Birth weight: Placental weight ratio as an indicator of placental efficiency in pregnancies complicated with gestational diabetes

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Abstract

Background: Placenta translates the maternal environment and the genetic factors that influence the birth weight. The growth of placenta is directly propotional to the nutrient transfer as it is the only foetal source for oxygen and nutrients. The present study was done to find any correlation that exists between the placental measurements and its association with birth weight.

Methods: 80 placentae were studied 40 from diabetic mothers and 40 from normal gestation. The gestational age and fetal weight were taken from the case sheets. Placental measurements were weight, central thickness, shape and placental ratio was calculated. The analysis of association of placental weight and birth weight were done with multiple linear regression.

Results: Our study demonstrates that there is a significant increase in weight and central thickness of placenta. Neonatal weight and placental ratio were also increased; there was no change in shape and site of attachment of umbilical cord in case of diabetic placenta when compared to normal. Birth weight and central thickness correlated with the placental weight ((r=0.733, p <0.0001) and (r=0.836, p <0.0001) for diabetic and normal placenta.

Conclusion: Measurements of placental parameters are reliable in predicting the placental growth and in estimating the foetal birth weight and it will help in understanding of maternal-placental programming of chronic diseases.

Keywords: Diabetic placenta, placental ratio, foetal birth weight

Introduction

Optimal foetal growth is possible with appropriate nutrient provision to the fetus and any kind of placental dysfunction can lead to reduced nutrient supply that can result in fetal growth restriction and foetus fails to achieve genetically predetermined growth [1, 2]. Total surface area of the syncytiotrophoblast that is available for exchange and the

activity of nutrient transporters that determines the ability of the placenta to deliver adequate nutrient supply to the fetus [3]. Previous studies states that the placenta regulates the nutrient transfer by morphological and functional adaptations that result in optimal fetal growth [4]. Placental efficiency is calculated by the ratio of placental weight to the fetal birth weight. It refers to the grams of fetus produced per gram of placenta [5]. Therefore placental efficiency is the near indicator of fetal nutritional requirements [6]. Placental adaptations can occur as a need for maternal and fetal cues that can effect the placental efficiency [6]. Such adaptations can occur for appropriate fetal growth and failure of these adaptations can result in fetus who are small or large for gestational age compared to their genetic potential [7].

Fetal birth weight determines the neonatal growth and development in later age of life and it is the key indicator of neonatal mortality and morbidity [8]. Factors influencing fetal birth weight are maternal nutrition, fetal genetics, sex and the extent of placental development in utero. Variations in fetal birth weight will result in macrosomia or fetal growth restriction which has a great clinical effect [9]. So placental weight and fetal weight is correlated throughout pregnancy which can later vary in later stages of pregnancy [10]. The various factors that influence the placental growth are maternal and fetal circulation, position of insertion of umbilical cord, intrauterine location of placenta and also maternal and foetal responses to inflammation [11, 12].

Placenta is a vital organ that promotes and maintains foetal development that has fetal and maternal surface to serve as a barrier for efficient nutrient transfer [13]. Absolute measure of infant size had a great correlation with the placental weight [14] and a low hematocrit was associated with higher placental weight and lesser fetal weight [15].

Pregnancies with hypertension resulted in higher birth weight placentas and there was no significant difference found in placental weight in the absence or presence of meconium stained amniotic fluid [16, 17]. Gross placental examination can be critical in early neonatal care, reproductive planning and in the risk assessment of neurological outcome of the infant [18]. The present study was done to study the placental efficiency in gestational diabetes in comparison to normal placenta and the findings of this study will have important implications in infant care and decision making in obstetrics.

Methodology

The total number of specimens used in the present study was 80 placentae, 40 from mothers with uncomplicated pregnancies as control group and 40 from mothers with Gestational diabetes were considered as a study group.

Inclusion criteria

Mothers in the age group of 20 to 39 years.

