillous space constituting maternal blood cells [29].

Based on the clinical complications of GDM and the progressive nature of the prevalence of GDM, despite the effective measures taken in the screening protocols by the national programs, updated documentation of the complications in our population due to GDM proves vital. So, in the present study, we analysed the morphological, morphometrical and histopathological features in the placentas of pregnant mothers diagnosed with GDM and compared the features with the placentas of healthy mothers. Specific biochemical parameters and neonatal outcomes were also determined by quantitative and qualitative analysis.

MATERIALS AND METHODS

Study design- The study was designed as an observational, cross-sectional, case-control type and carried out in a tertiary care hospital (northeastern part of Tamilnadu) for 6 months (2018).

Study sample & Size- Convenience Sampling was done. The study comprised thirty placentas of mothers with normal blood glucose levels (controls), thirty placentas of mothers diagnosed with GDM (cases), thirty Neonates born to the control mothers (controls), thirty Neonates born to the mothers diagnosed with GDM (cases). The sample size was calculated based on the time constraint.

Inclusion criteria- Primigravida, singleton pregnancy, mothers who completed a full term and were between 21 and 35 years old were included in this study.

Exclusion criteria- Placenta of mothers and their neonates who endured complications other than GDM during pregnancy were excluded from the study.

Copyright © 2024| SSR-IIJLS by Society for Scientific Research under a CC BY-NC 4.0 International License

DOI: 10.21276/SSR-IIJLS.2024.10.5.5

Methodology

Diagnosis of GDM- GDM was diagnosed according to the guidelines of "Diabetes in Pregnancy Study of India (DIPSI)" in which 75 g of glucose dose was given to the mother orally. If the blood glucose level was observed to be greater than 140 mg/dl after 2 hours, it was diagnosed as a case of GDM [1].

Placental parameters assessed in controls and cases

Morphology- The shape of the placenta was assessed as discoid or irregular. Type of placenta (based on umbilical cord attachment) was assessed as central (Fig. 1A), marginal (Fig. 1B), and eccentric (Fig. 1C) [3].

Morphometry

- ✓ Placental weight was measured in grams (g), using a digital weighing balance (Fig. 1D) [3].
- ✓ Placental diameter was measured in centimetres (cm), using inch tape and the maximum diameter observed was taken as the diameter of placenta (Fig. 1E) [3].
- ✓ Placental volume was measured in cubic millimetres (mm^3) using Archimede's volume displacement principle (Fig. 1F) [3].
- ✓ The number of placental cotyledons was counted on the maternal side (Fig. 1G) [3].

Fig. 1 (A-G): Morphological and Morphometrical parameters of placenta

Histopathological features- The placental bits were taken from the sites of ischemia in cases and near the umbilical cord attachment in controls. By routine Hematoxylin & Eosin staining the following parameters were observed in both controls and cases of the placenta: Basement membrane thickening, Fibrinoid necrosis, Syncytial knots, Villous fibrosis, Chorangiosis, Villous oedema, Villous Hofbauer cells and Stromal calcification [10-21].

Neonatal outcomes for both controls and case- Newborn Birth weight was measured in Kilograms (kg) by weighing machine [3] and the Feto-placental weight ratio was calculated as the ratio of newborn birth weight to placental weight [3]. Most common presentations in GDM such as Macrosomia, Jaundice, Congenital malformations, Hypoglycaemia, Shoulder dystocia, Sepsis, Respiratory distress syndrome, NICU admission and Mortality were observed for neonatal outcomes [24-26].

Biochemical profiles for both controls and cases- Total cholesterol, High-density lipoprotein (HDL), Low-density lipoprotein (LDL) and HemoglobinA1C (HbA1C) were documented from the records [10].

Statistical analysis- Student's unpaired 't' test was performed to compare the quantifiable means between the controls and the cases.

Ethical approval- Institutional Human Ethical Committee approval (ECR/774/INST/TN/2015) and patient consent were obtained.

