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PATHOPYSIOLOGICAL CONDITION IN INFANTS OF DIABETIC MOTHERS

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Annotation
The global incidence of diabetes during pregnancy is rising. Infants born to mothers with diabetes mellitus (DM) in pregnancy are predisposed to short- and long-term complications. The extent of these complications depends on the type of diabetes (pregestational or gestational); onset and duration of glucose intolerance; severity of diabetes (degree of glucose intolerance, presence of complications); and therapeutic control.

Keywords: hyperglycemia, macrosomia, insulin, hyperinsulinemia, hypoxia, hyperbilirubinemia.

Maternal plasma glucose can cross the placenta by facilitated diffusion, maternal hyperglycemia leads to fetal hyperglycemia. Early-onset placental vasculopathy may cause growth restriction and may alter organogenesis (diabetic embryopathy) with recognizable patterns of congenital anomalies. Poorly controlled GDM and hyperglycemia can cause macrosomia.
In the second trimester, the fetal pancreas responds to the rise in glucose levels by producing insulin, leading to fetal hyperinsulinemia. Fetal hyperglycemia and hyperinsulinemia drive the multisystemic pathology present in IDMs. Elevated fetal insulin levels, upregulated glucose transporters, and increased intracellular glucose concentrations can enhance mitochondrial oxidative phosphorylation. The resulting increase in the production of reactive oxygen species can contribute to diabetic embryopathy. Chronically upregulated fetal metabolic rate and oxygen

consumption lead to relative hypoxemia, which in turn elevate proangiogenic factors such as leptin, vascular endothelial growth factor, fibroblast growth factor 2, and matrix metalloproteinases (MMPs) such as MMP14 and MMP15, which lead to altered tissue histoarchitecture and epigenetic changes.

IDMs are at a higher risk of hypoxia and ischemia at peri- and intrapartum stages than are infants of nondiabetic mothers. Fetal hypoxemia raises erythropoietin levels, which can stimulate erythroid progenitor growth that is already activated by hyperinsulinemia. These two phenomena can lead to polycythemia and consequently to neonatal hyperviscosity syndrome. Many fetal regions have concomitant tissue hypoxia due to increased glycation of hemoglobin and low concentrations of 2,3-diphosphoglycerate, which increase erythropoietin expression and red blood cell (RBC) production. Some premature infants develop polycythemia, and the larger RBC mass and turnover may increase the bilirubin loads to exceed the capacity of the developing liver and cause hyperbilirubinemia.

In the fetal metabolic environment, even small changes can induce epigenetic modifications with altered gene expression and phenotypic changes. The risk of diabetic embryopathy increases with prolonged fetal exposure to maternal hyperglycemia. Insulin binds to the type I insulin-like growth factor receptor to induce intracellular phosphorylation pathways, which activate cellular growth-promoting factors. In IDMs, myocardial hypertrophy leads to cardiomegaly with asymmetric, disproportionate septal hypertrophy. There is also widespread cellular apoptosis with altered genetics and epigenetic systems, resulting in dysmorphogenesis.

In conclusion, the most important risk factors for maternal diabetes are maternal age ≥35 years, urban residence, and low socioeconomic status. Prevalence varies by race and ethnicity; Black women have higher rates of pregestational diabetes, whereas Asian women are more susceptible to GDM. Latinas are at higher risk of both pregestational diabetes and GDM. Obesity, family history of diabetes, high parity, and older age at first birth increase the risk of gestational diabetes.

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