MODERN PRINCIPLES OF PATHOMORPHOLOGY OF PLACENTAL DEFICIENCY

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Abstract. Placental insufficiency is a syndrome based on morphofunctional changes of the placenta, when it worsens, fetal growth slows down and hypoxia develops. Placental insufficiency is a common complication of pregnancy, it is observed in 50-77% of cases of inability to carry a fetus, in preeclampsia - 32%, in extragenital diseases - 25-45%, in addition of viral and bacterial infection - up to 60%. This article presents information about the modern principles of the pathomorphology of placental insufficiency.

Key words: placenta, deficiency, pathomorphology, pregnancy.

СОВРЕМЕННЫЕ ОСНОВЫ ПАТОМОРФОЛОГИИ ПЛАЦЕНТАРНОЙ НЕДОСТАТОЧНОСТИ

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Резюме. Плацентарная недостаточность – синдром, в основе которого лежат морфофункциональные изменения плаценты, при ее усугублении замедляется рост плода и развивается гипоксия. Плацентарная недостаточность - частое осложнение беременности, наблюдается в 50-77% случаев невозможности вынашивания плода, при гестозе - 32%, при экстрагенитальных заболеваниях - 25-45%, кроме вирусной и бактериальной инфекции - до до 60%. В данной статье представлены сведения о современных принципах патоморфологии плацентарной недостаточности.

Ключевые слова: плацента, недостаточность, патоморфология, беременность.

YO’LDOSH YETISHMOVCHILIGI PATOMORFOLOGIYASINING ZAMONAVIY TAMOYILLARI

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Buxoro davlat tibbiyot instituti

Annotatsiya. Plasenta yetishmovchiligi - yo'ldoshning morfofunksional o'zgarishlariga asoslangan sindrom bo'lib, u yomonlashganda homila o'sishi sekinlashadi va gipoksiya rivojlanadi. Plasenta yetishmovchiligi homiladorlikning keng tarqalgan asorati bo'lib, u homilani ko'tara olmaslik holatlarining 50-77 foizida, preeklampsiyada - 32 foizda, ekstragenital kasalliklarda - 25-45 foizda, virusli va bakterial infektsiyaga 60% gacha qo'shimcha ravishda kuzatiladi. Ushbu maqolada platsenta yetishmovchiligining patomorfologiyasining zamonaviy tamoyillari haqida ma'lumot berilgan.

Kalit so'zlar: platsenta, yetishmovchilik, patomorfologiya, homiladorlik.

Relevance. Placental insufficiency occurs in 10.3% of women who gave birth on time, and up to 49% of women who gave birth prematurely. Placental insufficiency causes retardation of fetal development in 60% of cases [1, 4]. Placental insufficiency according to the time and mechanism of development: primary - develops due to the violation of implantation and placentation processes before the 16th week of pregnancy, secondary - develops after the 16th week of pregnancy due to the effect of exogenous factors on the fetus and placenta. 1. Primary placental insufficiency (genetic, endocrine, infection - effect on zygote, blastocyst and placenta formation, enzyme deficiency in decidual tissue - ovarian dysfunction). Anatomical changes in primary placental insufficiency are manifested in placental structure, location, adhesion, vascularization defects, and chorion development disorders. Disorders of fetal development, birth defects. Clinically, the risk of the fetus breaking from the uterine wall increases, or the fetus terminates in the early periods by itself (abortion). Pathological non-differentiation of the placenta occurs in the following types. The type of embryonic teats, the reason: the reasons for the cessation of placental development in the early stages of embryogenesis: observed in toxicosis, diabetes, infections. Macroscopically, the placenta is enlarged, swollen,