Exclusion criteria

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

The gestational age and fetal weight were taken from the case sheets. The placenta with attached membranes and umbilical cord was collected soon after delivery

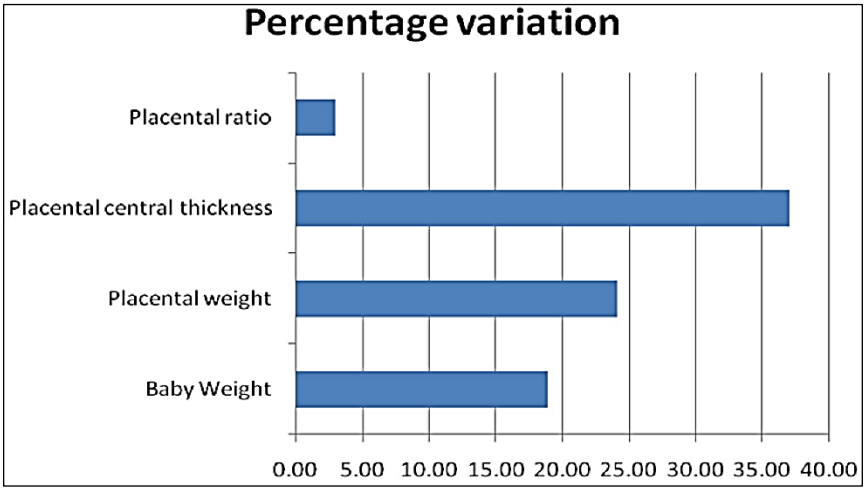
washed in running tap water to clean all blood. Both the surfaces of the placenta was examined after cutting the membranes from the edge of the placenta. Maternal surface of the placenta was inspected for its completeness, calcification and infarction. The placenta was then weighed in weighing machine graduated in grams. Thickness of the placenta is measured by piercing a needle through the center of the placenta

Results

Majority of normal cases were in the age group of 20-24 about 16 cases. About 10 cases were in the age group of 25-30 years of age. 9 cases were in the age group of 30- 35.only 5 cases were above the age of 35. The age range for mothers with gestational diabetes was 25-39 years. The majority of mothers were 25-30 years. 18 cases were in this age group.15 cases were in the age group of 30-35 years. Only 4 cases were in the age group of 20-24 years. In the present study placental weight ranges between 398-890 grams. The mean baby weight in normal pregnancies were 2.48kg whereas in gestational diabetic pregnancies were 3.06 kg (Table 1). The difference between case and control group were statistically significant ($p<0.001$). The mean placental weight in control group were 415.50grams whereas in diabetic placentae, mean placental weight was 547.38 grams. The difference between two groups were stastically significant ($p<0.001$). Mean central thickness of study group when compared with control group were stastically significant ($p<0.001$). The mean placental ratio in control group was 175.03 compared to study group was 180.27.the difference between two values was found to be statistically significant ($p<0.5$).

Table 1

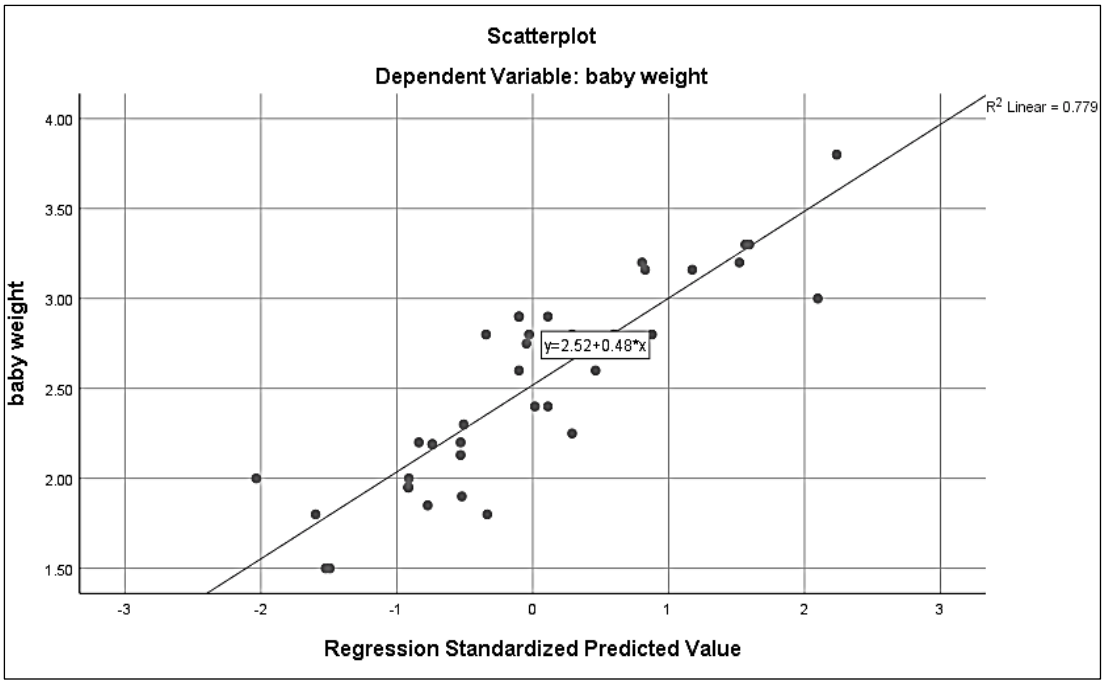
Parameters	Normal Group	Diabetic group	P value
Mean Fetal birth weight	2.5139±0.5428	3.0595±0.46628	P≤0.001
Mean placental weight	414.27±56.531	547.05±134.017	P≤0.001
Mean placental central thickness	1.866±0.4762	3.010± 0.3299	P≤0.001
Mean Age	27.22±4.866	29.31±4.293	P≤0.001
Placental ratio	175.03±40.47	180.27±38.84	P≤0.5



