MODERN ASPECTS OF STUDYING THE ROLE OF THE FETOPLACENTAL SYSTEM IN PREMERATE BIRTH.

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Key words: prematurity, fetoplacental insufficiency, microbiota.
Annotation. Infection of the fetoplacental system is a widespread obstetric and gynecological pathology, which is one of the main causes of spontaneous miscarriages, often causes intrauterine fetal death and premature birth, is the leading cause of purulent-septic complications in puerperas and is one of the most significant etiological factors of neonatal morbidity and infantile mortality. The high medical and social significance of this problem has led to the growing interest of specialists in all issues related to the infectious pathology of the reproductive system of women, placenta, fetuses and newborns. Despite the fact that a number of works are devoted to the study of the fetoplacental complex (FPC) in preterm birth, most of them do not affect the relationship between the state of the fetus, biochemical parameters of the fetoplacental system (FPS) and the morphological state of the placenta.

СОВРЕМЕННЫЕ АСПЕКТЫ ИЗУЧЕНИЯ РОЛИ ФЕТОПЛАЦЕНТАРНОЙ СИСТЕМЫ ПРИ ПРЕЖДЕВРЕМЕННЫХ РОДАХ.

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Ключевые слова: недоношенность, фетоплацентарная недостаточность, микробиота.
Аннотация. Инфицирование фетоплацентарной системы – широко распространенная акушерско-гинекологическая патология, которая является одной из основных причин самопроизвольных выкидышей, нередко вызывает внутриутробную гибель плода и преждевременные роды, служит ведущей причиной гнойно-септических осложнений у родильниц и входит в число наиболее значимых этиологических факторов неонатальной заболеваемости и младенческой смертности. Высокая медико-социальная значимость данной проблемы обусловила нарастающий интерес специалистов ко всем вопросам, касающимся инфекционной патологии детородной системы женщин, последов, плодов и новорожденных. Несмотря на то, что изучению фетоплацентарного комплекса (ФПК) при преждевременных родах посвящен ряд работ, большинство из них при изучении не затрагивает взаимосвязи между состоянием плода, биохимическими параметрами фетоплацентарной системы (ФПС) и морфологическим состоянием плаценты.

Relevance of the topic. The problem of miscarriage continues to be one of the priority areas in obstetrics. The frequency of premature births is 5-12‰ of the total number of births. Their increase is associated with indicators such as perinatal and early neonatal morbidity and mortality. Premature babies account for up to 50% of stillbirths, 60-70% of early neonatal and 65-75% of infant mortality. Premature babies are born stillborn 8-13 times more often than full-term babies, and die in the first week of life 20-30 times more often. Perinatal mortality for preterm birth is 33 times higher than for full-term birth.

Among women of childbearing age, there are many people with extragenital infections and

foci of chronic infection. Their health index does not exceed 50%. Premature babies account for up to 50% of stillbirths (60-70% of early neonatal and 65-75% of infant mortality).

Currently, there are six groups of reasons leading to termination of pregnancy: endocrine, genetic, infectious, anatomical, immune, idiopathic. According to modern concepts, any unfavorable course of pregnancy is transformed for the fetus, first of all, into hypoxia, which develops with chronic placental insufficiency.

Despite the fact that a number of studies have been devoted to the study of the fetoplacental complex (FPC) during premature birth, most of them do not study the correlation between the condition of the fetus, immunological parameters and the morphological state of the placenta.

The lack of common views on immunological relationships in the mother-placenta-fetus system prevents effective pathogenetic therapy for preterm pregnancy. Currently, there are practically no studies devoted to the study of functional changes in the FPS with various types of immunocorrection for the purpose of preventing preterm birth. Despite the fact that a number of studies have been devoted to the study of the fetoplacental complex (FPC) during preterm birth, most of them do not address the relationship between the condition of the fetus, the biochemical parameters of the fetoplacental system (FPS) and the morphological state of the placenta.

At this time, extensive material has been accumulated regarding the morphological manifestations of placenta pathology. At the same time, numerous questions have arisen regarding the optimal methodological approaches to practical placentology, the rational interpretation of those structural and functional changes that occur in the placenta, fetal membranes and umbilical cord during infectious pathology of the mother and fetus, as well as the optimal interaction of obstetric and neonatal services. According to many researchers, depending on the nature of the damage to the placenta, 3 forms of FPN are distinguished: placental-membrane with a decrease in the ability of the placental membrane to transport metabolites; cellular-parenchymal - due to a violation of the cellular activity of the trophoblast; hemodynamic – a decrease in placental blood flow [7, 9].

However, a number of authors believe that in the clinic it is rarely possible to identify an isolated violation of one of these structures of the placenta, since they are closely interconnected, and a violation of one of them inevitably entails changes in other links. In addition, placental insufficiency (PI) develops mainly with pathological variants of maturation, manifested by changes in all structural elements of the placenta. These include premature, delayed and dissociated “maturation” of the placenta. [5,8] Premature or early “maturation” of the placenta is characterized by shortening and decreased thickness of the stem villi and a large number of terminal villi, which are sometimes located so closely to each other that they impede blood circulation in the intervillous space. The essence of the delayed “maturation” of the placenta is that immature villi do not mature, but turn into stromal villi. The villous tree and the entire placenta become larger, with terminal villi sparsely distributed. In the dissociated variant, all types of villi maturation are observed [3, 5, 6]. Thus, the condition of the fetus and the outcome of pregnancy with STIs depends both on the degree of structural changes in the placenta (impaired maturation, involutionary dystrophic and inflammatory changes), and on the intensity of the development of compensatory devices in it, the development of syncytial nodules and syncytiocapillary nodules and synitiocapillary membranes, as well as the volume of circulating blood in the intervillous space [1, 2].

