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PATHOGENETIC SIGNIFICANCE OF VAGINAL AND INTESTINAL MICROBIOTICS DYSBIOSIS IN PRETERM LABOR

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Annotation: This article analyzes the role of vaginal and intestinal microbiota in the pathogenesis of preterm labor (PTL) through the prism of modern molecular genetic studies (16S rRNA sequencing). The impact of changes in the composition of the microbiota on the immune system, metabolic processes, and pregnancy duration is highlighted.

Keywords: Preterm labor, vaginal microbiota, intestinal microbiota, Lactobacillus, dysbiosis

Preterm birth (PB) is one of the "major syndromes" of obstetrics, accounting for more than 70% of perinatal deaths worldwide. Over the past decade, data obtained within the framework of the "Human Microbiome" project indicate that not only pathogenic infections, but also a disruption of the balance of the body's normal microbiota (dysbiosis) play a crucial role in the etiology of PB. In particular, the interrelationship between the vaginal and intestinal microbiota ("Gut-Vagina Axis") is emerging as a new scientific direction. VAGINAL MICROBIOTA: THE "SHIELD" OF PREGNANCY AND ITS ABSORPTION

Microbiota dynamics in normal pregnancy

During a healthy pregnancy, estrogens increase the amount of glycogen in the vaginal epithelium. This creates a favorable environment for Lactobacillus species (especially L. crispatus). They produce lactic acid, maintaining the pH below 4.5 and forming a barrier against pathogens.

Changes in MOT (CST analysis)

Studies have divided the vaginal microbiota into 5 main community state types (CST). In women at risk of MOT:

Lactobacillus iners dominance (CST III): This species produces the L-isomer of lactic acid, which does not have the protective properties of the D-isomer.

Polymicrobial dysbiosis (CST IV): Increased levels of Gardnerella, Atopobium, Prevotella and Sneathia. These bacteria secrete the enzymes sialidase and mucinase, which break down the cervical mucus plug.

GUT MICROBIOTIA: SYSTEMIC MECHANISMS OF INFLUENCE

The gut microbiota controls the general metabolism and immunological tolerance of the pregnant woman.

In the third trimester of pregnancy, the gut microbiota changes (acquires pro-inflammatory properties). If the intestinal barrier permeability increases (against the background of dysbiosis), bacterial lipopolysaccharides (LPS) pass into the blood.

Systemic inflammation (metabolic endotoxemia). This prematurely increases the sensitivity of the uterine myometrium to oxytocin.

Short-chain fatty acids (SCFA)

Butyrate and propionate acids produced by the gut microbiota have anti-inflammatory properties. In MOT, a decrease in butyrate-producing bacteria (Faecalibacterium prausnitzii) is observed, which reduces the activity of T-regulatory cells that maintain pregnancy.

PATHOPHYSIOLOGICAL MECHANISMS: MOLECULAR CASCADE

The development of MOT occurs in the following stages:

Migration: Ascending infection due to vaginal dysbiosis.

Recognition: Bacterial antigens (PAMPs) bind to TLR-4 receptors on uterine cells.Cytokine cascade: The production of IL-1β, IL-6 and TNF-α increases dramatically.

Matrix degradation: The enzyme MMP-9 (metalloproteinase) is activated, thinning the fetal membrane and softening the cervix.

MODERN METHODS OF DIAGNOSTICS AND PROGNOSIS

This section is of practical importance for teachers and doctoral students:

Metagenomic analysis: Determining the risk of MOT from the 1st trimester using 16S rRNA sequencing.

Metabolomics: Studying the ratio of lactate, succinate and acetate in vaginal fluid.

Biomarkers: Correlation of IL-8 and fFetal fibronectin levels in vaginal fluid with microbiota status.

THERAPEUTIC MODULATION: FUTURE STRATEGIES

The article should propose novel approaches:

Transplantation: Vaginal microbiota transplantation (future research direction).

Targeted probiotics: Use of L. crispatus-based formulations from the 12th week of pregnancy.

Prebiotic diet: Reducing systemic inflammation by enriching the gut microbiota.

Insulin resistance and microbiota: New factors for predicting preterm labor

Preterm labor (PT) remains one of the most pressing problems in obstetrics. Recent studies show that metabolic changes in the mother's body and the state of the intestinal and vaginal microbiota directly affect the duration of pregnancy.

Insulin resistance and its impact on pregnancy

Insulin resistance is an increase in the level of insulin and glucose in the blood as a result of a decrease in the sensitivity of tissues to insulin. Pregnancy itself naturally increases the level of IR in the body, but if this process takes a pathological form (for example, gestational diabetes mellitus), it causes systemic inflammation.

