Etiopathogenesis of Changes in the Hemodynamics of the Uterus and Placenta with Intrauterine Infection

Rakhimova Kamola Yuldashevna

Andijan State Medical Institute
Master of the Department of Obstetrics and Gynecology
E-mail: rahimova_kamola@mail.ru

ABSTRACT

Intrauterine infection of the fetus is an antenatal risk that causes fetal malformations and severe perinatal losses due to morphofunctional and hemodynamic disorders of the fetoplacental system.

Keywords: Fetoplacental insufficiency, intrauterine infection of the fetus, intrauterine fetal hypoxia.

Fetoplacental insufficiency, which develops as a result of hemodynamic disorders of the uterus, placenta and fetal system due to internal infection of the fetus, has become one of the leading causes of perinatal morbidity and intrauterine fetal death, an intractable problem in modern obstetrics.

When fetoplacental insufficiency is formed, infection is considered one of the reasons leading to a change in the perinatal outcomes of the health status of a pregnant woman and a newborn child.

The frequency of fetoplacental insufficiency ranges from 3-4% to 45%, and perinatal pathology is observed in 24.2%. [11,6,7]

Real Columbus (1559) was the first to propose the term placenta to use the fetus-mother-uterus system. The placenta is considered a multifunctional organ that performs transport, endocrine, immune, protective and other important functions necessary for fetal development. [17]

Fetoplacental insufficiency is the result of structural and functional disorders of the placenta, resulting in a lack of substances and gas exchange, blood circulation, respiratory function necessary for the formation of the fetus.

Internal infection of the fetus refers to a group of diseases that develop as a result of transmission of infection from an infected mother to the fetus.

The spectrum of microorganisms that cause intrauterine infection of the fetus is different, among them are more common those that cause urogenital infections, such as chlamydia, mycoplasma, cytomegalovirus, herpes simplex virus.

It is proved that the causative agents of internal infections of the fetus are more than 27 types of bacteria and many viruses, parasites and 6 types of fungi. According to researchers [8], chlamydia make up 17-50% of the main pathogens of antenatal infections, viruses (herpes simplex 7-47%, cytomegaloviruses 28-91.6%, enteroviruses 8-17%) and pathogens of intranatal infections make up the group of Streptococci of group B 3-12%, staphylococci 1-9%, fungi of the genus Candida 3-7%. [1,2,4,9,12]

As a result of damage to the placenta under the influence of infectious agents, its morphofunctional properties are lost and in 80.4% of cases leads to the development of fetoplacental insufficiency [3,4,12].

In the pathogenesis of fetoplacental insufficiency, first due to damage to the endothelium of placental vessels, invasion of trophoblasts, changes in the process and gestational restructuring of spiral arteries, later due to insufficiency of placental vessels, pathology of the hemostasis system, morphofunctional changes of the placenta (hypoplasia, dystrophy, obliteration and vascular spasm, microthrombosis, calcification, infarction), as well as arterial and a number of venous blood flow. [5,8,9,16]

The main factor in providing oxygen to the fetus is the uterus and placental system, in which, at the stage of maturation of the placental villi, as a result of stimulation of the proliferative ability of cytotrophoblasts due to hypoxia, transcription of vascular endothelial factor and the formation of villi capillarization by the type of placental branching. [13,11]

According to some authors, acute viral infections can cause fetal malformations and intrauterine death due to fetal and placental lesions. According to these studies, acute viral infections cause circulatory disorders in the uterus, placenta and fetus due to damage to the endothelium of the fetoplacental system vessels [5,7,16].

Morphological changes in fetoplacental insufficiency are characteristic of degenerative-dystrophic disorders, and its pathomorphological manifestations consist mainly in infarcts, fibrinoid deposits in the interstitial space, fibrin rearrangement of the villi epithelium, collagenization of the blood vessel wall and stroma, the number of small blood vessels in the terminal villi is decreasing. Capillary thrombosis, villous stroma edema, infarction, villous thrombosis are also characteristic. As a result, the structural system of the placenta leads to an increase in fibrinoid deposits, the formation of infarction and petrification of villi, a decrease in the volume of the interstitial space, the formation of other hemodynamic pathologies. [8,13]

The pathogenesis of disorders of uteroplacental circulation is characterized by a violation of blood flow or obstruction of venous blood flow in the interstitial space, as well as changes in rheological and coagulation properties of blood, a decrease in capillary blood flow of chorionic villi. [9,10]

In clinical practice, primary and secondary fetoplacental insufficiency are distinguished. Primary and secondary fetoplacental insufficiency can be acute and chronic. The mechanism of their development plays an important role in the blood circulation of the uterus, placenta vessels. [5,6,11]

In modern obstetrics, the classification of hemodynamic disorders of the uterus, placenta and fetal system is divided into 3 levels according to pathological changes: [13,15]

I. Level: Blood circulation in the vessels of the utero-placental-fetal system is intensively reduced.
II. Level: The placental system occurs when critical blood vessels suddenly change blood flow.
III. Level: Hemodynamic critical parameters (reversible deficiency of diastolic blood flow) and hypoxic changes in fetal condition are observed.