Copyright © 2024| SSR-IIJLS by Society for Scientific Research under a CC BY-NC 4.0 International License

DOI: 10.21276/SSR-IIJLS.2024.10.5.5

RESULTS

Placenta (Controls)

Morphological parameters- The placental shape was discoid (100%). The placental type based on umbilical cord attachment was central in 20 (66.67%), eccentric in 7 (23.33%) and marginal in 3 (10%) (Fig. 2).

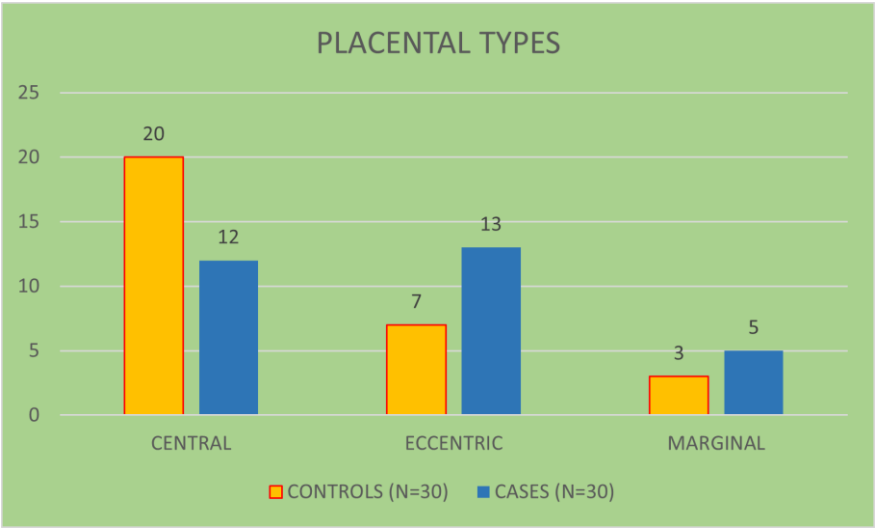

Fig. 2: Placental types based on umbilical cord attachment

Morphometrical parameters- The mean value with SD, documented for the placental weight, placental diameter, placental volume and placental cotyledons were 495.23±72.84 g, 18.37±1.35 cm, 384.97±61.40 mm^3 and 17.30±3.15 respectively (Table 1).

Table 1: Morphometrical parameters of placenta between controls and cases

Parameters	Controls (N=30)		Cases (N=30)	
	Mean	**SD**	**Mean**	**SD**
Placental weight in grams	495.23	72.84	520.67	90.62
Placental diameter in cm	18.37	1.35	19.32	2.18
Placental volume in mm^3	384.97	61.40	403.8	105
Number of placental cotyledons	17.30	3.15	17.37	2.34

Histopathological features- We observed 2(6.67%) specimens with basement membrane thickening, 1(3.33%) specimen with fibrinoid necrosis, 1(3.33%) specimen with syncytial knots, 3(10%) specimens with villous hofbauer cells and 10(33.33%) specimens with stromal calcification (Table 2).

Table 2: Histopathological features of the placenta between controls and cases

Features	Controls (N=30)		Cases (N=30)	
	Report	**Distribution (%)**	**Report**	**Distribution (%)**
Basement membrane thickening	2	6.67	20	66.67
Fibrinoid necrosis	1	3.33	22	73.33
Syncytial knots	1	3.33	19	63.33
Villous fibrosis	0	0	17	56.67
Chorangiosis	0	0	6	20

Copyright © 2024| SSR-IIJLS by Society for Scientific Research under a CC BY-NC 4.0 International License

DOI: 10.21276/SSR-IIJLS.2024.10.5.5

Villous oedema	0	0	11	36.67
Villous hofbauer cells	3	10	16	53.33
Stromal calcification	10	33.33	13	43.33

Neonatal outcomes- The mean with SD for newborn birth weight and feto-placental weight ratio were 2.87±0.34 kg and 5.88±0.87, respectively. We observed, 1(3.33%) macrosomia, 2(6.67%) jaundice,1(3.33%) sepsis and 4(13.33%) NICU admissions as neonatal outcomes (Table 3).

