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Prognosis of Fetoplasentar Insufficiency in Pregnant Women with Preterm Obstetric Care and Optimize Preventive Measures

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Abstract

premature labor causes great problems for the health of the mother and child is. Fetoplasentar accounts for 20-30% of the kelip output cause of early delivery in recent years there is a shortage. Lack of a fetoplasentary system to date it remains one of the pressing problems of modern obstetrics.

Keywords: premature labor, fetoplasentar insufficiency, placental abruption morphofunctional diseases, extragenital diseases, ferroptosis, tocolytics, spasmolytics, amino acids.

The purpose of the study: the increase in the incidence of women is causing not only an increase in the incidence of asexual childbirth, but also an increase in perinatal mortality. 20-30% of them are associated with placenta pathology. Premature labor causes great health problems for the mother and child.Premature FPY , which occurs with a violation of the placenta process and leads to the underdevelopment of the placenta, is one of the causes of premature birth. Therefore, at present, it is relevant to develop and prevent modern diagnostic methods that allow you to identify pathological changes in the fetoplasentar complex at the initial, pre-clinical stage of the disease.Research materials and methods: Fetoplasentar insufficiency (FPI) is a complex of symptoms arising from morphofunctional changes in the placenta and is the result of the complex response of the fetus and placenta to various pathological conditions of the mother's organism. The main links in FPI pathogenesis are disorders of the uterine and fetoplasentar blood flow, disorders of the metabolic, trophic, hormonal functions of the placenta. Complications due to Fetoplasentar insufficiency include delay syndrome of intrauterine development of the fetus, acute and chronic fetal hypoxia, premature aging of the placenta , undeveloped pregnancy. FPI, which occurs with impaired placentation, is one of the causes of premature birth.

FPI incidence is observed in 20-30% of all births and 23% of cases in preterm birth.

Therefore, at present, the introduction of modern diagnostic methods into lactation and clinical practice, which allow to identify pathological changes in the fetoplasentar complex at the pre-clinical stage, is of undoubted interest.

Of undoubted interest is the basic concept of neurogenic regulation of protective-adaptive mechanisms that provide homeostasis in the mother-placentaplod system. Physiological processes in the body of pregnant women associated with a change in hormonal status include the proliferation of

Yani histammine of some mediators involved in the development of inflammatory and allergic reactions, increased flow of cytokines from fat cells of IL-3, il-4 interleukins occurs. Cytokines, in turn, facilitate the infiltration of eosinophils, T-lymphocytes and basophils into the nasal mucosa. Leukotrienes, which are secreted from infiltrating inflammatory cells, especially eosinophils, play a major role. Also, immunoglobulin leads to an increase in e - IgE (antibodies) and agronulocytes, which in turn can cause inflammation in various organs and systems in pregnancy. Histamine contributes to embryo–uterine interaction due to its vasoactive, differentiation, and growth-promoting properties, and elevated histamine levels in the blood in pregnancy are known to cause a variety of negative outcomes, such as fetal miscarriage risk, risk of premature birth, and preeclampsia. Histamine mediates vasoconstriction in the umbilical arteries. Increased systemic inflammation, which leads to endothelial dysfunction due to an increase in the concentration of anti-inflammatory substances from the dysfunctional placenta, is another important route. Also, from the placenta, sFlt-1 (soluble Fms-Like Terosine Kinase-1,soluble FMS-tyrosine kinase-1) is associated with activation of the Complement System, (sflt-1 function occupies a key place in its pathophysiology in antiangiogenic protein, soluble FMS-like tyrosine kinase-1 (sFlt-1). sFlt-1 is released from a number of tissues into the bloodstream, where it resists vascular endothelial growth factor and placental growth factor activity, leading to endothelial dysfunction) which can cause fat cells to release histamine.The high expression of the enzyme histidine decarboxylase, which produces histamine in the placenta, the presence of histamine receptors on the fetal-maternal border, and EHRF indicate the physiological role of histamine during pregnancy. The balance between histamine and diamine oxidase seems critical for uncomplicated pregnancies. A decrease in diamine oxidase activity leads to several heterogeneous complications of pregnancy, such as diabetes, the risk of miscarriage, and an underdeveloped pregnancy, and a violation of trophoblasty. Ferroptosis is a newly defined form of programmed cell death that is clearly defined by oxidative-active iron-dependent peroxidation of phospholipids containing polyunsaturated fatty acids and the inability to recover after lipid peroxidation. Recently, this specific form of lipotoxic cell death has been found to be associated with many human diseases, including ischemic-reperfusion heart damage, brain damage, acute kidney damage, cancer, and asthma. Interestingly, the parameters associated with ferroptosis are related to the physiology of the placenta and damage to the trophoblast. Such conditions include hypoxia-reperfusion during the development of the placenta, physiological contractions of the uterus or pathological changes of the placenta bed, excess iron in the trophoblast, preeclampsia, delayed fetal growth and signs of lipotoxicity in the pathophysiology of major placenta diseases such as premature birth, as well as decreased glutathione peroxidation capacity and decreased lipid peroxidation. glutathione peroxidase-4 plays a role in protecting placenta trophoblasts from ferroptosis damage. Our text is being researched on new possibilities for the regulation of ferroptosis as a means of protecting placenta trophoblasts from lipotoxic damage . Regulated cell death is a key component of many physiological and pathological processes. Necrotic cell death can occur in response to stress that damages cell membranes and organelles, leading to an uncontrolled cascade of fatal events. Intensive study of cell death patterns revealed various forms of programmed cell death cascades such as apoptosis, necroptosis, pyroptosis and entosis . Ferroptosis is a newly identified form of iron-dependent cell death caused by the accumulation of specific hydroxy-peroxidated phospholipids (Hp-PL) whose hydroxy-peroxidated fatty acids are arachidonic acid or adrenalic acid. It is usually bound to phosphatidylethanolamine (called hydroxy-peroxidized phosphatidylethanolamine or Hp-PE).). Ferroptosis therefore reflects an over-production of HP-PL or a lack of metabolic recovery ability to convert HP-PL into harmless forms of phospholipids. The accumulation of Hp-PL triggers a cascade of signals determined by unique morphological,