lumpy. Microscopically, the papillae are multi-branched, the stroma is swollen, there are many Kashenko-Hoffbauer cells, there are many stromal channels, there are no syncytial tufts, there are papillae without vessels, the gap between the papillae is widened. Consequence: spontaneous abortion. Often placental anomaly, malposition of the navel, inability to carry the fetus and hypotrophy of the fetus [5]. The form of the intermediate type of teats: Reasons: the placenta has stopped developing in the middle period. Micro: 80-110 μm teats are abundant, lateral teats are few, small means there are mature and immature teats of intermediate form, often born before 37 weeks. Type of sclerosed chaotic teats: Causes: Develops in the 2nd trimester of pregnancy. Macro: the placenta is small, hypoplastic. Micro: many small papillae, few capillaries, stroma rich in cells, no syncytial folds, little or no embryonic papillae. In this case, spontaneous abortion in the fetus, antenatal death, severe gestosis, continues with an abundance of water. Consequences: placental hypoplasia, fetal hypotrophy are often observed. 40% fatality rate. Dissociated variant is the most common type. In the 2nd and 3rd trimester, irregular development of cotyledons is observed. It is observed in gestosis and diabetes. In addition to micro: mature teats, there are immature, embryonic and hypovascularized teats. In late toxicosis: dystrophy of the nipples and decidual tissue, hemorrhages between the nipples, white and hemorrhagic infarcts are twice as common. 2. Secondary placental insufficiency (exogenous effects on placental formation) develops in the second half of pregnancy, it becomes acute and chronic. The acute type is manifested by premature placental abruption, retroplacental hemorrhage, intervilli hemorrhage, decidual tissue hemorrhage, teat stroma hemorrhage, thrombi, infarction, fullness, teat angiomatosis. The chronic type is complicated by retardation of fetal development, chronic hypoxia, intrauterine death. Micro: compensatory changes in the placenta are mixed: resorbed, increase in terminal papillae, enrichment of capillaries, syncytiotrophoblasts, from involutive-dystrophic changes: increase in fibrinoid content, narrowing of the interpapillary space, fibrosis of papillae, vascular sclerosis, calcinosis), inflammatory changes, sometimes, from circulatory changes: infarction, thrombosis, hyperemia, decreased vascularization, obliteration of vessels, hyperplasia of vessels; delayed placentation: acceleration, deceleration, hypoplasia, pathological immaturity. Placental insufficiency can be compensatory or decompensatory depending on the change in its functional state.

Compensatory placental insufficiency - uterus-placental and feto-placental blood circulation is intact, it is a violation of metabolic processes in the placenta. Decompensatory placental insufficiency is defined as uterine-placental and feto-placental blood circulation disorders. Exogenous and endogenous factors are distinguished among the causes of placental insufficiency. Endogenous factors lead to various variants of the placentation process, resulting in the development of primary placental insufficiency due to impaired blood circulation and enzyme deficiency. Utero-placental and feto-placental blood circulation disorders and secondary placental insufficiency develop under the influence of a large number of exogenous pathological factors. Causes of primary placental insufficiency: genetic factors; bacterial and viral infection; endocrine factors; enzyme deficiency in decidual tissue. These reasons affect the anatomical structure, location, adhesion of the placenta, as well as angiogenesis and vascularization, formation of placental teats. These reasons are observed in case of inability to bear pregnancy, pregnancy after infertility. The causes of secondary placental insufficiency are pregnancy complications and extragenital diseases. There are many causes of placental insufficiency, and the risk factors leading to it may also be different. Social factors: mother's age is over 30, under 18, lack of nutrition, heavy physical work, mental and emotional state, occupational injuries, smoking, drinking and taking drugs. Extragenital diseases: cardiovascular, kidney, liver, lung, blood, endocrine diseases. Obstetric-gynecological factors: medical history: menstrual cycle disorders, infertility, inability to carry a fetus, premature births; gynecological: abnormalities of the reproductive system, uterine fibroids; obstetrical and extragenital diseases: gestosis, risk of miscarriage, multiplicity, placental malposition, infection, autoimmune diseases; Congenital diseases of mother and fetus: external factors: radiation, physical and chemical effects, drugs. Periods of uterine-placental blood circulation disorders: violation of endovascular migration of trophoblasts, failure of invasion of the non-mammary area of the placenta, violation of placental teat differentiation. Violation of endovascular migration of trophoblasts in the I-trimester of pregnancy leads to the development of necrosis on the placenta, and eventually to the death of the fetus. lack of invasion of the placental area causes incomplete reconstruction of the spiral arteries, resulting in placental hypoplasia and fetal hypotrophy. As a result, the spiral arteries are not transformed along their entire length, the decidual part is reconstructed, but the myometrial

part undergoes vasoconstriction without reconstruction. Violation of the differentiation of placental ducts is an important factor in the development of placental insufficiency. In this case, placental teats age quickly, develop incompletely, various types of teats appear on the placenta. The formation of the syncytiocapillary border is disturbed, the metabolism slows down due to the appearance of fibroblasts and collagen fibers in this border. Violation of blood circulation in the utero-placental system leads to ischemia, microcirculation, accumulation of immune compounds in the basal layer of trophoblasts, disruption of their structure, retardation of angiogenesis, disruption of the passage of oxygen and nutrients through the placenta. The condition of ischemia activates the system of peroxide oxidation of lipids in the tissue, as a result of which free radicals of oxygen appear, which also activates neutrophils to produce elastase and damage the vascular endothelium.

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