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Effects of Diabetes and Hypertension on Placenta

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Placental insufficiency is the failure of the placenta to supply nutrients to the fetus and remove toxic wastes; it is a common cause of intra uterine growth restriction (IUGR), fetal distress peri and postnatal mortality. Specifically gestational diabetes and hypertension during pregnancy alters placental architecture which contributes to insufficiency of the placenta. Placenta is the most accurate record of infant's prenatal experiences. Clinical condition such as hypertension and diabetes that complicate pregnancy leads to significant structural changes in placenta, these histological changes and morphological alterations in placenta lead to insufficiency, resulting in IUGR and fetal distress. In the present study morphology of placentas collected from diabetic and hypertensive pregnancy will be observed, the histo-pathological findings will be recorded and correlated to birth weight, APGAR scores and congenital anomalies of babies and will be compared to the findings in normal placentas also. This study will be beneficial for obstetrician and pediatrician in gathering knowledge about patho genesis of placental insufficiency; it will add to the knowledge on placental morphology and will high light the abnormalities in placental anatomy.

Keywords: Placental insufficiency; congenital anomalies; toxic wastes; hypertension.

1. INTRODUCTION

Placental insufficiency is the failure of the placenta to supply nutrients to the fetus and remove toxic wastes; it is a complication of pregnancy where placenta which grows during pregnancy to feed the unborn child cannot bring enough nutrients. A normal placenta at term is reddish brown in color, rounded, flattened, and discoid about 450-500 grams in weight 15-20 centimeters in diameter and 2-4 centimeters thick. Umbilical cord can arise almost at any point and fetal membranes at margins. It is derived from fetal and maternal tissue both, the maternal portion is deciduas basalis and fetal portion is called chorion fundosum [1].

2. OBSERVATION

Placental insufficiency is a common cause of intra uterine growth restriction (IUGR), fetal distress peri and postnatal mortality. The reason for this in some cases is unknown but in others it is strongly associated with diabetes, hypertension, viral infections and smoking, or if it is not properly implanted at accurate site. Specifically gestational diabetes and hypertension during pregnancy which alters placental architecture contributes to insufficiency of this structure which brings oxygen and nutrients to fetus and helps remove carbon dioxide and waste [2,3]. This is a common problem encountered by Obstetrician and Pediatrician now a days.

Placenta is the most accurate record of infant's prenatal experiences [4]. Clinical condition such as hypertension and diabetes if complicate pregnancy leads to significant structural changes in placenta [5]. These histological changes and morphological alterations in placenta lead to insufficiency, resulting in IUGR and fetal distress. Normal physiological changes occur in spiral arteries in which endovascular plugs penetrate their lumen and normally disappear at the end of 2nd trimester [6]. In some utero-placental vessel these physiological changes do not take place because of hypertensive complication leading to acute atherosclerosis [7].

More recent studies have shown relation in hypertension and placental lesion and established relation between lesion and clinical severity but still microscopic features are to be reviewed in depth [8,9].

The term placenta was used for the first time in 1559, it is a mirror that reflects the intra uterine status of fetus, fetal distress, intrauterine fetal death and placental abnormalities are common in pregnancy induced hypertension and it is noticed that placental surface area is less in cases related to fetal distress [2]. Physiological changes in spiral arteries occurs in normal pregnancy but are absent in pre ecclemptics. [10]. Placental ischemic alterations are resulting from decrease in maternal blood supply to the placenta associate with hypertensive syndrome of pregnancy [11].

Placenta produce hormones in addition to other function and these have an opposite effect on insulin which causes hyperglycemia in mothers ultimately contribute to fetal growth, this result in gestational diabetes which disappear often after delivery, but if mothers have type I diabetes mellitus chances of congenital birth defect increases [12]. In recent study done in 2008 by Beverley et al. They correlated the placental weight which decreases in response to mutation in glucokinase gene of fetus [13]. Glucose, amino acid and other substrates cross placenta via different mechanisms [14], recently a significant link has been observed between measurement of umbilical cord insulin and placental weight [15]. Placental weight increases in diabetic pregnancies a condition known to be associated with fetal hyper insulinemia although this could be caused by the maternal hyperglycemic environment rather than fetal insulin levels [16].

Scott in 2008 demonstrated that both fetal and placental weight increases in presence of maternal diabetes, their respective weight remains high and fetal weight-to placental weight ratio index (FPI) is not significantly reduced [17,18]. Lower FPI is driven primarily by higher placental weight in both control subjects and off springs of mothers with type I diabetes and is also associated with poorer maternal glycemic control in offspring of mothers with type 1 diabetes [3].

Adiponectin concentration in umbilical cord serum is positively associated to the fetus and placental weight ratio [19]. This association reflects augmentation of insulin sensitivity and consequently increased fetal growth direct effect of fetal adiponectin on placenta are not clear which suggests that other mechanisms are also involved [20,21].

3. CONCLUSION

High blood pressure (hypertension) during pregnancy can prevent the placenta (the food supply for the baby in the womb) from receiving enough blood. The lowered amount of blood to the placenta can lead to a low birth weight. Other complications can occur from high blood pressure during pregnancy Diabetes-induced placental malformation decreases placental efficiency and fetal size. Hyperglycemia disrupts rat trophoblast stem cell differentiation into invasive trophoblast. Diabetes impairs intrauterine trophoblast invasion. Hyperglycemia adversely affects placental adaptations to hypoxia.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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