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Assessment of Fetoplasentar Deficiency in Pregnant Women Who Have Preterm Obstetric Care

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Abstract: an increase in female morbidity and a decrease in the index of women's health in reproductive age are approaching. Only 32% of pregnant women are somatically healthy. The emergence of a more problematic situation with reproductive health is increasing every day, the indicators of negative impact on the ability of modern socio-economic and environmental factors to give birth and healthy offspring and give birth.Including chronic gynecological diseases inflammatory endometriosis, endocrine diseases and infertility, there is a steady increase in the number of diseases. As a result, not only an increase in the incidence of asexual childbirth is observed, but also an increase in perinatal mortality, 20-48% of which are associated with placental pathology. At the same time, it also increases the factors that cause indicators. Premature labor causes great health problems for the mother and child.

Keywords: premature labor, fetoplasentar insufficiency, morphofunctional diseases of the placenta, extragenital diseases, Nootropes, tocolytics, spasmolytics, amino acids.

Preterm labor is said to stop prematurely during the gestational period from the 22nd week to the 37th week of pregnancy. Taking into account the gestational period of the fetus, we divide early childbirth into 3 periods 1) from the 22nd week to the 27th Week, 2) from the 28th week to the 33rd week, 3) taking into account the labor processes that occur from the 34th week to the 37th week, the following reasons are the risk factor of this process: socio-biological external extracorporeal factors related radioactive radiation). Obstetrics-gynecological Anamnesis data (the fact that the mother is too young is due to the fact that she is not physically ready for motherhood or older birth mothers, premature births from Anamnesis have been observed, there are many medical abortions, many fetal, aggravated obstetric Anamnesis), extragenital diseases (diabetes mellitus, pyelonephritis, myopia, peripheral vascular varicose veins, obesity, bronchial asthma, pneumonia, urogenital diseases, viral diseases) ,pregnancy complications (pregnancy hypertension, eclampsia preeclampsia, position of the placenta ahead, early displacement of the placenta, fetoplasentar insufficiency, excessive or excessive amount of rock water, feathering of the fetus, cervical insufficiency, uterine malformation). Fetoplasentar insufficiency is a complex of morphofunctional diseases of the placenta, which develop due to various extragenital and gynecological pathologies from the fetus and placenta, as well as complications of pregnancy, primary insufficiency occurs in the early stages of pregnancy (16-18 weeks), placenta formation and infectious viral, bacterial diseases, endocrine, it occurs during the stages of organogenesis under the influence of yatrogenic factors. Secondary fetoplasentar insufficiency initially develops with a normally formed placenta, under the influence of maternal factors or complications of pregnancy. Complications caused by perinatal morbidity and fetal perinatal death fetoplasentar insufficiency include delayed fetal intrauterine development syndrome, acute and chronic fetal hypoxia, premature placental aging , undeveloped pregnancy. Premature fetoplasentar insufficiency , 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 introduce into clinical practice modern diagnostic methods that allow you to identify pathological

changes in the fetoplasentar complex at the initial, pre-clinical stage of the disease. 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 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. But that's not all. An allergic reaction also leads to an increase in immunoglobulin E - IgE (antibodies) and agronulocytes, which in turn can cause inflammation with symptoms in various organs and systems (lungs, skin, eyes and nose). Histamine has been hypothesized to contribute 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 of premature birth, and preeclampsia. It is known that during pregnancy, the placenta is able to produce a large amount of diamine oxidase (histaminase), as a result of which the amount of serum histamine during this period is reduced, which contributes to the lesser manifestation of allergic conditions during pregnancy. But histamine mediates vasoconstriction in the umbilical arteries, and the severity of vessels and preeclampsia in human pregnancy increases by increasing the level of histamine in the blood. 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. According to some reports, delayed fetal development syndrome with an uncompensated form of placental insufficiency is diagnosed in about 70% of pregnant women. If the complications that lead to placental insufficiency cannot be treated or are not carried out at all, the ability of the placenta to compensate for the disorders that occur in it begins to weaken, and subcommensated placental insufficiency develops. The stage of fatigue of protective mechanisms begins, which leads to a delay in the development of the fetus of various weights. The development of pathological changes leads to decompensation of placental insufficiency, manifested by a serious delay in intrauterine development due to a sharp slowdown in the growth of fetal development to death. Mothers with manifestations of placental insufficiency during pregnancy can give birth to very viable and healthy children (this depends on the severity of the pathology and treatment). And yet they are complicated by diseases during the period of intrauterine development and in the newborn period.Of the possible complications, the following can be distinguished: hypotrophy(delay in fetal development); fetal hypoxia during childbirth, later manifested by impaired cerebral circulation in newborns; developmental abnormalities, certain developmental disorders joint dysplasia, heart defects, cystic lesions in internal limbs, fetal breathing disorders, pneumonia; frequent colds after arrival in the world; intestinal diseases; neurological diseases.

