PLACENTAL INSUFFICIENCY IN PREGNANT WOMEN WITH DIABETES MILLETUS

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Abstract. Placental insufficiency (PN) is a syndrome consisting of a complex of disorders of the trophic, endocrine and metabolic functions of the placenta, leading to its inability to maintain an adequate and sufficient exchange between the body of the mother and the fetus. Fetoplacental insufficiency can develop in a number of somatic diseases of a woman, chronic infections of endocrinopathies (diabetes mellitus (DM), hypo- and hyperfunction of the thyroid gland, pathology of the hypothalamus and adrenal glands) and a number of other pathological conditions. Among a number of factors that form the normal homeostasis of the fetus, of course, a significant role is played not only by the state of health of the mother, not only the genetic program of the development of the fetus, but also by the morpho-functional usefulness of the placenta.
In the development of this syndrome, two main pathways of formation can be distinguished. They can exist independently or be combined with each other:
1. Trophic insufficiency, in which the absorption and assimilation of nutritious foods is disturbed, as well as the synthesis of the fetus's own metabolic products.
2. Respiratory failure, consisting in impaired transport of oxygen and carbon dioxide.

Key words: Placenta, vascularisation, invasion, insulin, lipids.

Introduction. The placenta is a foetal organ situated between mother and foetus. It is essential for foetal growth and development. In addition to serving as a conduit for maternal fuels destined to nourish the growing foetus it fulfils a wide spectrum of other functions including the synthesis of various hormones and growth factors, detoxification of maternal xenobiotics, immunologic barrier and dissipation of thermic energy resulting from foetal metabolism. Owing to its position the placenta is exposed to regulatory influences of mother and foetus albeit at different surfaces, i.e. the microvillous syncytiotrophoblast membrane as well as the basal syncytiotrophoblast membrane and the endothelial cells.
Depending on the state of compensatory-adaptive reactions, PN is subdivided into compensated, subcompensated and decompensated.
In addition, chronic placental failure is divided into three forms: predominantly uteroplacental, isolated placental and predominantly fetoplacental forms.
Grades of chronic placental failure by severity can also be presented as follows:
1st degree - initial structural changes in the placenta. Without organometric shifts, the volume of macropathology is not more than 5%.
2nd degree - a clear tendency to a decrease in the mass of the placenta and fetus, macropathology of the order of 7-15%, variants of immaturity such as intermediate differentiated villi or dissociated development of cotyledons. Focal level of compensatory reactions.
3rd degree - severe placental hypoplasia, fetal or newborn death, macropathy more than 16-20%, early variants of pathological immaturity or the predominance of chaotic, sclerosed villi, weakness or absence of compensatory reactions.
The main clinical manifestations of PN are: intrauterine growth retardation; chronic fetal hypoxia; premature aging (maturation) of the placenta; insufficient weight gain of a pregnant woman; the threat of termination of pregnancy; premature birth; ante- and intrapartum fetal death.
Diabetes mellitus and pregnancy are extremely unfavorable combinations for both the mother and the fetus. According to the international classification of the 10th revision, diabetes during

pregnancy is divided into pregestational and gestational. Type I diabetes mellitus is of particular importance. However, in obstetric practice, it is necessary to take into account diabetes in terms of its severity (mild, moderate, severe) and the state of compensation (compensation, subcompensation and decompensation).

Materials and methods.

All cases were divided into the following groups:
Group 1 - placentas from 15 women with diabetes mellitus I with the birth of a live child.
Group 2 - 15 placentas of women with type II diabetes with antenatal and perinatal fetal death.

The aim of our study was a comprehensive multilevel study of the placentas of women with type I diabetes and type II diabetes.

