IMPACT OF CONCURRENT DIABETES AND SICKLE CELL ANAEMIA ON FULL-TERM PLACENTA IN CHHATTISGARH REGION

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ABSTRACT

BACKGROUND

The placenta is a fetomaternal organ with important metabolic, endocrine and immunologic functions besides being responsible for nutrition, respiration and excretion for the foetus. It is the most accurate record of infant's prenatal experience. Placental examination reflects prenatal factors and postnatal foetal outcomes.

MATERIALS AND METHODS

In the present study, 150 full-term placentas were studied morphologically in which 50 placentae were collected from normal mothers, 50 were from mothers with diabetes and 50 were from mothers with sickle cell disease. Placentae were examined for shape, weight, volume with surface area, number of cotyledons and attachment of umbilical cord.

RESULTS

Cotyledon count was significantly higher in placentas of diabetic cases. The most common shape of the placenta was discoidal in both normal cases (68%) and in mothers with diabetes (48%). The triangular shape of placenta was significantly higher in cases of diabetes (16%) in comparison to normal placentas (4%). Irregular shape was found in only 6% of normal placenta, while it was 10% in cases of diabetic cases, which was significantly higher. Attachment of umbilical cord was found most commonly as central in normal cases (32%), while as eccentric in diabetic cases (34%). Marginal attachment of umbilical cord was found significantly higher in cases of diabetes (30%) in comparison to normal placenta (16%). The weight of placenta was significantly higher in cases of diabetes (mean 545.64 gm) in comparison to normal subjects (mean 468.88 gm). The mean volume of placenta was also significantly higher in cases of diabetes (mean 700.20 mL) in comparison to normal subjects (mean 576.44 mL). The placental surface area was also affected by diabetes and it was found higher 22 (mean 318.82 cm) than normal subjects (mean 273.12 cm).

CONCLUSION

The present study indicated that diabetes and sickling adversely affects gross placental parameters. This might be the probable reason behind worst foetal outcome in mothers with diabetes and sickling. Placental examination reflects prenatal factors and postnatal foetal outcomes.

KEYWORDS

Placenta, Sickling, Diabetes, Cotyledon, Umbilical Cord.

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BACKGROUND

The placenta is a complex foetal organ that fulfils pleiotropic roles during foetal growth. It is the most accurate record of infant's prenatal experience.[1] Placenta is a fetomaternal organ with important metabolic, endocrine and immunologic functions besides being responsible for nutrition, respiration and excretion for the foetus. As per Crawford et al, the human placenta provides a paradox, since it is one of the most readily available structure for examination and yet it is one of the least known.[2] Acting as a barrier, it has a role in protecting the foetus from noxious agents.

Placenta being a foetal organ shares the same stress and strain to which the foetus is exposed. Thus, any disease process affecting the mother and foetus also has a great impact on placenta. Pregnancy complications like hypertension, sickling or gestational diabetes are reflected in the placenta in a significant way macroscopically. Diabetes

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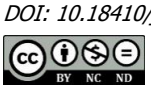


complicates 1-3% of all pregnancies.[3,4] Many numbers of structural and functional changes occurs in placenta with diabetes. The changes depends on glycaemic control quality, treatment modality and time duration of exposure to diabetes. Infants born to diabetic mothers tend to be large for gestational age and have a 2-fold increase in the incidence of being born with a major anomaly.[5] Likewise, neonatal morbidity is increased as a direct result of foetal macrosomia and congenital malformations associated with maternal diabetes.[6,7] Diabetes adversely affects both foetal and placental outcome. The present study comprises of morphometric analysis of normal, sickling and diabetic placenta and their correlation. If this disease is diagnosed at an early stage by blood sugar, urea test and clinical examination, added precaution can be instituted during antenatal period and labour to reduce the further risk to mother and foetus.

