FEATURES OF DIABETES MELLITUS DURING PREGNANCY

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ABSTRACT

In modern conditions, pregnancy and childbirth in patients with diabetes mellitus pose a number of serious problems for endocrinologists and obstetricians. On the one hand, pregnancy complicates the course of the disease, complicating the normal intrauterine development of the fetus, which requires constant monitoring of the condition of the pregnant woman. On the other hand, diabetes mellitus is the cause of a variety of obstetric pathologies. According to WHO recommendations, the following types of diabetes mellitus in pregnant women are distinguished: 1) type I diabetes mellitus, diagnosed before pregnancy; 2) type II diabetes mellitus diagnosed before pregnancy; 3) gestational diabetes (this name includes any impaired glucose tolerance that occurs during pregnancy). The prevalence of all types of diabetes mellitus among pregnant women is 3-3.5%. The incidence of diabetes mellitus type I and II is 0.5-0.7%, diabetes in pregnant women - 1-3%. Perinatal mortality with a combination of diabetes mellitus and pregnancy is 3-5%, and in the absence of such a combination - 1-2%.

INTRODUCTION

The clinical course of diabetes mellitus during pregnancy has a number of features. Even in healthy women, pregnancy is accompanied by significant changes in carbohydrate and fat metabolism. Thus, the level of glucose in the blood plasma of a pregnant woman progressively decreases as the duration of pregnancy increases. The reasons for this are: 1) increased absorption of glucose by the placenta; 2) inhibition of gluconeogenesis due to a decrease in the level of amino acids in the blood. In later

stages of pregnancy, lipolysis increases, leading to increased levels of triglycerides and free fatty acids in the blood. The consequence of this is an increase in the processes of ketogenesis. Activation of ketogenesis also occurs due to the effect of placental hormones on maternal hepatocytes. Ketone bodies freely penetrate the placenta and are utilized by the liver and brain of the fetus as an energy source. In the first half of pregnancy, there is an increase in the sensitivity of maternal tissues to the action of insulin due to a decrease in fasting glucose levels. In the second half of pregnancy, the secretion of placental hormones, which have a counter-insular effect, sharply increases, which significantly reduces the utilization of glucose by maternal tissues. This results in a significant increase in postprandial blood glucose levels in pregnant women compared to non-pregnant women. Constant moderate hyperglycemia causes the development of physiological hyperinsulinemia. Hyperinsulinemia is also aggravated by the development of insulin resistance due to the high activity of placental hormones. It is known that maternal insulin does not cross the placenta. By the 10-12th week of pregnancy, β-cells first appear in the fetal pancreas, providing insulin secretion. The presence of moderate hyperglycemia in the mother causes an increase in the level of glucose in the blood of the fetus, which causes functional hyperinsulinism in the fetus with the risk of developing macrosomia and hypoglycemia. If a pregnant woman has diabetes mellitus, the course of the disease has a number of features that require correction of glucose-lowering therapy depending on the stage of pregnancy. Thus, the first half of pregnancy is accompanied by an improvement in the course of diabetes mellitus due to the transfer of glucose from the mother’s bloodstream to the fetus, which is expressed in a decrease in the need for insulin preparations. On the contrary, the second half of pregnancy is characterized by an increase in hyperglycemia and glucosuria, a risk of developing diabetic ketoacidosis, and an increased need for insulin. This is explained by the activation of the functioning of the placenta, which secretes contrainsular hormones (placental lactogen, estrogens, progesterone). Their action leads to a decrease in the body’s sensitivity to both endogenous and exogenous insulin. After the 34-36th week of pregnancy, the need for insulin, as a rule, decreases again. Obviously, the reason for this is the high activity of [3-cells of the fetal pancreas, which causes a very significant consumption of sugar from the mother’s blood. During childbirth, a pregnant woman’s glycemia may increase, and failure to adjust the insulin dose can lead to ketosis. However, immediately after childbirth, stress relief and loss of secretion of contrainsular hormones lead to a significant decrease in blood sugar. In this case, the need for insulin for a short time (3-4 days) becomes less than before pregnancy. After this, the course of diabetes returns to normal and is characterized by a transition to the previous dose of insulin (as before pregnancy).

The presence of diabetes mellitus in a pregnant woman poses a serious danger to

the health and life of a woman. It should be noted that pregnancy contributes to the early onset and progression of complications of diabetes mellitus - retinopathy, nephropathy and polyneuropathy. In addition, there is a high risk of developing diabetic ketoacidosis, since even in the absence of severe hyperglycemia, the level of ketone bodies in the blood plasma increases significantly. The labile course of diabetes mellitus in a pregnant woman can lead to the development of severe hypoglycemic conditions, which are sometimes aggravated by intensive insulin therapy. Particular attention should be paid to changes in the blood coagulation system with a combination of diabetes mellitus and pregnancy.

Both pregnancy and diabetes mellitus alone are known to predispose to the development of disseminated intravascular coagulation (DIC). Naturally, when they are combined, the risk of this syndrome increases significantly. The central link in the development of DIC is the vascular-platelet component of hemostasis - an imbalance of the thromboxane-prostacyclin system develops with a predominance of the vasoconstrictive and aggregative effects of thromboxane. There are two possible options for the development of internal combustion engines. With one of them, a predominant activation of the platelet component occurs without a significant deviation of other components of hemostasis. In another case, an increase in the aggregation properties of platelets is observed in combination with the activation of procoagulants, a decrease in antithrombin activity, and an increase in the final products of fibrinolysis.

During pregnancy, there is an increase in the overall coagulation potential, the functional activity of platelets with a moderate decrease in their number, a decrease in the fibrinolytic activity of the blood and the main inhibitor of blood coagulation - antithrombin III. These changes are adaptive in nature and are aimed at adequate formation of the fetoplacental complex and limiting blood loss during childbirth. The mechanism of the noted changes is the invasion of trophoblast cells into the wall of the spiral arteries of the uterus, replacement of the internal elastic membrane and media with fibrinoid, disruption of the integrity of the endothelium with exposure of collagen structures, and the formation of the intervillous space. These physiological changes activate the hemostatic system and lead to changes in hemodynamics in a pregnant woman.

The most severe complication of pregnancy with diabetes mellitus is intrauterine fetal death, the frequency of which averages 12-12.5% and depends on the degree of compensation of the disease. The main causes of fetal death: hypoglycemia or ketoacidosis in the mother, diabetic damage to the placental vessels with the development of hormonal deficiency. Even with good compensation of diabetes mellitus, intrauterine fetal death is detected in 3-4% of cases.

In patients with diabetes, natural delivery at 38-39 weeks of pregnancy is always

preferable. However, in some cases, delivery by cesarean section is indicated. Indications for it are: severe vascular complications of diabetes mellitus (nephropathy and retinopathy) progressing during pregnancy; labile course of diabetes; progressive fetal hypoxia; severe toxicosis of pregnancy; breech presentation; the gigantic size of the fetus, revealed by ultrasound. Artificial delivery is carried out at a gestational age of at least 36-37 weeks. Delivery at a shorter period leads to a significant increase in the risk of developing respiratory disorders in the fetus due to the functional immaturity of the lung tissue. Thus, the presence of diabetes mellitus in a pregnant woman poses a large number of complex questions for the obstetrician-gynecologist and endocrinologist and requires significant efforts both to maintain the pregnancy and to ensure optimal delivery and viability.

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