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THE EFFECT OF GESTATIONAL DIABETES MELLITUS ON THE FETUS

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ABSTRACT

Gestational Diabetes Mellitus (GDM) is a prevalent metabolic disorder that occurs during pregnancy, characterized by glucose intolerance with onset or first recognition during gestation. This condition has significant implications for both maternal and fetal health. This review aims to explore the effects of GDM on fetal development and outcomes, examining the underlying mechanisms and potential long-term consequences for the offspring.

Keywords

Gestational Diabetes Mellitus, insulin resistance, type 1 diabetes mellitus, macrosomic infant, obesity

Introduction

Diabetes mellitus is an endocrine metabolic disease that is caused by chronic hyperglycemia resulting from a disorder in the secretion and action of insulin. Diabetes mellitus in pregnant women is of three types (in accordance with WHO recommendations): 1) type 1 diabetes mellitus (non-insulin dependent), diagnosed before pregnancy; 2) diabetes mellitus type 2 (insulin-dependent), diagnosed before pregnancy; 3) gestational diabetes mellitus (diagnosed after pregnancy). Gestational Diabetes Mellitus (GDM) is a condition in which non-diabetic women exhibit high blood glucose levels during pregnancy. It is one of the most common complications of pregnancy, affecting approximately 7% of all pregnancies globally. The rising prevalence of obesity and sedentary lifestyles has contributed to an increase in GDM cases. Understanding the impact of GDM on the fetus is crucial for developing effective management strategies and mitigating adverse outcomes.

The pathophysiology of GDM involves insulin resistance and impaired insulin secretion. During pregnancy, hormonal changes, including increased levels of placental hormones such as human placental lactogen, progesterone, and cortisol, contribute to insulin resistance. In women with GDM, the pancreas cannot produce sufficient insulin to overcome this resistance, leading to hyperglycemia. During pregnancy, the consumption of glucose by the pregnant woman's body slows down so that it reaches the fetus in sufficient quantities. Glucose transport across the

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placenta is carried out by glucose transporters. This is accomplished through the formation of a number of processes. First of all, these are placental hormones (placental lactogen and progesterone), which have a blocking effect on maternal insulin and reduce the activity of enzymes involved in glucose metabolism. The production of these hormones increases during pregnancy and reaches its maximum level in the third trimester. In the second trimester, the response of tissues to the effects of insulin decreases - insulin resistance with hyperinsulinemia is formed. Insulin resistance is not the only etiological factor in the development of gestational diabetes. A decrease in insulin secretion and the predominance of bound insulin are of great pathogenetic importance. As a result of a decrease in insulin secretion and insulin resistance, the metabolism of glucose, fatty acids, amino acids is disrupted, acetone bodies are formed and oxidative stress is formed.

Impact of GDM on Fetal Development

Against the background of diabetes mellitus, primarily from the second trimester of pregnancy, features in the development and growth of the fetus are revealed, which by the 24th–26th week of gestation are clearly divided into three typical patterns. The first is characterized by intrauterine growth retardation and ends with the birth of a low-birth-weight child with vaguely expressed signs of diabetic fetopathy. The second clinical picture develops from the 26th–28th week of gestation. Pregnancy ends with the birth of children with average anthropometric parameters without pronounced signs of diabetic fetopathy. In the third case, starting from 26 weeks of pregnancy, the fetus is characterized by a significant excess of anthropometric population norms, pregnancy ends in the birth of children with macrosomia and pronounced signs of diabetic fetopathy. Maternal hyperglycemia leads to increased glucose transfer to the fetus through the placenta, resulting in fetal hyperglycemia. This stimulates the fetal pancreas to produce more insulin, a condition known as fetal hyperinsulinemia. Elevated fetal insulin levels promote excessive growth and fat deposition, leading to macrosomia (birth weight > 4000 g). Macrosomic infants are at risk for birth complications such as shoulder dystocia, brachial plexus injury, and cesarean delivery. Although the risk of congenital anomalies is primarily associated with pregestational diabetes, poorly controlled GDM in the first trimester can increase the risk of congenital malformations. These may include cardiovascular defects, neural tube defects, and caudal regression syndrome. Against the background of diabetes mellitus, significant changes in local and general immunity occur, which, along with glucosuria, contributes to the development of urinary tract infections in 16% of pregnant women. Asymptomatic bacteriuria in patients occurs 2–3 times more often than in the population, and clinically pronounced pyelonephritis is diagnosed in 6%.