MATERIALS AND METHODS

In the present study, 150 full-term placenta cases were collected from patients attending Department of Obstetrics and Gynaecology in tertiary care hospital of medical college, Raipur from Chhattisgarh region. Out of these, 50 were collected from normal mothers (having normal haemoglobin level), 50 were collected from mothers who were diagnosed for sickle cell anaemia and 50 from diabetic mothers. In each case, placenta was washed with normal saline and studied morphologically for weight using weighing machine and diameter using Vernier calliper scale. Volume of placenta was measured by volume displacement method. Shape of the placenta was observed and classified as oval, rounded, discoid or irregular. The attachment of umbilical cord was noted and classified as central, marginal or others. Maternal surface was examined for the number of cotyledons. The morphometric parameters were analysed statistically with Anova test.

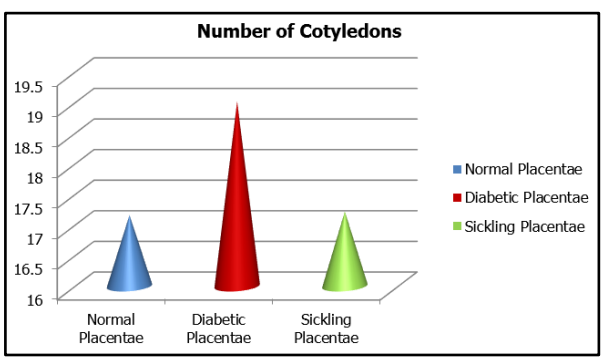
RESULTS

The most common shape of the placenta was discoidal in all normal cases (68%) in mothers with diabetes (48%) and placentae of sickling mothers (50%). The second most common shape of the placenta was oval and it was found significantly higher in mothers with sickle cell disease (32%) than in normal cases (22%) (p value <0.05). The triangular shape of placenta was significantly higher in cases of diabetes (16%) in comparison to normal placentae (4%), whereas it was less in sickled placentae (6%) (p value <0.05). Irregular shape was found in only 6% of normal placenta, while it was 10% in cases of diabetic cases and 16% in sickled placentae, which was significantly higher (p value <0.05).

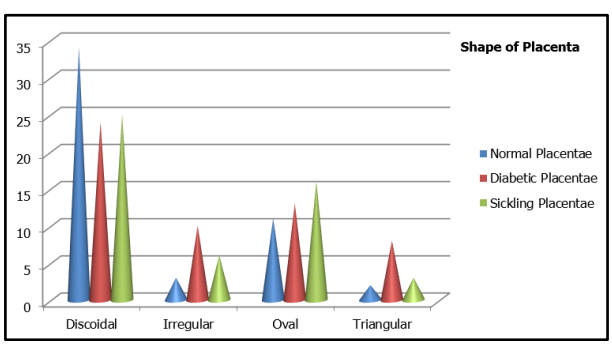
The number of cotyledon was found to be significantly higher (19.16 ± 4.21) in placenta of diabetic cases in comparison to normal placentae (17.13 ± 3.62), whereas no change was observed in cotyledon count in placentas of sickling cases.

Attachment of umbilical cord was found most commonly as central in normal cases (32%), while eccentric in diabetic cases (34%) and near centre in sickling cases (36%). Marginal attachment of umbilical cord was found significantly higher in cases of diabetes (30%) and in sickling (26%) in comparison to normal placenta (16%) (p value <0.05).