Recurrence of infection in the second and early third trimester of pregnancy contributes to the intensification of fibroplasia and foci of necrotic changes. reduction of the vascular bed,

intensification of inflammation processes in it.

Purpose of the study: to develop a concept of the pathogenesis of premature birth based on morpho-functional studies

Material and methods. In order to study the characteristics of the clinical course and outcome of Since then, we have analyzed 120 birth histories, of which 90 were premature and 30 were timely. Depending on the duration of pregnancy, all women with premature birth were divided into 3 groups: the first group consisted of 30 (25%) women whose pregnancy was terminated between 22 and 27 weeks; the second group consisted of 30 (25%) women with abortion in the period from 28 to 32 weeks and the third group included 30 (25%) women with pregnancy loss in the period of 33-37 weeks. The control group consisted of 30 women whose pregnancy proceeded without the threat of termination, and they were admitted for childbirth at a gestational age of 38-40 weeks.

Results. The study of anamnesis from birth histories revealed a high frequency of previous diseases in women with preterm birth. From the data obtained it is clear that the frequency of detection of extragenital pathology among women with premature and timely births is not the same. In women with preterm birth, extragenital pathology was observed much more often than in the control group. It should be noted that chronic tonsillitis was more common in women with premature births than in women with timely births. 10.6% of women in group 1, 15.0% of women in group 2 and 9.5% of women in group 3 suffered from chronic tonsillitis, while in women with timely births this figure was significantly lower (4.6%). The most common pathology was pyelonephritis, which was observed 2-3 times more often than in women who gave birth on time. Irregular menstrual cycles, algomenorrhea, oligomenorrhea were observed significantly more often in women with premature birth, especially in women in group 1, compared to women in the control group.

Timely onset of menarche (11-14 years) was noted in 96.9% of women with timely births, and also in 37.1%, 34.4% and 35.7% of women with premature births, respectively.

Late onset of menarche (15-16 years) was detected in 3.1% of the control group and, accordingly, in the groups 64.6%, 65.6% and 64.3% of women with preterm birth.

Almost every fifth woman with a history of premature birth had an irregular cycle, while in the control group this figure was 5.4%. Significantly more often than in the group of women with timely births, algodismenorrhea occurred in women with premature births (21.4%, 13.1% and 15.2%, respectively) in the group of women whose pregnancy was terminated between 22 and 27, from 28 to 33 weeks and 34-37 weeks, while in the control group this pathology was observed in 4.6% of women. A high percentage of polymenorrhea was revealed in women with premature birth compared to the control group: 18.6%, 17.5% and 16.7% versus 2.3%, respectively.

Consequently, as the menstrual function of the female body is disrupted, the risk of premature birth arises.

Based on the above clinical and statistical analysis, we can distinguish three main groups of factors that influenced the incidence of preterm birth:

• the first group - socio-economic and demographic factors (nature of work, level of medical care, marital status of the mother);

• second group - socio-biological factors (age of pregnant women, outcome of previous pregnancies, multiple births);

• third group - clinical factors. They can be systematized as follows:

- infectious diseases of the mother (viral infection, toxoplasmosis, chlamydia);

- complications of pregnancy (hypertensive conditions during pregnancy, abnormal position of the fetus);

- pathological changes in the female genital organs (chronic inflammation of the uterine appendages, endometritis, uterine fibroids, uterine malformation, colpitis);

- extragenital diseases of the mother (chronic tonsillitis, pyelonephritis, rheumatism).

Conclusion: The problem of miscarriage continues to be one of the priority areas in obstetrics. The frequency of premature births is 5-12‰ of the total number of births. Their increase is associated with indicators such as perinatal and early neonatal morbidity and mortality. Premature babies account for up to 50% of stillbirths, 60-70% of early neonatal and 65-75% of infant mortality. Premature babies are born stillborn 8-13 times more often than full-term babies, and die in the first week of life 20-30 times more often. Perinatal mortality for preterm birth is 33 times higher than for full-term birth.

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The lack of common views on immunological relationships in the mother-placenta-fetus system prevents effective pathogenetic therapy for preterm pregnancy. Currently, there are practically no studies devoted to the study of functional changes in the FPS with various types of immunocorrection for the purpose of preventing preterm birth. Conclusions: When UGI persists in pregnant women, complications arise that can be considered as a positive feedback mechanism that increases the inflammatory response in the uterus, which leads to premature birth and thereby protects the maternal body from infection by getting rid of an infected pregnancy. The identified deviations in the uterus and the structure of the placenta indicate an unfavorable effect of ultrasonography on almost all parts of the placenta, causing changes in them, which subsequently, in the event of a breakdown in adaptive-compensatory reserves, lead to functional fetoplacental insufficiency and depend on the period gestation.

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