Placental Modifications Secondary To Maternal Hyperglycaemia Resulted In Impaired Pregnancy Outcomes

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ABSTRACT

INTRODUCTION: Gestational diabetes mellitus is a common metabolic disorder during pregnancy. This disease is characterized by persistent hyperglycaemia and is known to cause various complications to mother and foetus. The effects of hyperglycaemia on placental architecture had not been fully elucidated before. **OBJECTIVE**: This study is therefore aimed to investigate the progression of placental damage during hyperglycaemic insult at mid and late gestation. **METHODS:** Pregnant female Sprague-Dawley rats received 45 mg/kg body weight of intraperitoneal (i.p) Streptozotocin on gestational day (GD) 7, followed by 10% glucose drink for 24 hours to prevent hypoglycaemic fatality. Control (Con) animals were injected with 1 mL of citrate buffer vehicle. Hyperglycaemia was confirmed on GD13. Maternal weight, food intake and blood glucose levels were regularly monitored. Placentae were collected for morphological and histological analyses at GD15 (mid-gestation) and GD21 (term). **RESULTS:** Maternal hyperglycaemia reduced pregnancy weight gain by more than 39% ($p<0.01$) despite increased in food intake by 27% ($p<0.01$). At GD21, the hyperglycaemic (STZ) group demonstrated a 14% increase in foetal resorption with 28% reduction in foeto-placental weight ratio ($p<0.01$). There was a one-fold increase in the percentage of areas occupied by glycogen cells (GCs) at GD15 ($p<0.01$) and up to two folds at GD21 ($p<0.01$) in the junctional zone (JZ) of STZ groups. The percentage area of maternal vascular space (MVS) in the labyrinth zone (LZ) of the STZ group also failed to match the vascular development seen in the Con group ($p>0.05$). **CONCLUSION**: This study demonstrated that maternal hyperglycaemia resulted in poor pregnancy weight gain despite hyperphagia. This condition altered placental architecture by converting trophoblast cells into the GCs, reducing the amount of functional placental tissues. Deterioration in placental vascular remodelling could have resulted in placental insufficiency manifested by increased foetal resorption and reduced foetal body weight.

Keywords: Hyperglycemia; Rat placenta; Glycogen cell; Placental vascularization; Streptozotocin