Table 3: Neonatal outcomes between controls and cases

Outcomes	Controls (N=30)		Cases (N=30)	
	Report	Distribution (%)	Report	Distribution (%)
Macrosomia	1	3.33	3	10
Jaundice	2	6.67	9	30
Congenital malformations	0	0	0	0
Hypoglycemia	0	0	1	3.33
Sepsis	1	3.33	1	3.33
Respiratory distress syndrome	0	0	2	6.67
NICU admission	4	13.33	11	36.67
Mortality	0	0	0	0

Biochemical parameters- The mean value for total cholesterol, HDL, LDL and HBA1C were 163.05±14.62,44.53±7.39, 96.15±10.27 and 4.90±0.43, respectively (Table 4).

Table 4: Biochemical parameters between controls and cases

Parameters	Controls (n=30)		Cases (n=30)	
	Mean	SD	Mean	SD
Total cholesterol (mg/dl)	163.05	14.62	200.52	41.85
HDL (mg/dl)	44.53	7.39	40.42	5.67
LDL (mg/dl)	96.15	10.27	112.48	15.91
HbA1C (%)	4.90	0.43	5.91	0.79

Placenta (Cases)

Morphological parameters- The placental shape was discoid (100%). The placental type based on umbilical cord attachment was central in 12(40%), eccentric in 13(43.33%) and marginal in 5(16.67%) (Fig. 2).

Morphometrical parameters- The mean value with SD documented for the placental weight, placental diameter, placental volume and placental cotyledons were 520.67±90.62 g, 19.32±2.18 cm,403.8±105 mm^3 and 17.37±2.34, respectively (Table 1).

Histopathological features- We observed 20(66.67%) specimens with basement membrane thickening, 22(73.33%) specimens with fibrinoid necrosis, 19(63.33%) specimens with syncytial knots, 17(56.67%) specimens with villous fibrosis, 6(20%) specimens with chorangiosis, 11(36.67%) specimens with villous edema, 16(53.33%) specimens with villous hofbauer cells and 13 (43.33%) specimens with stromal calcification (figure 8). In addition, intraplacental arteriopathy, congestion, haemorrhages, and infarct were observed in a case (Table 2, Fig. 3).

Copyright © 2024| SSR-IIJLS by Society for Scientific Research under a CC BY-NC 4.0 International License