Research results

The placentas of women with newborns with normal body weight are characterized by a round shape or in the form of an irregular oval with a weight of 540 ± 20 g. The area occupied by cavities, heart attacks and hematomas usually does not exceed 5-8%. In cases of complicated pregnancy, primarily with gestosis in the second half of pregnancy, the content of such areas increases.
The maternal surface of the placenta is medium- and large-lobed, with poorly expressed grooves, its color depends on the form of the disease: in conditions of latent and mild form of diabetes with uneven blood filling - with light and dark areas, moderate as a result of ischemia - pale, anemic, severe - full-blooded, red placentas. Some expansion of fibrinoid layers is noted in both the maternal and fruit surfaces. The fields of sclerosis are revealed. Also in the maternal surface, attention is drawn to the narrowing of the lumen of the vessels, especially the veins, which occurs due to the sclerosis of the vessel walls developing in this group. Both in the lumen of the vessels and outside it, an increase in the content of lymphocytes is observed.
When studying the placenta, special attention is paid to the villous tree. The fact of its uneven development has been determined. So, in some cotyledons, its significant branching was revealed, up to the 12th order. In these areas, the villi are usually full-blooded. Erythrocyte accumulations are also observed in the intervillous space. However, the area occupied by such villi did not exceed 25-30% of the total area. Among the rest of the villi, sclerosed (5-10%), edematous and fibrinoid changes were observed.
In these cotyledons, the terminal villi more often had a relatively good preservation of syncytiotrophoblast with its desquamation only in a part of the villi (20-25%). There were also clearly defined syncytiocapillary membranes and a fairly large number of syncytial nodules. However, in these areas there is a small amount of immature villi. Vascularization of villi is uneven and is in direct proportion to the severity, duration of the disease, clinical course and fetal condition.
With perinatal fetal death, in comparison with the control group (17.2 x 16.3 x 1.8 cm; 511 g), we noted an increase in both the weight (620 ± 15 g) and the size of the placenta (20 x 18 x 3.5 cm). The fruit membranes are usually greenish-gray in color. In some cases, with areas of hemorrhage. Attachment of the umbilical cord is either marginal or paracentral. There are areas with both white and red heart attacks, cavities, calcifications, occupying up to 10-12% of the area. Microscopically, both in the fetal and in the maternal surface, in a number of cases, a thickening of the fibrinoid layers is revealed. In the maternal surface, the vessels are partially or completely hardened. In the part of the vessels - blood clots. Leukocytes and erythrocytes were observed in the lumen. In the villous tree, intermediate villi and terminal villi of small caliber prevail. The syncytiotrophoblast is not damaged, but the content of syncytial nodules is low. The basement membranes are preserved. In the stroma, the number of areas with fibrinoid necrosis is increased. In the lumen of the capillaries, the content of lymphocytes increased significantly, and only individual erythrocytes were found. The intervillous space is enlarged. The content of fibrinoid in it is increased, glued villi are observed. Among the villi, sclerosed ones predominate.

Electron microscopic examination shows that the number of syncytiotrophoblast microvilli is reduced. They are also shorter and thinner. The number of syncytiotrophoblast nuclei is reduced. Preserved irregular shapes, with a predominant location of condensed chromatin near the nuclear envelope and in the form of separate islands throughout the entire area of the nucleus. In the cytoplasm of syncytiotrophoblast mitochondria are observed, mainly with vacuolization and destroyed cristae, lysosomes, a small number of free-lying ribosomes, vacuoles. Areas of necrosis increase. The basement membrane is preserved. A wide layer of collagen fibers is expressed behind it. In the stroma, areas with fibrinoid necrosis increase. A well-defined layer of collagen fibers is also observed near the basement membrane of the vessels. The basement membrane of the capillaries is loosened and is completely absent in some areas. Plasmolemma of endothelial cells is smoothed with fragmentary disorders. The number of nuclei in endothelial cells is reduced. They take on an irregular oval shape. Condensed chromatin is located in them both near the nuclear envelope and in the form of separate islets. Nuclear pores are well expressed. In the cytoplasm of endothelial cells - single mitochondria, lipid granules, fragments of the expanded endoplasmic reticulum, free-ice ribosomes and polysomes, vacuoles. In the lumen of the capillaries, mainly lymphocytes and up to 25% of erythrocytes are observed. Plasmolemma is also often destroyed in blood cells. A significant part of the vessels is sclerosed.

With neonatal fetal death, the mass of the placenta ranges from 240-250g. Microscopically, polymorphism in the villous tree is noted. So, along with villi with hyperplastic stroma and large syncytial nodules, sclerosed and edematous villi are found. Sclerosis of the walls, mainly of arterioles, is often determined in the vessels, and plasma impregnation is observed in small vessels. The basement membranes are thickened.

Conclusion. We observed infarction, hematoma, calcification, and fibrin on the maternal and fetal placental surfaces of women with DM. In addition, women with GDM and post-term infants had more calcium deposits on the maternal placental surface of the placenta, and mothers of female newborns had a larger number of calcium and fibrin deposits on the maternal placental surface.

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