The severity of the condition of the newborn may also be due to the fact that with the development of hypoxia, the fetus causes premature birth, or sometimes an increase in hypoxia and, accordingly, the severity of the condition of the fetus, due to pathological signs, premature termination of pregnancy is necessary.These processes are detected and concluded in screening tests at 18-22 weeks of pregnancy.

The lack of a Fetoplasental system remains one of the pressing problems of modern obstetrics to this day. These include intrauterine growth delay and acute and chronic fetal hypoxia . One of the main methods of diagnosis of Fetoplasentar insufficiency is ultrasound dopplerometry, in which the parameters of blood flow in the fetal uterine arteries, umbilical artery and middle cerebral artery are determined . Indications of dysfunction in the maternal-placenta-fetal system also include: decreased fetal head, abdomen, limb size according to ultrasound fetometry, placenta thickness, and mismatch of maturation to the gestational age. Despite the fact that at different periods of pregnancy there are different treatment schemes for fetoplasentar insufficiency, the search for more effective ways to treat and prevent this pathology continues. Arterial hypotension is most often observed in the first three months of pregnancy, the state of anemia is the cause of the complication of this condition: early toxicosis (from 6.1% to 38.4%), gestosis (within 18.9% - 34.7%, premature childbirth (6.2% - 20.1%). one of the common complications of pregnancy is the early onset of fetoplasental insufficiency, which also leads to olip. In pregnant women, the saturation of the blood with oxygen decreases. It is known that arterial hypotension is an important factor in the appearance of intrauterine hypoxia.

Uteroplacental causes delayed fetal development (up to 35%) due to decreased blood flow. In this case, the condition improves by removing the pregnant woman from the state of anemia. We achieve an improvement in the condition by using amino acid-rich agents (Sermin, Cuamine), enriching the diet with more protein-rich agents, and again by introducing iron preparations in the second three months. It is in hypertensive cases that 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 diseases (amino acids, protein mixtures; drugs that increase the resistance of brain and fetal tissues to hypoxia. Intermittent or prolonged increases in myometrium tone are known to cause circulatory disorders in the fetoplasentar space due to decreased venous flow. In this regard, in patients with increased contractile activity of the uterus, b-adrenomimetics should be introduced into fetoplasentar insufficiency complex therapy, which have a tocolytic effect and improve uterine blood flow in small doses by reducing vascular resistance at the level of arterioles. To prevent the negative effects of B-adrenomimetics on the cardiovascular system, it is advisable to combine their intake with the intake of cardiotonic drugs. However,there is evidence that the use of low-dose acetyl salicylic acid (Aspirin, Tromb Ass, Thrombapol)is effective in preventing fetoplasentar insufficiency and preeclampsia in women with uterine blood flow disorders. Therefore, along with long-term antithrombotic therapy (Clexane), it often leads to an improvement in the indicators of the fetoplasental system. It is possible to use piracetam from nootropes ,riboxin from metabolizing enhancers, Dipyridamol, Sermin from amino acids, Tivortin, Actovegin, Group B vitamin complexes, Nifedipine from tokolitics, Verapamil, Magnesium sulfate from spasmolithics, papaverine preparations. Of the drugs that can be used in early pregnancy, the drug Tivortin is also considered harmless.The drug Tivortin has cytoprotective, antioxidant, antitipoxic, membrane-stabilizing, detoxifying properties, belongs to the amino acid class arginine (a-amino-δ-guanidinovalerian acid) and has an active and versatile cell regulator effect of many vital functions of the body, playing a certain role in maintaining hormonal balance in the body. Arginine is involved in the synthesis of blood, Proline, polyamine, agmatin, affects the processes of fibrinogenolysis and has a membrane depolarizing effect. Arginine is a substrate for the enzyme non-synthase and is an enzyme that catalyzes the synthesis of nitric oxide in endotheliocytes. The drug activates guanylate cyclase and increases the level of SGMF in the vascular endothelium, reduces the adgesia and aggregation of leukocytes and platelets into the vascular endothelium .VCAM-1 and MCP-1 inhibit the synthesis of adhesive protein, thereby preventing the formation and development of atherosclerotic plaques, inhibiting the synthesis of endotelin-1, a vasoconstrictor and stimulant of smooth muscle cells of the vascular wall. This helps to reduce uterine, vascular hypertension. Fetoplasentar improves blood circulation prevents the formation of atherosclerotic plaques. We can see that the heart rate of the fetus and the number of heartbeats of the mother are normalized 30-40 minutes after the time of inection to pregnant people. The course of treatment is at least 5 days. Oral 200 ml vial is done by ingesting 3

times 15ml for 10-15 days, or intravenous 100ml for 5 -7 days 1mahal per day for a faster effect. Analysis of modern literature suggests that fetoplasental insufficiency is often treated separately from its cause. Circulatory disorders in the vessels of the fetoplasental complex are noted due to hypovolemia, thrombosis and increased vascular resistance, and an insufficient supply is concluded.

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