DOI: 10.21276/SSR-IIJLS.2024.10.5.5

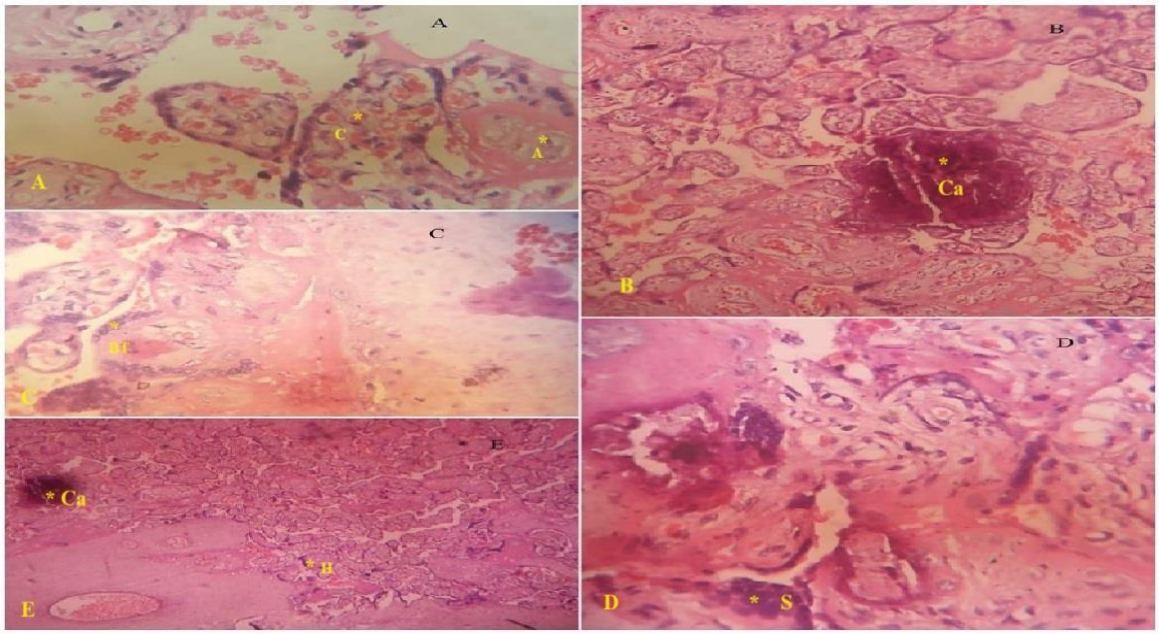

IMAGE 3

A) Placenta with arteriopathy (A) and focal chorangiosis (C)
B) Placenta with calcification (Ca)
C) Placenta with basement membrane thickening (BT)
D) Placenta with syncytial knots(S)
E) Placenta with calcification, hofbauer cells (H)

Fig. 3: Histopathological features of placenta in GDM cases

Neonatal outcomes- The neonatal birth weight was observed as 3.03±0.43 kg and the fetoplacental weight ratio was observed with a mean of 6.13±1.66. We reported 3(10%) macrosomia, 9(30%) jaundice, 1 (3.33%), hypoglycemia, 1(3.33%), sepsis 2 (6.67%), respiratory distress syndrome and 11 (36.67%) NICU admissions as neonatal outcomes (Table 3).

Biochemical analysis- The mean values for Total cholesterol, HDL, LDL and HBA1C were 200.52±41.85,40.42±5.67,112.48±15.91and 5.91±0.79 respectively (Table 4).

Statistical analysis between controls and cases- Student's unpaired 't' test was performed to compare the quantifiable means between the controls and the cases. The inference is that there was a significant ($p<0.05$) difference between controls and cases in placental diameter, total cholesterol, HDL, LDL and HbA1C (Table 5).

Table 5: Statistical analysis between controls and cases

Placenta	p-value
Placental weight in g	0.24
Placental diameter in cm	0.04*
Placental volume in mm^3	0.40
Number of placental cotyledons	0.91
Neonatal Outcomes	
Newborn Birth weight in kg	0.11
Feto-placental weight ratio	0.40
Biochemical Parameters	
Total cholesterol (mg/dl)	0.0001*
HDL (mg/dl)	0.02*
LDL (mg/dl)	0.0001*
HbA1C (%)	0.0001*

** Statistically significant p-values (p<0.05)*

DISCUSSION

The fetoplacental environment highly influences the normal shape of the placenta (discoid) and its morphometry. The variable placental arborization and the vascular fractal underlying the tissues where the umbilical cord attaches play a significant role in the morphological and morphometrical architecture of the placenta.

Kleiber's law of allometric scaling states that "the Basic Metabolic Rate (BMR) in tubes (vessels) is directly proportional to the three-fourths of the mass in the structure [28]." By the abovementioned law, we hypothesise that "Centrally attached umbilical cord with its well distributed vascular fractal shall determine regular shaped placenta and its morphometrical

parameters like diameter, weight, volume and distribution of cotyledons."

