



NEW APPROACHES TO TIMELY DIAGNOSIS OF DIABETES IN PREGNANCY

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Abstract: Nowadays, pregnancy is more and more often accompanied by carbohydrate metabolism disorders, which, unfortunately, are not detected in time. This is due to the lack of a unified approach to the diagnosis of gestational diabetes in Russia. Thanks to the Russian National Consensus "Gestational Diabetes Mellitus: Diagnosis, Treatment, Postpartum Care", it will be possible to avoid most pregnancy complications in women at risk of gestational diabetes. The article highlights The relevance of detecting carbohydrate metabolism disorders during pregnancy, possible and most frequent complications that occur in pregnant women with pregestational and gestational diabetes, a new approach to early diagnosis and management of women with newly detected changes in carbohydrate metabolism.

Key words: diabetes mellitus, pregnancy, diabetic fetopathy, treatment.

Pregnancy is a physiological state of a woman, but at the same time it puts a tremendous load on all processes in the body. Often, pregnancy as an indicator reveals hidden, compensated disorders in the functioning of certain organs and systems outside pregnancy. Metabolism in general, and carbohydrate metabolism in particular, are the first to undergo changes. 0.3% of women of reproductive age suffer from diabetes, in 0.2-0.3% of all pregnancies a woman initially has diabetes mellitus, and in 1-14% of cases, pregnancy is complicated by the detection of gestational diabetes. According to the WHO, the incidence of gestational diabetes mellitus (GDM) is 3-17.8% in the world, and 6.3% or more in Russia [1]. Such variability in the incidence of gestational diabetes mellitus is explained by the lack of uniform criteria for diagnosing GDM throughout the world and in our country, as well as non-specific clinical manifestations, and sometimes asymptomatic course of the disease. The relevance of the problem of carbohydrate metabolism disorders during pregnancy is also associated with the complications that can develop in the mother and her fetus at different stages of gestation. Moreover, undesirable outcomes for the mother and fetus to a greater or lesser extent occur in any variant of carbohydrate metabolism disorders, whether it is pregestational diabetes mellitus of the first or second type or impaired glucose tolerance existing before pregnancy, or changes are diagnosed only during pregnancy. For example, in systematic reviews of group studies, it was shown that gestational diabetes increases the risk of developing type 2 diabetes mellitus after a few years by 7 times, and the fact that preeclampsia is detected in a pregnant woman without diabetes increases this risk by 1.8 times and increases the risk of hypertension by 3.4 times [2]. In order to better understand the processes that lead to the formation of complications of diabetes mellitus during pregnancy, it is necessary to understand how carbohydrate metabolism changes during pregnancy in a healthy woman. Pregnancy is a physiological stress test for the β cells of the pancreas, being a "diabetogenic factor" for the body. The synthesis of steroid hormones (placental lactogen, estrogens, progesterone) by the placenta, as well as an increase in the formation of cortisol by the adrenal cortex with a simultaneous change in the metabolism and tissue effect of insulin, the accelerated destruction of insulin by the kidneys