Structural changes in the placental system (inflammatory, dystrophic) cause a negative course of pregnancy as a result of internal infection of the fetus. During pregnancy I-II trimesters, the development of signs of fibroplasia, necrotic changes, clinical signs of circulatory disorders depends on the recurrence of infection. As a result, an infectious and inflammatory process develops in the fetoplacental system, and damage to the placenta, vessels of the trophoblast causes microthrombosis and microcirculation disorders. [8,16]

As a result of internal infection of the fetus, 19.4% of pregnant women are diagnosed with placental abruption. Also, pathologies of amniotic fluid are detected in 24.6%, dehydration and excess water develop. [5,10,11]

Intrauterine infection of the fetus is the 1st-3rd cause of death of newborns and causes perinatal losses from 11% to 45%. [9,12,16]

References:

1. Белоцерковцева Л.Д., Каспарова А.Э., Коваленко Л.В., Мордовина И.И. Перинатальные инфекции: настоящий взгляд на проблему. Современные методы профилактики вертикального пути передачи ВИЧ: Учеб. пособ. – Сургут: Изд-во СурГУ, 2008.
2. Буданов П.В., Стрижаков А.Н. Этиология, патогенез, диагностика и лечение внутриутробной инфекции // Вопросы гинекологии, акушерства и перинатологии. – М., 2010. – Т. 9. №3. – С.61-71.
3. Бочарова И.И., Аксенов А.Н., Башакин Н.Ф. и др. Итоги и перспективы научных исследований по проблеме внутриутробной инфекции новорожденных // Рос. Вест. акушера-гинек. – 2007. – № 5. – С. 60–63
4. Кан. Н.Е. Орджоникидзе В.Н. Современные представления о внутриутробной инфекции. Акуш и гин. 2014. 6. 3-5.
5. Коколина В.Ф., Картелищев а.В., Васильева О.а. Фетоплацентарная недостаточность: руководство для врачей. – М.: иД МеДПРаКТиКа – М., 2006. – 224 с.
6. Кулаков В.И., Орджоникидзе Н.В., Тютюник В.Л. Плацентарная недостаточность и инфекция. М., 2004. – 494 с.9.
7. Краснопольский В.И. Формирование и патология плаценты. М.: Медицина. 2007. – 112 с.10.
8. Лукьянова Е.В., Волощук и.Н. и др. Роль нарушений формирования ворсинчатого дерева в патогенезе плацентарной недостаточности // акушерство и гинекология. – 2009. – №2. – С.5-7.
9. Нисевич Л.Л Талалаев А.Г Каск Л.Н Внутриутробная инфекция мать-плацента – плод. // Детские инфекции. 2008. С. 9-13.
10. Павлова Н.Г., Аржанова О.Н., Зайнулина М.С. и др. Плацентарная недостаточность// Учебнометодическое пособие / Под ред. Э.К. Айламазяна. – Санкт-Петербург., Изд – во Н – Л.-2007. – 32 с.
11. Пустотина О.А., Бубнова Н.И., Ежова Л.С., Младковская Т.Б. Клиникоморфологическая характеристика фетоплацентарного комплекса при много- и маловодии инфекционного генеза // Акуш. и гинек. – 2008. – № 2. – С. 19–22
12. Самсыгина Г.А. Современные проблемы внутриутробных инфекций. // Педиатрия. 2010. С. 34-35.
13. Серов В.Н. Плацентарная недостаточность // Трудный пациент. – 2005. – Том 3. – № 2. – С. 17-20.
14. Сидорова И.С., Макаров И.Ю., Матвиенко Н.А. Внутриутробные инфекции. – М.: МИА, 2006. – 176 с.
15. Стрижаков а.Н., Тимохина Т.Ф., Баева О.Р. Фетоплацентарная недостаточность: патогенез, диагностика, лечение //Вопросы гинекологии, акушерства и перинатологии. – 2003 – Т.2 – №.– С.2-11
16. Шокирова С.М. Ибрагимова С.Р. Негматшаева Х.Н. Юлдашева О.С. Мирзаабдуллахожиева О.У. Современные аспекты этиопатогенеза и перспективы прогнозирования осложнений при внутриутробном инфицировании плода // Молодой ученый. 2014. С. 228-233.
17. Costerton W., Veeh R., Shirtliff M. The application of biofilm science to the study and control of chronic bacterial infections. Clin Invest 2003; 112: 1466–1477.