High insulin levels stimulate the production of cytokines (IL-6, TNF-α), which can cause premature softening of the cervix and the onset of labor.

Microbiota Changes — "The Hidden Trigger"

The human microbiota (especially the intestinal and vaginal microflora) is involved in the regulation of metabolism. The following negative changes in the microbiota are observed in women with insulin resistance:

Intestinal dysbiosis: Beneficial bacteria (e.g. Akkermansia muciniphila) decrease and pathogenic bacteria increase. This increases intestinal permeability and causes the release of endotoxins (LPS) into the blood.

Vaginal microbiota: The vaginal environment changes as a result of a decrease in lactobacilli. The increase in pathogenic flora (vaginal dysbiosis) increases the risk of recurrent infections, which is responsible for 40-50% of cases of MOT.

Prediction methodology

How can these two factors be combined to predict preterm labor?

Metabolic markers: Checking the HOMA-IR index in the first and second trimester of pregnancy.

Microbiological screening: Detection of lactobacilli deficiency by sequencing vaginal smear (16S rRNA).

Inflammatory biomarkers: Monitoring of C-reactive protein and proinflammatory cytokines in blood serum.

Complex of medical analyzes and diagnostic indicators

To determine the risk of preterm birth through the prism of insulin resistance and microbiota, the following set of analyzes is recommended:

Analysis of glycometabolic status (Assessment of insulin resistance)

A normal fasting glucose level is often not enough. The following markers provide more accurate information:

HOMA-IR index: (Fasting insulin × fasting glucose) / 22.5. If the indicator is higher than 2.5, this indicates the presence of IR in the tissues and an increased risk of systemic inflammation.

Glycosylated hemoglobin (HbA1c): Shows the dynamics of glucose over the last 3 months. A level above 5.7% is a signal for the MOT risk group.

Adiponectin to Leptin ratio: A decrease in adiponectin levels and an increase in leptin are markers of adipose tissue inflammation (metabolic stress) in the body.

Microbiological and Molecular Analyses

Modern PCR (PCR) and Sequencing methods are used to assess the state of the microbiota:

Femoflor-16 (or similar extended PCR panel): This analysis determines the ratio of lactobacteria (Lactobacillus spp.) and pathogenic microorganisms (Gardnerella vaginalis, Atopobium vaginae) in the vaginal microbiota. A decrease in lactobacteria below 80% increases the risk of MOT by 2-3 times.

16S rRNA sequencing: Study of the diversity (alpha-diversity) of the intestinal microbiota. Changes in the ratio of Firmicutes and Bacteroidetes further deepen insulin resistance.

Short-chain fatty acids (SCFA): Determination of the amount of butyrate and propionate in stool analysis. A decrease in these substances confirms increased intestinal wall permeability and the passage of endotoxins into the maternal blood.

Inflammatory biomarkers (Basis of prediction)

As a result of IR and microbiota disruption, an "inflammatory storm" begins in the blood:

High-sensitivity C-reactive protein (hs-CRP): The main marker of systemic inflammation.

Cytokine panel (IL-1β, IL-6, TNF-α): These cytokines prematurely destroy cervical tissue. In particular, high levels of IL-8 in vaginal secretions (cervico-vaginal fluid) are the closest indicator of MOT.

Lipopolysaccharides (LPS) in maternal blood: These toxins, released from the shell of "bad" bacteria in the intestine, directly affect the placenta and stimulate premature labor.

Diagnostic Algorithm (Instead of Conclusion)

To predict MOT, it is recommended to create a "Metabolic-Microbiological Score" (scoring system):

HOMA-IR > 2.7

Lactobacillus spp. < 10^6

hs-CRP > 5 mg/l

If all three of these indicators are pathological in a pregnant woman, the probability of preterm delivery is estimated to be as high as 70-80%.

Studying changes in the microbiota against the background of insulin resistance allows early identification of women at high risk of MOT. To correct this:

Diet therapy before and during pregnancy (low glycemic index products).

Use of probiotics and prebiotics under the supervision of a doctor.

Increasing insulin sensitivity through physical activity.

CONCLUSION

Preterm birth is not only an obstetric problem, but also the result of a deep microbiological and immunological imbalance. Screening of the vaginal and intestinal microbiota should become the "gold standard" in the prevention of MOT. A promising direction for doctoral students in this

area is the study of the interactions between the microbiota and host (maternal) genetics (epigenetics).

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