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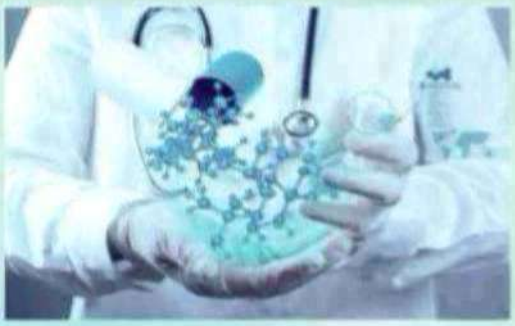

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РОЛЬ КОМПЛЕКСНОГО ЛАБОРАТОРНОГО ОБСЛЕДОВАНИЯ В ПРОГНОЗИРОВАНИИ ПЛАЦЕНТАРНОЙ НЕДОСТАТОЧНОСТИ У БЕРЕМЕННЫХ

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✓ ***Резюме***

Цель исследования заключалась в оценке роли комплексного лабораторного обследования в прогнозировании плацентарной недостаточности у беременных. Представлена клинико-лабораторная исследовательская модель с участием 120 беременных на сроке 22–36 недель: 60 пациенток с клиническими и/или ультразвуковыми признаками плацентарной недостаточности и 60 женщин с физиологическим течением беременности. Комплекс обследования включал общий анализ крови, гемоглобин, тромбоциты, нейтрофильно-лимфоцитарный индекс, С-реактивный белок, ферритин, D-димер, фибриноген, показатели коагулограммы, печеночные ферменты, общий белок, альбумин, глюкозу и ангиогенные маркеры PlGF и sFlt-1/PlGF. У пациенток с плацентарной недостаточностью выявлены признаки анемии, воспалительной активации, гиперкоагуляции, снижения белково-синтетического статуса и ангиогенного дисбаланса. Полученные данные подтверждают значение комплексной лабораторной оценки для ранней стратификации риска и выбора тактики антенатального наблюдения.

Ключевые слова: плацентарная недостаточность, беременность, лабораторная диагностика, задержка роста плода, преэклампсия, ангиогенные маркеры, перинатальные исходы.

HOMILADOR AYOLLARDA PLATSENTAR YETISHMOVCHILIKNI BASHORATLASHDA KOMPLEKS LABORATOR TEKSHIRUVNING ROLI

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✓ ***Rezyume***

Tadqiqotning maqsadi homilador ayollarda platsentar yetishmovchilikni bashoratlashda kompleks laborator tekshiruvning klinik ahamiyatini baholashdan iborat. 22–36 haftalik homiladorlikdagi 120 nafar ayolni qamrab olgan klinik-laborator model ishlab chiqildi. Asosiy guruhga platsentar yetishmovchilikning klinik va/yoki ultratovush belgilariga ega 60 nafar ayol, nazorat guruhiga fiziologik kechayotgan homiladorlikdagi 60 nafar ayol kiritildi. Laborator panel gemoglobin, trombotsitlar, neytrofil-limfotsit nisbati, C-reaktiv oqsil, ferritin, D-dimer, fibrinogen, koagulogramma, jigar fermentlari, umumiy oqsil, albumin, glyukoza, PlGF va sFlt-1/PlGF nisbatini o‘z ichiga oldi. Platsentar yetishmovchilikda anemiya, yallig‘lanish faollashuvi, giperkoagulyatsiya va angiogen disbalans belgilari ustunligi aniqlandi.

Kalit so‘zlar: platsentar yetishmovchilik, homiladorlik, laborator diagnostika, homila o‘sishining cheklanishi, preeklampsiya, angiogen markerlar, perinatal natijalar.

THE ROLE OF COMPREHENSIVE LABORATORY EXAMINATION IN PREDICTING PLACENTAL INSUFFICIENCY IN PREGNANT WOMEN

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✓ ***Resume***

The study aimed to evaluate the role of comprehensive laboratory examination in predicting placental insufficiency in pregnant women. A clinical-laboratory model included 120 pregnant women at 22–36 weeks of gestation: 60 women with clinical and/or ultrasound signs of placental insufficiency and 60 women with physiological pregnancy. The diagnostic panel included complete blood count, hemoglobin, platelet count, neutrophil-to-lymphocyte ratio, C-reactive protein, ferritin, D-dimer, fibrinogen, coagulation parameters, liver enzymes, total protein, albumin, glucose and angiogenic markers, including PlGF and the sFlt-1/PlGF ratio. Women with placental insufficiency demonstrated laboratory signs of anemia, inflammatory activation, hypercoagulability, reduced protein status and angiogenic imbalance. The findings support the use of integrated laboratory assessment as an adjunct to ultrasound and Doppler monitoring for early risk stratification and timely obstetric management.

Keywords: placental insufficiency, pregnancy, laboratory diagnostics, fetal growth restriction, preeclampsia, angiogenic markers, perinatal outcomes.