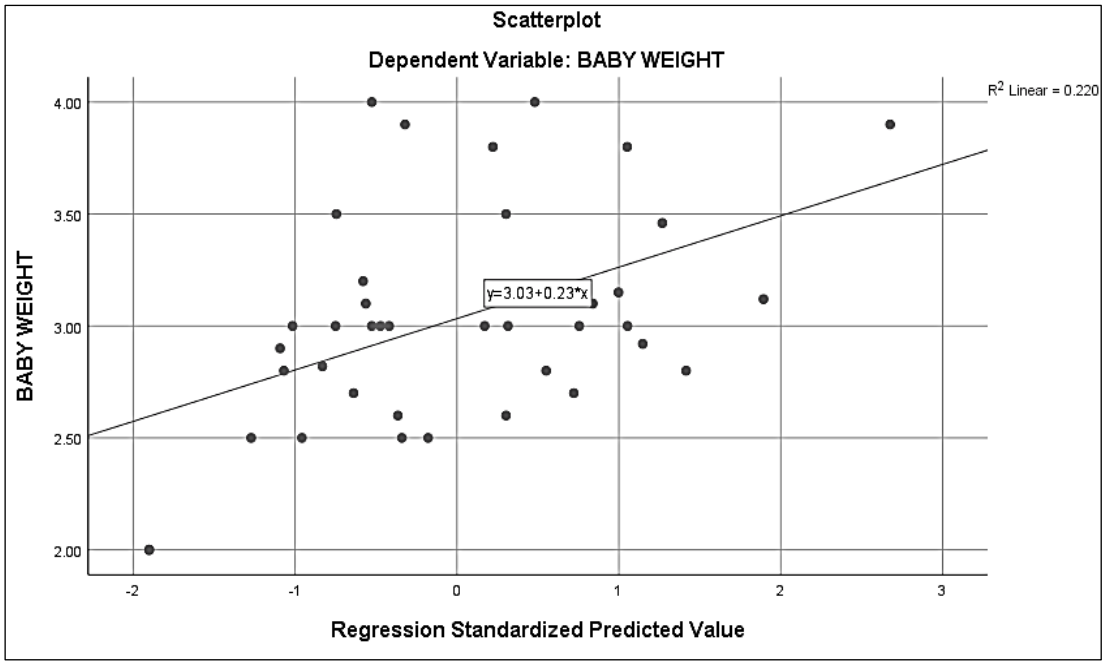
Graph 1

The above graph shows the percentage variation between normal and Gestational Diabetic group. The baby weight shows a percentage variation of 18.86. Placental weight shows a variation of 24.09. Percentage variation between study and control group in case of placental central thickness and placental ratio was 37.05 and 2.90 respectively. In the normal group there was a significant positive correlation between fetal birth weight and placental weight (r=0.733, p <0.0001) and central thickness of placenta (r=0.836, p <0.0001). Table 2 displays the results of regression model of birth weight. Unadjusted model with placental weight, central thickness, placental shape and umbilical cord attachment were 0.001, 0.182, -0.117 and 0.143 respectively in diabetic placenta and 0.003, 0.692, 0.035 and -0.081 respectively in normal placenta.