In our study, about morphology, we observed 100% of discoid shaped placenta in both controls (n=30) and cases (n=30). Central type of placenta based on umbilical cord attachment was observed in around 66.67% of controls (n=20) and forty percentage (40%) of GDM cases (n=12). The above observation of a complete discoid type without any irregularities in shape, even though the eccentric and marginal types co-existed with the central type, infers that there was a deviation from Kleiber's law based on applying physics over the external morphology. But we postulate in biological terms that around 33.33% of contributions from eccentric and marginal types in controls and 60% of contributions from eccentric and marginal types in cases, which attributed significantly to the microstructural irregularities (increased syncytial knots formation, increased cytotrophoblasts proliferation and increased fibrin deposition) which enhanced the mass of the placenta and thereby maintaining the shape of the placenta which resulted to be discoid rather an irregular type in our study population.

Tandon *et al.* [5], in their study of 50 placentae (25 controls and 25 cases) among the Lucknow population documented 11 discoid & 14 irregularly shaped placenta in controls and 12 discoid & 13 irregularly shaped placentae in cases. In contrast, we observed all the cases (N=30) and controls (N=30) with the discoid-shaped placenta in our study. The differences could be due to implications of Kleiber's law, genetic differences, or variations in ethnicity.

In our study, about the morphometry, the average placental diameter in GDM cases (n=30) was around 520 g and in controls (n=30), it was around 495 g, which reflected a significant difference statistically ($p<0.05$). Other parameters like weight, volume and cotyledons were also greater in GDM cases than in controls, but statistical significance was not evidenced. The increase in morphometrical parameters of our study could again be attributed to Kleiber's law, indicating that hyperglycaemic status in GDM mothers leads to elevated BMR in the placental tissues, promoting the proliferation of tissues and thereby increasing the diameter, weight, volume and cotyledons.

While comparing with the study done by Bhanu *et al.* [3] in 96 placenta (48 controls and 48 cases) among the Nellore population, in both of our studies, we could observe that there was an increase in measurements in cases than in controls of all the morphometry considered. But we had statistical significance while considering placental diameter, whereas they had significance for all the parameters considered (diameter, weight, volume, cotyledons). Tandon *et al.* [5] reported that in 50 placentae of the Lucknow population, the placental weight decreased in cases, and the placental diameter increased in cases compared to the controls. In our study, we differed in our report about the placental weight as we had an increase in placental weight in cases compared to controls. This scenario may be attributed to the variation in insulin resistance among individuals and genetic and ethnic differences.

Normal insulin resistance occurring during pregnancy in controls and increased Insulin resistance in GDM cases caused hyperglycemic environment in developing villi led to histopathological abnormalities like Basement membrane thickening [66.67% (N=20) in cases, 6.67% (N=2) in controls], Fibrinoid necrosis [73.33% (N=22) in cases, 3.33% (N=1) in controls], Syncytial knots [63.33% (N=19) in cases, 3.33% (N=1) in controls], Villous fibrosis [56.67% (N=17) in cases, 0% (N=0) in controls], cholangitis [20% (N=6) in cases,0% (N=0) in controls], Villous oedema [36.67% (N=11) in cases, 0% (N=0) in controls], Villous Hofbauer cells [53.33% (N=16) in cases, 10% (N=3) in controls] and Stromal calcification [43.33% (N=13) cases, 33.33% (N=10) controls] in our study [28].

All the above findings were distinctly observed in GDM cases. The proposed theories for each feature shall be: Basement membrane thickening and excessive syncytial knots formations can be attributed to Kleiber's law, and chorangiosis is due to hyperglycemia, which resulted in tissue proliferation. The overexpression of water channel aquaporin 9 shall be responsible for the accumulation of tissue fluid which resulted in villous edema. Edema resulted in inflammation, which contracted Hofbauer cells, resulting in villous fibrosis, stromal calcification and fibrinoid necrosis due to ischemia, which could be evidenced on the surface. The absence of villous edema, villous fibrosis and cholangitis in controls indicated their significance in GDM-complicated pregnancies.

Mishra *et al.* [10] in the Jabalpur population reported chorangiosis, syncytial knots, villous fibrosis and edema and basement membrane thickening in GDM cases. We also reported the above features along with villous

fibrosis, stromal calcification and Hofbauer cells in GDM cases. The differences between our study and the above study could be due to the variation in the genetic expression of aquaporin channels, resulting in villous edema and further inflammatory complications.

