Section: Miscellaneous

Original Research Article

DIVERGENT EFFECTS OF MATERNAL DIABETES AND MALNUTRITION ON PLACENTAL MORPHOLOGY AND FETAL GROWTH: A PROSPECTIVE COHORT STUDY

Sushil Kumar Singh[1], Ranjeet Singh Arya[2], Ritu Bala Soni[3]

[1]*Assistant Professor, Department of Obstetrics and Gynaecology, Rani Durgawati Medical College, Banda, Uttar Pradesh, India*
[2]*Professor and HOD, Department of Anatomy, School of Medical Sciences, Sri Satya Sai University of Technology and Medical Sciences, Sehore, Madhya Pradesh, India*
[3]*Associate Professor, Department of Anatomy, Gandhi Medical College, Bhopal, Madhya Pradesh, India*

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Corresponding Author:
Dr. Ritu Bala Soni,
Associate Professor, Department of Anatomy, Gandhi Medical College, Bhopal, Madhya Pradesh, India
Email: ritubala602@gmail.com

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ABSTRACT

Background: Maternal diabetes mellitus and malnutrition are prevalent metabolic insults known to adversely affect placental development and fetal outcomes. This study was designed to compare the gross morphological changes in full-term placentas from these two distinct pathological conditions against those from normal pregnancies and correlate them with neonatal birth weight.

Materials and Methods: A prospective, comparative study was conducted on 100 full-term placentas collected post-delivery. A systematic gross examination documented placental weight, diameter, umbilical cord length, and neonatal birth weight.

Results: The results showed that the diabetic group exhibited a significant increase in mean placental weight, diameter, umbilical cord length, and neonatal birth weight when compared to the control group. Conversely, the malnourished group demonstrated a significant reduction in all these parameters. No significant differences were observed in the umbilical cord insertion site or the number of vessels across the groups.

Conclusion: These findings demonstrate that maternal diabetes and malnutrition induce divergent morphological changes in the placenta, leading to fetal overgrowth (macrosomia) and growth restriction, respectively. This highlights the critical role of the maternal metabolic environment in determining feto-placental health and underscores the importance of targeted nutritional and glycemic management to ensure favorable neonatal outcomes.

Keywords: Placenta, Diabetes, Malnutrition, Pregnancy, Malnourished.

INTRODUCTION

The placenta is a transient yet indispensable organ that functions as the vital maternal-fetal interface throughout gestation. It is a composite structure, originating from both fetal trophoblastic tissue (chorion) and maternal uterine tissue (decidua basalis). Its primary role is to facilitate physiological exchange between the maternal and fetal circulations. Until the fetal organ systems achieve full functionality, the placenta performs a remarkable array of critical tasks. These include respiratory gas exchange (O_2, CO_2), the excretion of fetal metabolic waste products like urea, and crucial endocrine functions, including the synthesis of hormones such as hCG, hPL, progesterone, and estrogens. Furthermore, it establishes an immunological barrier, protecting the fetus from maternal rejection.

Clinically, the human placenta is classified as haemochorial (maternal blood is in direct contact with the fetal chorion), chorioallantoic, and deciduate.

At term, the gross morphology of the placenta is a flattened, discoid organ weighing approximately 470 grams, with a volume of about 500 ml and a diameter of 185 mm. It is thickest at its center, tapering toward the periphery, and presents with two distinct surfaces: fetal and maternal.

Fetal Surface: The fetal surface, covered by the translucent amnion, is characteristically smooth and glistening. This transparency allows for the visualization of the underlying chorionic plate and its

vasculature. The umbilical cord, typically 50 cm in length and 1-2 cm in diameter, has a central or slightly eccentric insertion on this surface. The cord contains two umbilical arteries and one umbilical vein (the right umbilical vein regresses during early development). From the cord insertion point, the major branches of these umbilical vessels radiate across the chorionic plate, with the larger-caliber veins running deeper than the arteries.

Maternal Surface: In stark contrast, the maternal surface is deep red, with a rough, spongy texture. It is demarcated into 15 to 30 convex lobes known as cotyledons by deep grooves, or sulci. These cotyledons are the functional units of the placenta and are formed by underlying placental septa that project from the decidua.

MATERIALS AND METHODS

For this comparative study, a total of 100 full-term placentas were prospectively collected post-delivery. The cohort was stratified into three groups: placentas from normal, uncomplicated pregnancies; those from pregnancies complicated by diabetes mellitus; and those from pregnancies affected by maternal malnutrition. Collection was contingent upon obtaining informed consent, and each specimen was accompanied by a detailed maternal case history and all relevant clinical and laboratory investigations.

Immediately following delivery, each placenta was thoroughly irrigated with normal saline to remove adherent blood clots. A systematic gross morphological examination was then performed, meticulously documenting the following parameters:

Biometrics: The weight (in grams) and the maximum diameter (in centimeters) of the placental disc were recorded.