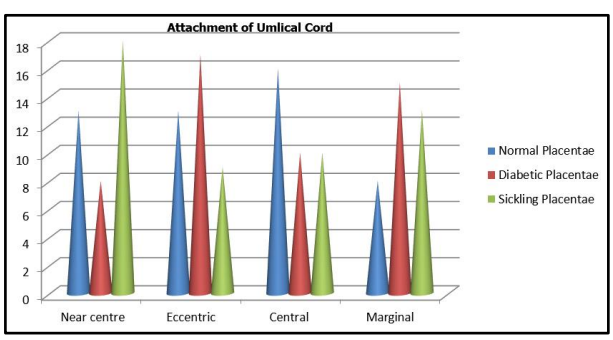
The weight of placenta was significantly higher in cases of diabetes (545.64 ± 88.03 gm) in comparison to normal subjects (468.88 ± 42.49 gm) and was significantly lower in cases of sickling (438.08 ± 45.22 gm) (p value <0.05). The mean volume of placenta was also significantly higher in cases of diabetes (700.20 ± 100.45 mL) in comparison to normal subjects (576.44 ± 119.39 mL) and significantly lower in case of sickling cases (mean 502.06 ± 148.71 mL) (p value <0.05). The placental surface area was also affected by diabetes and it was found higher (318.82 ± 45.19 cm) than normal subjects (273.12 ± 42.53 cm), whereas it was found lower (262.14 ± 52.43 cm^2) in sickling placentae in comparison to normal subjects (273.12 ± 42.53 cm^2) (p value <0.05).



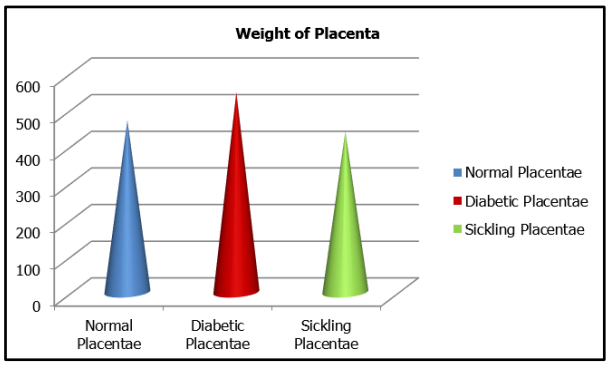
Graph 1. Number of Cotyledons



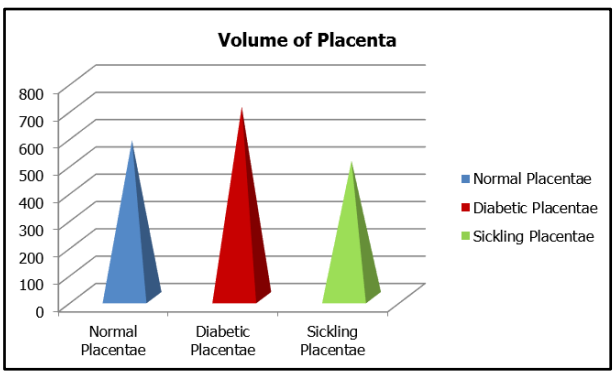
Graph 2. Shape of Placenta



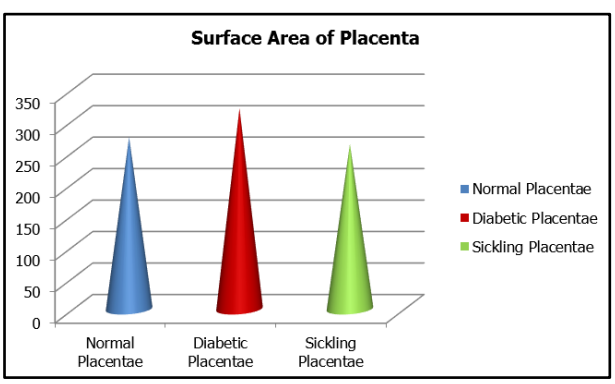
Graph 3. Attachment of Umbilical Cord



Graph 4. Weight of Placenta



Graph 5. Volume of Placenta



Graph 6. Surface Area of Placenta

Figure 1. Discoidal Shape Having Eccentric Attachment of Umbilical Cord in Normal Placenta

Figure 2. Discoidal Placenta having Marginal Attachment of Umbilical Cord in Diabetes Cases

Figure 3. Irregular Placenta with Eccentric Attachment of Umbilical Cord in Sickling Cases