In our study, abundant Syncytial knots formation, excessive proliferation of cytotrophoblasts, basement membrane thickening, villous edema, villous fibrosis in villi and excessive fibrinoid necrosis in intervillous spaces in most of the GDM cases than in controls had led to to insufficient or insignificant surface area for the exchange of nutrients between the mother and foetus [19,21]. Maternal hyperglycaemia results in fetal hyperglycaemia, leading to fetal hyperinsulinemia followed by an increase in lipids and glucose [25].

Above models propose to neonatal complications like Increased newborn birth weight, increased feto-placenta weight ratio, Macrosomia [10% (N=3) in cases,3.33% (N1) in controls], Jaundice [30% (N=9) in cases,6.67% (N=2) in controls], NICU admission [36.67% (N=11) in cases,13.33% (N=4) in controls], Respiratory distress syndrome [6.67% (N=2) in cases,0% (N=0) in controls], Hypoglycaemia [3.33% (N=1) in cases,0% (N=0) in controls] and Sepsis [3.33% (N=1) in cases,3.33% (N=1) in controls] more in GDM cases than controls.

Though the placental features were proliferative, they were pathological and hindered the feto-maternal exchange. Increased newborn birth weight, increased feto-placenta weight ratio and macrosomia shall be hinged to the hyperglycaemic environment created in a fetus due to GDM.

While comparing with the study done by Bhanu *et al.* [3] in 96 neonates (48 controls and 48 cases) among the Nellore population, we observed an increase in birth weight and fetoplacental ratio in cases than in controls. However, we had no statistical significance, but they had significance for both parameters. While comparing with Mishra *et al.* [10] study in the Jabalpur population, they had macrosomia as the most common presentation while we had NICU admission as the most common and in both our studies mortality was the least common complication.

The natural phenomenon of developing insulin resistance by mothers as the fetus grows creates a hyperglycemic environment (lipolysis due to insulin resistance), which is overcome by the adequate insulin produced by the pancreas in normal mothers. This phenomenon of glucose transfer to the fetus enhanced by lipolysis is facilitated anabolism which favors fetal growth. In some mother's, insufficient insulin production from the pancreas to balance insulin resistance results in GDM. Further, this leads to altered lipolysis and dyslipidemia which may result in cardiovascular disorders complicated pregnancy or pre-term delivery [30]. In our study we observed elevated HbA1C [5.91% in cases, 4.9%0 in controls], Total cholesterol [200.52 mg/dl in cases,163.05 mg/dl in controls], LDL [112.48 mg/dl in cases, 96.15 mg/dl in controls] and reduced HDL [40.71 mg/dl in cases, 44.53 in controls]. The above observation statistically ($p<0.05$) reflected a significant difference between cases and controls, enhancing the chances for the above-said complications in cases. While comparing the study by Mishra *et al.* [10] in the Jabalpur population, both our studies showed significant dyslipidaemia changes associated with GDM.

CONCLUSIONS

This study offers significant insights into the parameters and features of the placenta and neonates associated with GDM in our population. Clinical monitoring and management to decrease the prevalence of GDM have been emphasised, with a significant increase in placental diameter, increased HbA1C, and dyslipidaemia in cases compared to controls. Increased incidence of neonatal complications like macrosomia, jaundice and NICU admissions had also been documented in cases than in controls.

However, due to time constraints, this study was done with a limited sample size. In the future, the study will be associated with immunohistochemical and radiological parameters with appropriate sample sizes to enhance the health care of both mothers and foetuses.

ACKNOWLEDGMENTS

The authors thank the donors of the placenta and parents of the newborn for consenting to the study.