Umbilical Cord: The cord was assessed for its insertion point, length, diameter, and the presence of any abnormalities.

Fetal Surface Examination: The fetal surface was carefully inspected for its characteristic color and translucency. It was also scrutinized for pathological changes, including the presence of subchorionic fibrin deposition, cysts, and evidence of thrombosis within the fetal surface vessels.

Maternal Surface Examination: The maternal surface was evaluated for its completeness, ensuring no cotyledons were retained. It was also examined for the integrity of the intercotyledonary septa, any lacerations, focal depressions indicative of infarcts, and signs of retroplacental hemorrhage or hematoma.

Inclusion Criteria

Participants were selected for this study based on the following criteria:

Maternal Age: All included cases were of women between 20 and 35 years of age.

Gestational Age: Only term pregnancies, defined as a gestational age of 38 to 42 weeks as confirmed by per-abdominal clinical examination, were included.

Population Homogeneity: The study population was selected from a single geographical region to minimize potential confounding variables related to racial, ethnic, or significant environmental differences.

The present study comprised a total of 100 placentas, which were prospectively categorized into one of three distinct groups based on the maternal clinical and laboratory profile: a control group, a diabetic group, and a malnourished group.

Control Group (Normal): Placentas in this group were sourced from healthy, non-anemic parturients with a hemoglobin (Hb) level of 12 g/dL or higher. These women had no clinical evidence of pallor or edema and had otherwise uncomplicated term pregnancies.

Diabetic Group: This group included placentas from mothers diagnosed with gestational or pre-existing diabetes. The diagnostic criteria for inclusion were a fasting blood glucose (FBG) level between 101 and 150 mg/dL and/or a 2-hour postprandial blood glucose (PPBG) level ranging from 120 to 307 mg/dL.

Malnourished Group: Placentas were assigned to this group if the mother presented with moderate to severe anemia, defined by a hemoglobin (Hb) level between 6 and 11 g/dL. Inclusion in this cohort was further supported by clinical findings of significant pallor and/or pedal edema.

RESULTS

Table 1: Total number of placentas examined

Group	Placentas	Percentage
Normal	30	30%
Malnourished	42	42%
Diabetic	28	28%
Total	100	100%

Total number of placenta examined were 100, out of which 30 were normal, 42 were malnourished and 28 were diabetic placentas.

Table 2: Diameter of placentas in each group

Group	Range (in cm)	Mean
Normal	14-21	17.2
Malnourished	10-19	14.8

Diabetic	15-21	17.6

In diabetic placentas the mean diameter was maximum and in malnourished placentas the mean diameter was least.

Table 3: Weight of placentas in each group

Group	Range (in grams)	Mean
Normal	450-510	485
Malnourished	390-500	445
Diabetic	490-560	525

In diabetic placentas the mean weight was highest and in malnourished placentas the mean diameter was lowest.

Table 4: Examination of umbilical cord and placental surfaces

Group	Umbilical Cord		Surfaces	
	Insertion	Number of vessels	Maternal	Fetal
Normal	Central	3	Normal	Normal
Malnourished	Central	3	Normal	Normal
Diabetic	Central	3	Normal	Normal

Upon gross examination, no significant variations were observed in the umbilical cord insertion site, the number of vessels, or in the macroscopic appearance of the fetal and maternal surfaces of the placentas across the three study groups.

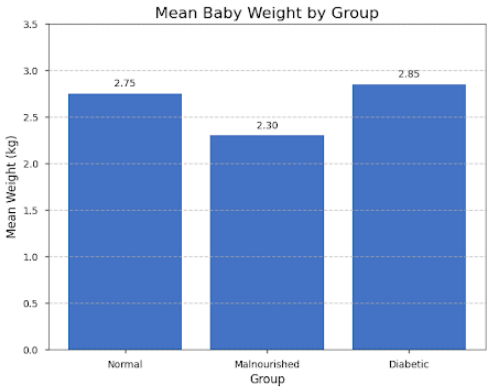
Table 5: Length of umbilical cord in each group

Group	Range (in cm)	Mean
Normal	38-76	57
Malnourished	34-72	53
Diabetic	40-82	61

Mean umbilical cord length was lowest in malnourished placentas and highest in diabetic placentas.

Table 6: Neonatal birth weight in each group

Group	Range (in kg)	Mean
Normal	2.4-3.1	2.75
Malnourished	2.0-2.6	2.3
Diabetic	2.3-3.4	2.85



The highest mean newborn birth weight was recorded in the group of diabetic placentas, while the lowest was observed among the malnourished placentas.

DISCUSSION

The present study was designed to evaluate and compare the morphological changes in placentas from diabetic and malnourished mothers against those from normal, uncomplicated pregnancies. Our findings reveal significant deviations in key placental and neonatal biometrics, which are largely in concordance with the existing medical literature.