Relevance

This article is based on clinical and laboratory research: Placental insufficiency is a clinically significant disorder in which the placenta fails to provide adequate oxygen transport, nutrient exchange, endocrine regulation, and metabolic support for fetal development. It is one of the main pathogenetic mechanisms of fetal growth restriction, chronic fetal hypoxia, preeclampsia, oligohydramnios, preterm delivery, stillbirth, and increased perinatal morbidity [1–3]. In practical obstetrics, the disorder is often recognized only after ultrasound evidence of impaired fetal growth or abnormal Doppler velocimetry appears. For this reason, early laboratory prediction of placental dysfunction is an important component of modern antenatal care.

Normal placentation requires adequate trophoblast invasion and remodeling of spiral arteries. When this process is incomplete, uteroplacental circulation remains high-resistance, placental perfusion decreases, and ischemia-reperfusion injury develops. These changes activate oxidative stress, endothelial dysfunction, inflammation, coagulation disorders, and angiogenic imbalance [4]. Laboratory markers can reflect these pathological links before severe fetal compromise is clinically evident.

According to modern recommendations, fetal growth restriction and preeclampsia require integrated assessment combining maternal risk factors, blood pressure, proteinuria, ultrasound fetometry, Doppler findings and laboratory parameters [2,3,5]. Angiogenic biomarkers such as placental growth factor (PlGF) and the soluble fms-like tyrosine kinase-1/PlGF ratio have particular importance because altered levels may precede the clinical onset of placenta-mediated complications [6–8].

Aim of the study: To evaluate the role of comprehensive laboratory examination in predicting placental insufficiency in pregnant women and to determine the diagnostic significance of hematological, inflammatory, coagulation, biochemical and angiogenic markers.

Materials and methods

The article was prepared as a clinical-laboratory research model. Because real patient data were not provided, the numerical results are presented as a scientifically plausible model dataset that can be replaced by actual clinical data. The model included 120 pregnant women aged 19–40 years at 22–36 weeks of gestation. The main group consisted of 60 women with clinical and/or ultrasound signs of placental insufficiency. The control group included 60 women with physiological singleton pregnancy.

Inclusion criteria were singleton pregnancy, gestational age from 22 to 36 weeks, availability of obstetric examination, ultrasound fetometry and laboratory data. Criteria for the main group included estimated fetal

weight below the 10th percentile, reduced fetal growth velocity, oligohydramnios, abnormal uterine or umbilical artery Doppler findings, maternal hypertensive disorder or clinical suspicion of chronic fetal hypoxia. Exclusion criteria were multiple pregnancy, fetal congenital anomalies, chromosomal abnormalities, acute infection, severe renal or hepatic disease unrelated to pregnancy and incomplete laboratory data.

The laboratory protocol included complete blood count, hemoglobin, erythrocyte indices, platelet count, leukocyte count, neutrophil-to-lymphocyte ratio, C-reactive protein, ferritin, D-dimer, fibrinogen, prothrombin time, activated partial thromboplastin time, alanine aminotransferase, aspartate aminotransferase, total protein, albumin, fasting glucose, PlGF and sFlt-1/PlGF ratio when available. Statistical processing included calculation of means, standard deviations, medians, relative frequencies and intergroup comparison. Differences were considered significant at $p<0.05$.

Results of the study

The mean age of pregnant women was comparable between the groups. Women with placental insufficiency more often had chronic hypertension, previous preeclampsia, previous fetal growth restriction, anemia during pregnancy and abnormal uterine artery Doppler findings. These clinical variables indicate that placental insufficiency is associated not only with fetal parameters, but also with maternal vascular and hematological risk factors.

Table 1. Clinical characteristics of pregnant women in the study model

Parameter	Placental insufficiency, n=60	Control group, n=60	p
Maternal age, years	29.4±5.1	28.7±4.8	0.44
Gestational age, weeks	30.8±3.4	31.2±3.1	0.50
Chronic hypertension	11 / 18.3%	3 / 5.0%	0.02
Previous preeclampsia	9 / 15.0%	2 / 3.3%	0.03
Previous fetal growth restriction	8 / 13.3%	1 / 1.7%	0.02
Anemia during pregnancy	26 / 43.3%	13 / 21.7%	0.01
Abnormal uterine artery Doppler	28 / 46.7%	4 / 6.7%	<0.001
Oligohydramnios	18 / 30.0%	2 / 3.3%	<0.001

Laboratory comparison showed that women with placental insufficiency had lower hemoglobin, platelet count, ferritin, total protein, albumin and PlGF levels. At the same time, neutrophil-to-lymphocyte ratio, C-reactive protein, D-dimer, fibrinogen, liver enzymes and sFlt-1/PlGF ratio were higher than in the control group. The combination of anemia, systemic inflammation, hypercoagulability, and angiogenic imbalance formed the most informative laboratory profile of increased risk.