DISCUSSION

The present study showed that the placental outcomes were grossly affected by concurrent diabetes and sickling. The mean weight of placenta was found to be significantly higher in placenta of diabetic cases (545.64 ± 88.03 gm), but it is significantly reduced in placenta of sickling cases (438.08 ± 45.22 gm) (Graph 4). Hosemann et al in his study of normal term pregnancy found the placental weight of 400-1000 grams, whereas Wigglesworth et al found placental weight to be 360-570 grams.[8,9] One of the characteristic features of a placenta in gestational diabetes is its increased weight, which is accompanied by enlarged surface areas of exchange on the maternal (syncytiotrophoblast) and foetal (endothelium) side.[10] In placentae with gestational diabetes, the inflammatory molecules concentration increases due to dysregulation of vascular, metabolic and inflammatory pathways resulting in increase in weight of placenta.[11] Varastehpour et al demonstrated an accumulation of ω-3 fatty acids in placenta of offspring of GDM mothers with increased adiposity at birth.[12]

The volume and surface area were also significantly higher in placenta of diabetic cases (700.20 ± 2100.45 mL volume and 318.82 ± 45.19 cm surface area) in comparison to normal placentas (576.44 ± 119.39 mL volume and 273.12 cm ± 42.53 cm surface area) (Graph 5). These

findings are in concurrence with the study of Desoye G et al and Fox.[13,14] Brinkman et al also elaborated similar results (3). One leading hypothesis is that increased TNFα, leptin and resist in contribute to enhancing insulin resistance in the GDM mother causing rise in placental surface area and volume.[15] The volume and surface area were also significantly lower in placenta of sickling cases (502.06 ± 148.71 mL volume and 262.14 ± 52.43 cm2 surface area) (Graph 6). These findings are in concurrence with the study of Fox and Udainia et al.[16,17]

The cotyledon count was found to be significantly higher in placenta of diabetic cases (19.02 ± 4.21) in comparison to normal subjects (17.16 ± 3.82) (Graph 1). No change was observed in cotyledon count in placentas of sickling cases as compared to normal. Similar findings were also reported by Thomson.[18]

Attachment of umbilical cord was found most commonly as central in normal cases (32%), while as eccentric in diabetic cases (34%). Marginal attachment of umbilical cord was found significantly higher in cases of diabetes (30%) in comparison to normal placenta (16%) (p value <0.05) (Graph 3). 67.1% eccentric insertion, 21.1% central insertion and 10.5% marginal insertion. It was found most commonly near centre in sickling cases (36%).

Pretorius also reported central insertion of umbilical cord in normal placenta (34%).[19] This may be due to blockage of circulatory system by sickle-shaped RBCs resulting in decrease in oxygen availability to tissues beyond the blockage.

The most common shape of the placenta was discoidal in both normal cases (68%) and in mothers with diabetes (48%) (Graph 2). Similar finding is observed by Kalousek et al.[20] The triangular shape of placenta was significantly higher in cases of diabetes (16%) in comparison to normal placentas (4%) (p value <0.05). Irregular shape was found in only 6% of normal placenta, while it was 10% in cases of diabetic cases, which was significantly higher (p value <0.05). The reason behind this finding is that diabetes alters the growth of placentae in beginning of gestation and it has long-term effect as diabetes buffers excess maternal glucose and increases vascular resistance.

The most common shape of the placenta was discoidal in mothers with sickle cell disease (50%) (Graph 2). Pretorius also found discoidal shape as commonest in normal placenta (60%).[19] The irregular and oval-shaped placentae were found more common in sickling cases in comparison to normal mothers placentae. This finding maybe due to disturbance in maternal circulation due to sickle-shaped RBCs.

CONCLUSION

The present study comprises of morphometric analysis of diabetic, sickling and normal placentae and their correlation. Placental coefficient can help to assess the severity of the toxaemia of pregnancy. Diabetes and sickling adversely affects both foetal and placental outcome. If this disease is diagnosed at an early stage by blood sugar, urea test and clinical examination, added precaution can be instituted during antenatal period and labour to reduce the further risk to both mother and foetus.