Placental Morphometrics

The mean placental diameter in our control group (15-20 cm) was found to be in agreement with the established ranges reported by Patten,[1] (1953) and Brews (1963).[2] Similarly, the average umbilical cord length observed in our study closely approximated the 54 cm average documented by Walkar and Pye (1960).[3]

Placental Weight: In our control group, the mean placental weight at term was determined to be 480 grams. Our analysis demonstrated a significant variation from this baseline in the pathological cohorts: the mean placental weight was highest in the diabetic group and lowest in the malnourished group. This finding of reduced placental weight in malnutrition is consistent with previous research. For instance, Wong,[4] (1966) observed a clear downward trend, reporting a mean placental weight of 339 grams in malnourished cases compared to 463 grams in controls. Further corroborating this, Krishna M, Agarwal KN,[5] (1979) attributed this significant placental weight reduction in malnutrition to a decrease in total cell number, suggesting placental hypoplasia.

Conversely, the increased placental weight observed in our diabetic cohort aligns with the work of Teasdale F (1985).[6] He postulated that placental macrosomia in diabetic pregnancies results from a parallel increase in both parenchymal and non-parenchymal tissues, a mechanism that likely explains our own observations.

Neonatal Birth Weight: The significantly lower mean birth weight observed in our malnourished cohort is a direct clinical manifestation of Intrauterine Growth Restriction, a finding strongly supported by the established literature. As detailed by Abu-Saad and Fraser (2010),[7] the maternal undernutrition seen in our patients likely led to placental insufficiency, thereby limiting the essential supply of nutrients required for adequate fetal growth. The low birth weights we recorded are therefore not an isolated finding but a predictable consequence of the maternal nutritional deficits

A critical finding of this study was the significant variation in mean neonatal birth weight across the different maternal cohorts. The highest mean weight was recorded in infants of diabetic mothers, while the lowest was observed in infants of malnourished mothers. This direct impact of maternal health on neonatal outcomes is well-supported. Auinger W,[8] (1977) also reported diminished newborn weight in cases of maternal malnutrition, concluding that the decrease was primarily dependent on the mother's own compromised nutritional state, a conclusion that our data appears to support.

The findings of our study, which show a significant increase in placental weight in the diabetic cohort, are in strong concordance with the conclusions of the systematic review by Huynh et al (2015).[9] In contrast to these findings, our malnourished cohort showed reduced weight, highlighting the divergent pathological pathways initiated by different maternal metabolic insults.

CONCLUSION

Gestational diabetes and maternal malnutrition are prevalent conditions that profoundly impact placental morphology and, consequently, fetal development. The findings of this study underscore the critical importance of optimizing the nutritional status of women, not only during pregnancy but throughout their reproductive years. The health trajectory of an individual is established in utero, making maternal health a cornerstone of long-term public and national health.

This principle is supported by the work of Chan KK, HoLF, Lao TT (2009),[10] who demonstrated that both maternal nutritional intake and gestational diabetes affect relative placental growth. They concluded that dietary modulation improves pregnancy outcomes in gestational diabetes, not just by enhancing glycemic control, but also by directly influencing placental growth through modifications in protein intake. Therefore, targeted nutritional interventions are a vital strategy for mitigating adverse pregnancy outcomes.

REFERENCES

1. Patten, B. M, Human Embroylogy .Second Ed. (1935) ,pp. 153-162. Marple Press .co. York P.A.
2. Brews,M.(1963) Holland & Brews Manual of Obsterics 12th ed. 1:45 Clay A and bourne A William Heinemann Medical book Ltd ,London .
3. Walkar C.W.&B.G, the lenth of Human umbilical cord .Brit med J,(1960),1:546-548
4. Wong, Latour J.P.A.Ame.Jour. of Obst.&Gynec. Vol. 94 No.7, (1966), pp.942 . April 1,66.
5. Krishna M ,Agarwal K.N. Effect of pregnancy anemia on cellular growth in the human placenta .Acta Paediatr Scand .1979 Nov ;68 (6):899-901 .
6. Teasdale F. Histomorphometery of placenta in class c diabetes mellitus .Placenta(1985) Jan –Feb;6(1) :69-81
7. Abu-Saad, K., & Fraser, D. (2010). Maternal undernutrition and intrauterine growth restriction. Journal of Internal Medicine, 268(1), 5–17.
8. Auniger W et al ,the influence of anemia on weight of child and placenta .Geburtshilie frauenheiked .(1977) Jul ,37 (7) :589-592.
9. Huynh, J., Dawson, D., Roberts, D., & Bentley-Lewis, R. (2015). A systematic review of placental pathology in maternal diabetes mellitus. Placenta, 36(2), 101-114.
10. Chan KK, HoLF, Lao TT (2009) Iron supplement in pregnancy and development of gestational diabetes –a randomised placebo-controlled Trial , BJOG ,Vol.116 ,issue 6 ,pages 789-798