and the activation of placental insulin during pregnancy lead to a state of physiological insulin resistance (IR) with compensatory hyperinsulinemia. The consequence of IR and insufficient insulin secretion to overcome it is an increase in the concentration of glucose, free fatty acids, some amino acids and ketones in the blood plasma. Each of these indicators, in turn, is comparable to the body weight of the newborn and, in some cases, to an unfavorable pregnancy outcome [3]. Physiological pregnancy is characterized by two main features in glucose homeostasis. First, there is a continuous transfer of glucose to the fetus and placenta. This is done with the help of transplacental glucose transporters (GLUT), mainly GLUT1 and GLUT3. Clinically, this is expressed in the development of "jejunal" hypoglycemia, so glucose levels decrease faster in a pregnant woman than in a non-pregnant woman, while lipolysis and ketogenesis are activated [4]. Secondly, from the second trimester of pregnancy, the sensitivity of tissues to insulin decreases. Insulin resistance is formed, which is realized in resistance to the stimulating effect of insulin on glucose utilization by skeletal muscles and adipose tissue, as well as in the inhibiting effect of insulin on the production of endogenous glucose by the liver. Similar changes are typical for both women with normal body weight and pregnant women with an increased body mass index (BMI more than 29.9 kg/m²). Although maternal blood lipids pass through the placenta with difficulty, maternal plasma triglycerides (TAGs) and non-esterified fatty acids (NEFAs) correlate with fetal lipids, development, and fetal fat mass under certain conditions [5]. Long-lasting insulin resistance leads to gradual compensatory stimulation of insulin secretion by pancreatic beta cells, so fasting insulin levels (basal) and stimulated insulin levels (phases 1 and 2 of insulin response) are elevated during pregnancy when performing an intravenous glucose test. In addition, insulin clearance increases with gestational age [6-8]. It should also be noted that the insulin molecule does not cross the placental barrier from the mother to the fetus, which is especially important in women with pregestational diabetes and initially elevated blood glucose levels. Regardless of the type of carbohydrate metabolism disorder, the problems encountered by the mother and her fetus are largely similar and depend on the degree of hyperglycemia and the time of initiation of therapy. Thus, all pregnant women with diabetes mellitus are characterized by various options for decompensation of carbohydrate metabolism disorders, and first of all, diabetic ketoacidosis (DKA), which is a sign of an extreme degree of insulin insufficiency. DKA can occur at all stages of pregnancy, especially in patients with type 1 diabetes. Late pregnancy complications such as preeclampsia occur in 70% of women with pregestational diabetes mellitus. The reason for such a high incidence of preeclampsia in this category of pregnant women is associated with the development of microangiopathy, especially in patients with a history of the disease for more than 10 years. But even gestational diabetes, detected only during pregnancy, is associated with the development of preeclampsia in 43% of women, which indicates the onset of metabolic disorders and changes in the microvasculature long before the actual diagnosis of diabetes. Polyhydramnios is another obstetric complication seen in pregnant women with various types of diabetes mellitus. Polyhydramnios is often associated with preeclampsia, severe fetal malformations, and perinatal mortality. It is more common in women with pregestational diabetes (in 30-60% of cases), but it is also observed in 10% of pregnant women with GDM. It is believed that polyhydramnios in DM develops due to polyuria of the fetus as a result of an increase in the transfer of glucose from the mother to the fetus, impaired secretion and absorption of amniotic fluid, FPN, associated with fetal abnormalities and infectious complications. Pregnant women with pregestational and gestational diabetes have an increased