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The birth of a healthy child largely depends on the proper functioning of the mechanisms that promptly trigger labor and then ensure spontaneous delivery. Severe course and late complications of diabetes mellitus, polyhydramnios, gestosis and urogenital infections are the main causes of premature birth in pregnant women with diabetes mellitus. Their frequency depends on the type of diabetes and ranges from 25 to 60%. The incidence of premature birth in patients with type 1 diabetes mellitus is 60%; timely spontaneous labor develops in only 23% of women. In approximately 20% of cases, childbirth is carried out promptly due to the acute development of polyhydramnios and the critical condition of the fetus. GDM increases the likelihood of preterm birth, which can lead to complications such as respiratory distress syndrome (RDS). The fetal lungs may be less mature due to the high insulin levels, which interfere with surfactant production, essential for lung function post-birth.

Newborns from mothers with diabetes, despite their large body weight, are considered premature and require special care. In the first hours of life, attention should be paid to identifying and controlling respiratory disorders, hypoglycemia, acidosis and central nervous system damage. In 50% of newborns from mothers with diabetes mellitus, dysglucosemia syndrome is observed in the early neonatal period. The manifestations of this syndrome in the form of hypo- and hyperglycemia depend on the severity and degree of compensation of the mother's underlying disease, the presence of complications of pregnancy (preeclampsia) and childbirth (trauma), and the nature of adaptation disorders in newborns in the early postnatal period. Children with diabetic fetopathy adapt much worse in the early neonatal period, which is expressed in the development of conjugation jaundice, toxic erythema, significant loss of body weight and slow recovery.

Postnatal hypoglycemia is a common complication in infants born to mothers with GDM. After birth, the neonate's insulin levels remain high while the maternal glucose supply is cut off, leading to a rapid drop in blood glucose levels. This can cause symptoms such as jitteriness, seizures, and in severe cases, neurological damage. Additionally, electrolyte imbalances, such as hypocalcemia and hypomagnesemia, can occur.

Long-Term Effects on Offspring

Offspring of mothers with GDM have a higher risk of developing metabolic syndrome and Type 2 diabetes later in life. The intrauterine environment characterized by hyperglycemia and hyperinsulinemia is believed to predispose these children to insulin resistance and beta-cell dysfunction. There is growing evidence that maternal GDM can affect neurodevelopmental outcomes in children. Potential impacts include cognitive impairments, attention deficit hyperactivity disorder (ADHD), and autism spectrum disorders (ASD). At an early age, no more

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than 1/4 of all patients are under the dynamic supervision of a neurologist, since the parents of these children consider their development to be quite satisfactory and have no complaints. However, 78.4% of children have disorders of the central nervous system, with the leading syndrome (16.4%) being a lag in neuropsychic development. The exact mechanisms are not well understood but may involve alterations in the fetal brain structure and function due to hyperglycemia and inflammation.

Particularly noteworthy is the increase in the incidence of endocrine pathology with age. In the structure of morbidity, malnutrition (obesity of varying severity) comes to the fore. Studies have shown a correlation between maternal GDM and increased risk of obesity in the offspring during childhood and adolescence. The mechanisms involve alterations in fetal adiposity, appetite regulation, and metabolic programming.

Management and Prevention

Effective management of GDM involves regular monitoring of blood glucose levels, a balanced diet, physical activity, and insulin therapy if necessary. Early diagnosis and tight glycemic control can significantly reduce the risk of adverse fetal outcomes. Prenatal care should also include education on lifestyle modifications to prevent GDM in future pregnancies.

Conclusion

Gestational Diabetes Mellitus has profound implications for fetal health, both during pregnancy and in the long term. Understanding these effects is essential for developing strategies to mitigate risks and improve outcomes for both mother and child. Further research is needed to elucidate the precise mechanisms by which GDM affects fetal development and to develop targeted interventions. Thus, monitoring children born to mothers with diabetes is a medical and social problem that determines not only the condition of children at birth, but also their quality of life in general. All this requires new approaches to prevention and follow-up of this cohort of patients throughout all periods of childhood.

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