biochemical, and metabolic stages that differ from other forms of cell death.Considering the central role of the placenta in the development of the fetus, its growth, maternal-fetal interaction, maternal homeostasis and adaptation to injury during pregnancy, it is not surprising that the placenta plays a central role in common and severe complications of pregnancy, such as delayed fetal growth, preeclampsia, premature birth and divorce . It is important to note that several arguments support the assumption that ferroptosis may play a key role in placenta dysfunction based on the underlying diseases of pregnant women, where the placenta usually undergoes hypoxia in early pregnancy to reoxygenation and later uterine contractions before pregnancy, and hypoxia-reoxygenation disorders during childbirth associated with the pathogenesis of placenta dysfunction placental trophoblasts iron-rich, because it is actively transferred to the developing fetus through the placenta trophoblastic lipid peroxidation has been noted when the placenta is damaged, and low levels of glutathione peroxidase 4 (GPX4), the main enzyme that protects cells against the accumulation of harmful Hp species, are PLL and ferroptosis (see below), which are associated with human placenta dysfunction and preeclampsia . Indeed, recent studies have identified the role of ferroptosis in trophoblast damage and clinically significant dysfunction of the placenta . In this review, we describe biochemical pathways for the development of ferroptosis, unique aspects of placental biology that can increase sensitivity to ferroptosis, and our recent work on novel regulators of placental ferroptosis.

Therefore , we can take measures to detect and prevent fetoplasentar insufficiency early through comprehensive examinations to study it, as well as through ultrasound exographic examinations of the placenta in fetoplasentar insufficiency, by determining the parameters of dopplerometry of the uterine blood flow.

Research results: despite the fact that there are different treatment schemes for fetoplasentar insufficiency at different periods of pregnancy, the search for more effective ways to treat and prevent this pathology continues. fetoplasentar insufficiency pharmacotherapy includes the following groups of drugs: drugs that help relax the muscles of the uterus (B-adrenomimetics, spasmolytics,calcium channel blockers); drugs that improve the microcirculation and rheological properties of the blood (platelet aggregation reducing agents, angioprotectors, anticoagulants); drugs that correct metabolic disorders (mixtures of amino acids); drugs that increase the resistance of brain and fetal tissues to hypoxia (antigipoxants, antioxidants, nootropes,ozonotherapies) give an effective result.Vitamin C ,vitamin E ,selenium Macroelements are used if ferroptosis is detectable.

Conclusion: at the pre-clinical stage, it is relevant to develop and introduce into clinical practice modern diagnostic methods that allow you to identify pathological changes in the fetoplasentar complex. The data obtained allows you to identify another reason for the development mechanism of premature birth and form a prevention algorithm.

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