CONTRIBUTION OF AUTHORS

Research concept- Mathivanan Dharmalingam
Research design- Thenmozhi Murugan, Preethi Rajamani
Supervision- Mathivanan Dharmalingam
Materials- Thenmozhi Murugan, Preethi Rajamani
Data collection- Thenmozhi Murugan, Preethi Rajamani
Data analysis and Interpretation- Mathivanan Dharmalingam

Copyright © 2024| SSR-IIJLS by Society for Scientific Research under a CC BY-NC 4.0 International License

DOI: 10.21276/SSR-IIJLS.2024.10.5.5

Literature search- Thenmozhi Murugan, Preethi Rajamani
Writing article- Thenmozhi Murugan, Preethi Rajamani
Critical review- Mathivanan Dharmalingam
Article editing- Thenmozhi Murugan, Preethi Rajamani
Final approval- Mathivanan Dharmalingam

REFERENCES

[1] Sarala G, Vanita J. Medical and Surgical illness complicating pregnancy. In: Mudaliar AL, Menon MKK's, Sarala G, Vanita J. Mudaliar and Menon's: Clinical obstetrics. 12th ed., Chennai; Orient Longman, 2015; pp. 326-33.

[2] Lai YM, Tan GC, Shah SA, Rahman RA, Saleh MFM, et al. non-hypertensive gestational diabetes mellitus: Placental histomorphology and its association with perinatal outcomes. Placenta, 2024; 147: 21-27.

[3] Bhanu SP, Devishankar K, Sujatha K, Velichety SD. Gross morphological study of gestational diabetes mellitus placenta from south Indian mothers compared with control placenta. IJAR, 2017; 5: 3521-26.

[4] Sawy NAE, Iqbal MS, Alkushi AG. Histomorphological study of placenta in gestational diabetes mellitus. Int J Morphol., 2018; 36: 687-92.

[5] Tandon A, Singh D, Mishra PP Mishra A. A Morphological and Histological study of placenta in normal and diabetic pregnancies. Int J Res Med Sci., 2018; 6: 1778-81.

[6] Ahmed TME, Halima AA. Effect of Gestational Diabetes on gross morphology, histology and histochemistry of human placenta. Endocrinol Metab Syndr., 2016; 5: 227.

[7] Saha S, Biswas S, Mitra D, Adhikari A, Saha C. Histologic and morphometric study of human placenta in gestational diabetes mellitus. IJAE, 2013; 119: 1-9.

[8] Saini P, Pankaj JP, Jain A, et al. Effect of gestational diabetes mellitus on gross morphology of placenta: a comparative study. Int J Anat Res., 2015; 3: 889–94.

[9] Memon S, Goswami P, Lata H, Agarwal GC. Gross and histological alteration in the placenta of mothers suffering from gestational diabetes. J. Liaquat Univ Med Heal Sci., 2015; 14: 16-20.

[10] Mishra P, Chakrabarti PR. Can diabetes with controlled glycemic status cause placental changes and affect foetal outcome? A histomorphology study from a tertiary care centre from Eastern India. IJMRR, 2017; 5(3): 273-78.

[11] Abdelhalim NY, Shehata MH, Gadallah HN, Syed WM, Othman AA. Morphological and ultrastructural changes in the placenta of the diabetic pregnant Egyptian women. Acta Histochem., 2018; 120: 490-503.

[12] Edu A, Teodorescu C, Dobjanschi CG, Socol ZZ, Teodorescu V, et al. Placenta changes in pregnancy with Gestational Diabetes. RJME, 2016; 57: 507-12.

[13] Liang X, Zhang J, Wang Y, Wu Y, Liu H, et al. Comparative study of microvascular structural changes in the gestational diabetic placenta. Diab Vasc Dis Res., 2023; 20(3): 1479.

[14] Carrasco-Wong I, Moller A, Giachini FR, Lima VV, Toledo F, et al. Placental structure in gestational diabetes mellitus. Biochimica et Biophysica Acta (BBA)-Mol Basis Dis., 2020; 2: 1866.