chance of developing spontaneous miscarriage and preterm birth, ranging from 15 to 31%, depending on the type of diabetes. Inflammatory diseases of the genitourinary tract in pregnant women with diabetes are also a common complication. Asymptomatic bacteriuria is observed 2 times more often than in pregnant women without diabetes. An atypical and asymptomatic course of pyelonephritis is possible, and its exacerbation occurs in more than half of pregnant women in combination with diabetic nephropathy. There was also an increase in the incidence of vulvovaginitis in pregnant women with diabetes, and more often candida, which is associated with impaired cellular and humoral immunity, glycosuria, and changes in the normal acidity of the vagina. Infectious inflammatory processes in the genitourinary tract in pregnant women with carbohydrate metabolism disorders contribute to the development of spontaneous abortions, intrauterine infection of the fetus and premature birth. Pregnant women with diabetes are also characterized by other obstetric complications. Often there is a lack of biological readiness of the pregnant woman's body for childbirth, premature discharge of amniotic fluid, abnormalities of labor, fetal asphyxia (high frequency of early delivery, immaturity of the fetus), clinically narrow pelvis, which is due to the high incidence of macrosomia, polyhydramnios. With concomitant diabetes mellitus, the need for cesarean section, including emergency section, is much more common than in healthy women, which is accompanied by an increased risk of surgical and postoperative complications. In addition to the development of possible pregnancy complications, women with pregestational and gestational diabetes also have an increased risk to their fetuses. The danger of fetal development disorders arises already at the stage of embryo- and fetogenesis. Until the 9th or 12th week of pregnancy, the embryo does not have its own insulin and does not penetrate the mother's insulin, and glucose passes unhindered. Maternal hyperglycemia leads to an increase in the concentration of glucose in the fetal bloodstream, which leads to an increased risk of congenital malformations (FRM). The prevalence of VPR in children from women with diabetes is at least 4 times higher than in the general population, reaching 6-8% in case of uncompensated hyperglycemia. Such congenital malformations as caudal dysplasia syndrome (absence or hypoplasia of the sacrum, coccyx, sometimes lumbar vertebrae, underdevelopment of the femurs), brain malformations (anencephaly - 9 times more), malformations of the MVP (aplasia of the kidneys, duplication of the ureters), heart - 5 times more often, transposition of organs, malformations of the bones of the facial skull are characteristic. Congenital malformations are not the only danger that awaits the fetus during pregnancy due to diabetes mellitus. Moreover, malformations are more often formed in fetuses against the background of poorly compensated pregestational diabetes, i.e. with hyperglycemia before pregnancy and in the first trimester. From the 12th week of the intrauterine period, the functioning of the fetal pancreas (pancreas) begins. Constant hyperglycemia in a woman during this period is the cause of reactive hypertrophy and hyperplasia of the pancreatic β cells in her fetus - hyperinsulinemia develops. Hyperinsulinemia leads to the formation of fetal macrosomia, which is the cause of a high incidence of birth injuries and asphyxia of newborns. In addition, increased insulin secretion inhibits synthesis lecithin in the fetal lungs and increases the risk of respiratory distress syndrome by up to 30% in newborns. In such children, the incidence of transient tachypnea is increased. Hyperinsulinemia also leads to a high risk of hypoglycemic conditions in the newborn, including severe ones, in the early postpartum period. In both pregestational and gestational diabetes mellitus, morphofunctional fetal syndrome is often formed – macrosomia and diabetic fetopathy, which includes characteristic signs (pastosity, hypertrichosis, purple skin coloration,



puffy full-blooded face (Cushingoid appearance) due to increased fat deposition, mainly in the upper part of the body (face, shoulder girdle), impaired postnatal adaptation, clinical symptoms of hypoglycemia, hypocalcemia, hypomagnesemia, respiratory distress syndrome, cardiomegaly in 30% of cases, CHD, other VPR, hepato- and splenomegaly) [9]. Diabetic fetopathy (DF) is a disease that manifests itself in a complex of phenotypic signs that developed in utero in the fetus during gestation. There is reason to believe that they appear from the early stages of pregnancy, when organs and systems are laid, under the influence of hormonal stimulation (lack of insulin, increased function of the adrenal glands, changes in the function of the thyroid gland) and metabolism (hyperglycemia, hypoproteinemia, hypertriglyceridemia, etc.) [10]. This is confirmed by the fact that signs of DF develop after 26 weeks. gestation, at 24 weeks. pregnant women with late miscarriages in abortions already have early manifestations of DF (body disproportion, characteristic appearance, large liver, etc.). The vast majority of children born to mothers with type 1 diabetes have DF (96%); 85% of newborns born to mothers with type 2 diabetes have signs of DF, and 49% of newborns with GDM have such signs [11]. Ultrasound can detect signs of diabetic fetopathy in a timely manner. This makes it possible to determine the tactics of pregnancy management in women with pregestational and gestational diabetes. Ultrasound examination shows a large fetus (abdominal diameter ≥ 75 th percentile), hepato-splenomegaly, cardiomegaly/cardiopathy, bicontour of the fetal head, edema and thickening of the subcutaneous fat layer, thickening of the cervical fold, newly detected or increasing polyhydramnios with the established diagnosis of GDM (if other causes of polyhydramnios are excluded).

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