Graph 2 and 3 shows that the fetal birth weight increases with higher value of placental weight. The linear relationship is expressed as Y=2.52+0.48(X) in case of diabetic placenta and Y=3.03+0.23(X) in normal placenta.



Graph 2: Correlation between fetal and placental weight in diabetic group



Graph 3: Correlation between fetal and placental weight in normal group

In regression analysis the adjusted R square was 0.130 in diabetic placenta compared to 0.754 in normal placenta. This was considered as this parameter was not affected by collinearity.

Even though there was a positive correlation of birth weight and placental weight, graph 3 shows wide variation in placental weight for any given birth weight. This suggests the variation in placental efficiency

Discussion

Comprehensive description of placental measurements and its correlation to fetal birth weight is a very useful guide for any future examination of placental structure. Decrease in fetoplacental ratio leads to small for gestational age infants and act as a indicator of nutrient transport and the uterine environment [19]. Foetal birth weight is influenced by difference in glucose metabolism, level of haemoglobin and macrovascular integrity which can be affected by diabetes mellitus and preeclampsia [20].

In the present study, the mean placental weight in control group was 415.50 grams whereas in Gestational diabetes the mean placental weight was 547.38 grams. The differences between two groups were statistically significant. Placental weight between two groups showed a percentage variation of 24.09. Fetal hyperglycemia may so derange the osmotic environment that injury or cell death results. This process involves endothelial cells of capillaries. The damaged endothelial cells may be replaced without subsequent removal of old basal lamina. New endothelial cells synthesize their own basal lamina leading to excessive thickness of basal lamina of fetal capillaries in the chorionic villi. The basal lamina of chorionic capillaries is the part of placental barrier, so its thickness will increase the whole thickness of placental barrier which may lead to reduced transport of oxygen and other nutrients across the

barrier. In response to this low oxygen transport the terminal villi showed hyperplasia which may be partially responsible for increase in weight of placentae in diabetic group [21]. Teasdale stated that a significant accumulation of non-parenchymal tissue and only a moderate increase in parenchymal tissue may be the cause of heavier placenta in gestational Diabetes [22].

The weight gain in diabetic's placentae may be attributed to macrosomia and compensatory hyperplasia. Macrosomia affects the fetus and fetal part of placenta i.e. chorionic plate and all types of villi. This macrosomia may be attributed to fetal hyperinsulinemia in response to hyperglycemia in fetuses of diabetic mothers [23].

According to Driscoll in 1965, Glucose passes the placenta readily and the fetus responds to hyperglycemia with hyperplasia of islet of Langhans and increased insulin secretion, the primary reason for fetal overweight in maternal diabetes [24].

The present study showed mean placental ratio in control group was 175.03 compared to study group was 180.27.the difference between two values was found to be statistically significant (p<0.5). The study findings are in correlations with the study conducted by Lao. TT in 1997 which showed increased placental ratio in Gestational Diabetic groups compared to control group. He concluded that gestational diabetes mellitus is the production of placental hormones that leads to maternal insulin resistance, and the placental size has been shown to be increased both in gestational and pre-gestational diabetic pregnancies. But an increased placental size could be related to the concomitant increase in fetal size, and it is not clear if a disproportionally bigger placenta is found in gestational diabetes mellitus [25].

An increased placental ratio represents an adaptative process by the feto-placental unit in an unfavorable maternal environment. When there is a limitation imposed on fetal growth velocity due to nutritional deficiencies, the placenta may undergo hypertrophy in an attempt to compensate. An increased placental ratio would be a sign of fetal growth disturbance [25].

Comprehensive knowledge on placental measurement sis very much essential to expand the existing knowledge which can determine the health of the baby in future.

Conclusion: The present study reveals that the incidence of Gestational Diabetes mellitus is more common in the age group of 25 to 30 years. The shape and cord insertion did not show significant variation. The morphometry of placental weight and central thickness was significantly higher in the study group compared to normal control group. The birth weight and placental ratio was also increased in GDM group. It can be concluded that the variability of foetal birth weight may be accounted to the placental measurements.

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