REFERENCES

[1] Benirschke K. Examination of placenta. Am J Obstet Gynecol 1961;18(3):309-333.

[2] Crawford JM. Vascular anatomy of human placenta. Am J Obstet Gynecol 1962;84:1543-1567.

[3] Brinkman CR. Classification and screening of diabetes mellitus during pregnancy. In: Nowayhid BS, Brinkman CR, Lieb SM, eds. Management of the diabetic pregnancy. New York: Elsevier Science 1987:1-10.

[4] Hollingsworth DR. Types of carbohydrate intolerance in pregnant women. In: Hollingsworth DR, ed. Pregnancy, diabetes and birth: a management guide. Baltimore: Williams & Wilkins 1984:2-16.

[5] Zonana J. Diabetes mellitus: considerations on genetic counselling. In: New MI, Fisher RH, eds. Diabetes and other endocrine disorders during pregnancy and in the newborn. New York: Alan R. Liss 1976:1-12.

[6] Greene MF. Congenital malformations. In: Brown FM, Hare JW, eds. Diabetes complicating pregnancy: the joslin clinic method. New York: Wiley & Sons 1995:181-196.

[7] Kuhl C, Moller-Jensen B. Intensified insulin treatment in diabetic pregnancy. In: Sutherland HW, Stowers JM, Pearson DWM, eds. Carbohydrate metabolism in pregnancy and the newborn IV. Berli: Springer-Verlag 1989:161-172.

[8] Hoseman H. Duration of pregnancy and weight of the placenta. Archives of Gynecology 1946;176:453.

[9] Wigglesworth JS. The gross and microscopic pathology of the prematurely delivered placenta. Journal of Obstetrics and Gynaecology of British Commonwealth 1962;69:934-943.

[10] Desoye G, Shafrir E. The human placenta in diabetic pregnancy. Diabetes Reviews 1996;4:70-89.

[11] Retnakaran R, Hanley AJ, Raif N, et al. C-reactive protein and gestational diabetes: the central role of maternal obesity. J Clin Endocrinol Metab 2003;88(8):3507-3512.

[12] Varastehpour A, Radaelli T, Minium J, et al. Activation of phospholipase A2 is associated with generation of placental lipid signals and foetal obesity. J Clin Endocrinol Metab 2005;91(1):248-255.

[13] Desoye G, Korgun ET, Ghaffari-Tabrizi N, et al. Is foetal macrosomia in adequately controlled diabetic women the result of a placental defect?--a hypothesis. J Matern Foetal Neonatal Med 2002;11(4):258-261.

[14] Fox H. The placenta in intra uterine growth retardation. In: Ward RHT, Smith SK, Donnai D, eds. Early foetal growth and development. London: RCOG Press 1994;223-235.

[15] Kirwan JP, Hauguel-De Mouzon S, Lepercq J, et al. TNF-alpha is a predictor of insulin resistance in human pregnancy. Diabetes 2002;51(7):2207-2213.
[16] Fox H. Pathology of the placenta. 2nd edn. London (UK): WB Saunders 1967:8-23.
[17] Udainia A, Bhagwat SS, Mehta CD. Relation between placental surface area, infarction and foetal distress in pregnancy induced hypertension with its clinical relevance. J Anat Soc Ind 2004;53(1):27-30.
[18] Thomson AM, Billewicz WZ, Hytten FE. The weight of the placenta in relation of birth weight. J Obstet Gynecol Brit Commonwealth 1969;76(10):865-872.
[19] Pretorius DH, Chau C, Poeltler DM, et al. Placental cord insertion, visualisation with prenatal ultrasonography. J Ultrasound Med 1996;15(8):585-593.
[20] Kalousek DK, Langlosis S. The effects of placental and somatic chromosomal mosaicism on foetal growth. In: Ward RHT, Smith SK, Donnai, eds. Early foetal growth and development. RCOG Press 1994:245-256.