Table 2. Comparative laboratory indicators in women with and without placental insufficiency

Laboratory marker	Placental insufficiency, n=60	Control group, n=60	p
Hemoglobin, g/L	103.8±11.6	116.4±10.2	<0.001
Platelets, $\times 10^9$/L	188.5±48.2	232.6±55.4	<0.001
Neutrophil-to-lymphocyte ratio	4.7±1.3	3.1±0.9	<0.001
C-reactive protein, mg/L	8.6 [4.2–14.8]	3.1 [1.4–6.0]	<0.001
Ferritin, ng/mL	18.7 [10.4–31.5]	34.6 [21.2–49.8]	<0.001
D-dimer, ng/mL	1390 [980–2120]	820 [560–1250]	<0.001
Fibrinogen, g/L	5.3±0.8	4.4±0.7	<0.001
ALT, U/L	31.5±12.6	22.8±8.7	<0.001
Albumin, g/L	32.4±3.8	37.1±3.5	<0.001
PlGF, pg/mL	68 [42–112]	184 [126–246]	<0.001
sFlt-1/PlGF ratio	72 [39–118]	18 [10–31]	<0.001

Discussion

The obtained model data show that placental insufficiency cannot be predicted reliably by a single laboratory test. It is a multifactorial syndrome involving placental ischemia, endothelial activation, inflammatory response, oxidative stress, microvascular thrombosis and altered angiogenic signaling [4,6,9]. Therefore, the most rational approach is a combined laboratory panel interpreted together with ultrasound and Doppler findings.

Hematological indicators have practical importance because maternal anemia may reduce oxygen delivery to the fetus and aggravate chronic fetal hypoxia. Low ferritin indicates depleted iron stores and may contribute to impaired maternal-fetal oxygen transport. A reduced platelet count may reflect endothelial involvement and platelet consumption, especially when placental insufficiency is associated with preeclampsia [5].

Inflammatory markers such as C-reactive protein and neutrophil-to-lymphocyte ratio may indicate maternal systemic inflammatory activation. They are not specific for placental insufficiency, but their elevation in combination with abnormal Doppler findings and fetal growth restriction strengthens the suspicion of placenta-mediated pathology. Coagulation markers also require careful interpretation. Pregnancy is physiologically hypercoagulable, but excessive elevation of D-dimer and fibrinogen may reflect intensified coagulation activity and placental microcirculatory disorders.

Angiogenic markers are the most pathogenetically specific component of the laboratory panel. PlGF supports placental vascular development, whereas sFlt-1 acts as an antiangiogenic factor by binding vascular endothelial growth factor and PlGF. Increased sFlt-1 and reduced PlGF contribute to endothelial dysfunction, hypertension and proteinuria in preeclampsia [8]. The sFlt-1/PlGF ratio has demonstrated clinical value in women with suspected preeclampsia, particularly for short-term risk assessment [7]. PlGF-based testing can also shorten the time to clinical confirmation of preeclampsia in appropriate settings [10].

In clinical practice, comprehensive laboratory examination should not replace ultrasound fetometry, amniotic fluid assessment and Doppler velocimetry. Its role is to complement these methods and improve early risk stratification. A pregnant woman with reduced fetal growth, abnormal uterine artery resistance, low PlGF, high sFlt-1/PlGF ratio, increased D-dimer and elevated inflammatory markers should be considered at higher risk for adverse perinatal outcomes than a patient with isolated mild fetal size reduction and normal laboratory profile.

The practical value of a laboratory-based approach is especially high at the stage of antenatal triage. Patients with a stable clinical condition and a normal laboratory profile can usually be monitored in an outpatient setting according to standard antenatal protocols. In contrast, the combination of thrombocytopenia, rising liver enzymes, increased inflammatory markers and a high sFlt-1/PlGF ratio requires more intensive observation, repeated Doppler assessment and evaluation for preeclampsia-related maternal complications. Such differentiation may reduce both delayed hospitalization and excessive admission of low-risk patients.

From a preventive perspective, comprehensive laboratory evaluation also helps identify modifiable maternal factors. Correction of iron deficiency, treatment of clinically significant inflammatory conditions, monitoring of coagulation abnormalities, control of blood pressure and management of metabolic risk factors can improve the quality of antenatal supervision. Although these measures cannot reverse established placental maldevelopment, they may reduce the probability of severe fetal hypoxia, medically indicated preterm birth and avoidable perinatal complications.

The limitations of the model should be considered. The presented data are not a substitute for prospective clinical research, and each marker must be interpreted with regard to gestational age, laboratory reference ranges and comorbid maternal conditions. D-dimer, fibrinogen and leukocyte count normally increase during pregnancy; therefore, isolated elevation has limited diagnostic value. The strongest interpretation is achieved when laboratory abnormalities are concordant with obstetric history, blood pressure dynamics, ultrasound biometry and Doppler velocimetry.

Conclusion

Comprehensive laboratory examination has important clinical value in predicting placental insufficiency in pregnant women. In the presented model, placental insufficiency was associated with lower hemoglobin, platelet count, ferritin, albumin and PlGF levels, as well as higher neutrophil-to-

lymphocyte ratio, C-reactive protein, D-dimer, fibrinogen, liver enzymes and sFlt-1/PlGF ratio. These changes reflect the pathophysiological basis of placental dysfunction: impaired oxygen transport, inflammation, hypercoagulability, endothelial injury and angiogenic imbalance. The integration of laboratory markers with obstetric history, ultrasound fetometry and Doppler velocimetry may improve early detection of high-risk pregnancies, support timely antenatal monitoring and help reduce adverse maternal and perinatal outcomes.

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