[15] Makhseed M, Musini VM, Ahmed MA, Al-Harmi J. Placental pathology in relation to the White's classification of diabetes mellitus. Arch Gynecol Obstet., 2002; 266: 136-40.

[16] Abdelhalim NY, Shehata MH, Gadallah HN, Syed WM, Othman AA. Morphological and ultrastructural changes in the placenta of the diabetic pregnant Egyptian women. Acta Histochem., 2018; 120: 490-503.

[17] Jauniaux E, Burton GJ. Villous histomorphometry and placental bed biopsy investigation in type I diabetic pregnancies. Placenta, 2006; 27: 468-74.

[18] Oglak SC, Obut M, Aşır F, Yılmaz EZ, Bolluk G, et al. Histopathological changes in the placentas of pregnant women with Gestational Diabetes Mellitus. JDDT, 2024; 14(4): 14-18.

[19] Rajkumar KK, Augustine G, Jithesh TK. Histological alterations of the placenta in gestational diabetes mellitus: Implications for fetoplacental transport and fetal outcome. Nat J Physiol Pharm Pharmacol., 2024; 14: 6.

[20] Augustine G, Pulikkathodi M, Jithesh TK RSJ. A study of placental histological changes in gestational diabetes mellitus on account of fetal hypoxia. Int J Med Sci Public Health, 2016; 5: 2457.

[21] Keche HA, Kazi S. Correlation of histology of placenta with fetal outcome in pregnancy induced hypertension. Nat J Clin Anatomy, 2018; 7: 24-29.

Copyright © 2024| SSR-IIJLS by Society for Scientific Research under a CC BY-NC 4.0 International License

DOI: 10.21276/SSR-IIJLS.2024.10.5.5

[22]Alejandro EU, Mamerto TP, Chung G, Villavieja A, Gaus NL, et al. Gestational Diabetes Mellitus: A Harbinger of the Vicious Cycle of Diabetes. Int J Mol Sci., 2020; 21(14): 5003.

[23]Calvo MJ, Parra H, Santeliz R, Bautista J, Luzardo E, et al. The Placental Role in Gestational Diabetes Mellitus: A Molecular Perspective touch. REV Endocrinol., 2024; 20(1): 10-18.

[24]Madhuri K, Jyothi I. A study on placental morphology in gestational diabetes. J. Evid. Based Med. Healthc., 2017; 4: 71–75.

[25]Kamana KC, Shakya S, Zhang H. Gestational diabetes mellitus and macrosomia: a literature review. Ann Nutr Metab., 2015; 66(2): 14-20.

[26]Persson B, Hanson U. Neonatal morbidities in Gestational Diabetes Mellitus. Diabetes Care, 1998; 2: 79-84.

[27]Taricco E, Radaelli T, Rossi G. Effects of gestational diabetes on fetal oxygen and glucose levels in vivo. BJOG An Int J Obstet Gynaecol., 2009; 116: 1729-35. doi: 10.1111/j.1471-0528.2009.02341.x.

[28]Salafiaa CM, Yampolsky M. Metabolic Scaling Law for Fetus and Placenta. Placenta, 2009; 30: 468-71.

[29]Standring S. Implantation and placentation. In: Gray H, Standring S, Ellis H, Berkovitz BKB, editors. Gray's anatomy: the anatomical basis of clinical practice. 42nd, Edinburgh; Elsevier Churchill Livingstone, 2021; pp. 178-86.

[30]Scholtens DM, Bain JR, Reisetter AC, Muehlbauer MJ , Nodzenski M, et al. Metabolic Networks and Metabolites Underlie Associations Between Maternal Glucose During Pregnancy and Newborn Size at Birth. Diabetes, 2016; 65(7): 2039–50.

Open Access Policy:
Authors/Contributors are responsible for originality, contents, correct references, and ethical issues. SSR-IIJLS publishes all articles under Creative Commons Attribution- Non-Commercial 4.0 International License (CC BY-NC). https://creativecommons.org/licenses/by-nc/4.0/legalcode