18. THE INFLUENCE OF TYPE 2 PREGESTATIONAL DIABETES MELLITUS ON THE FETUS

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Introduction. The prevalence of type 2 diabetes among women of reproductive age is increasing. According to the latest data from the International Diabetes Federation in 2019, one in six live births is affected by hyperglycemia during pregnancy, that represents a risk for the intrauterine development of the fetus, and despite significant advances in glycemic control of pregnancies with diabetes, adverse outcomes for the fetus are still very common.

Aim of study. To elucidate the biochemical mechanisms of influence of type 2 diabetes on the intrauterine development of the fetus with the aim of improving diagnosis, treatment and preventing the occurrence of adverse effects.

Methods and materials. To achieve the proposed goal, a bibliographic search was performed using 10 bibliographic sources, between the 2018-2023, including those of the Medical Scientific Library of USMF "*Nicolae Testemitanu*", data of the electronic libraries such as PubMed, MedScape, Medline, Diabetes Care and Diabetologia.

Results. Maternal hyperglycemia increases apoptosis in the embryo, which is specifically observed in neuroepithelial cells. An important role also has oxidative stress, which increases the BAX:BCL-2 ratio- indicator of apoptosis level in glioma cells, associated with the increase of cytochrome C in mitochondria and the activation of caspase 3 in embryonic cells. Increased glucose transfer from mother to the fetus induces fetal hyperglycemia, pancreatic β-cell hyperplasia, and consequently fetal hyperinsulinemia (FHI). Insulin influences glucose uptake and lipogenesis via the glucose transporter GLUT-4, expressed in fetal adipose tissue and induces fetal macrosomia. The placenta acts as a passive conduit for maternal glucose to the fetus, especially towards the end of gestation. Therefore, increased glucose transport from diabetic mothers, together with FHI, stimulates fetal triacylglycerol formation and deposition of excess fetal adipose tissue. FHI can delay lung development in the fetus of a diabetic mother. A physiological level of insulin plays a role as a stimulatory hormone in the synthesis of surfactant, but a high level of insulin can inhibit the lipid components of the surfactant: phosphatidylcholine (PC), phosphatidylglycerol (PG) and phosphatidylinositol (PI) and low level of surfactant protein expression.Thus, it decreases the ability of surfactant to reduce alveolar surface tension, increasing the risk of respiratory distress syndrome (RDS). Pregnant women usually have a pathological weight gain, associated with a high production of pro-inflammatory cytokines and adipokines - TNF-α, IL-1β, IL-6, responsible for insulin resistance.

Conclusion. Maintaining maternal glucose in optimal parameters is essential for the normal development of the fetus by reducing the risk of congenital malformations and preventing postpartum complications.

Keywords. Maternal hyperglycemia, diabetes, insulin resistance, fetal hyperinsulinemia.