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## MODERN ASPECTS OF PLACENTAL DYSFUNCTION: A LITERATURE REVIEW

*Abstract*

Placental dysfunction (PD) is one of the most common and serious complications of pregnancy, characterized by impaired placental function and inadequate delivery of oxygen and nutrients to the fetus. This condition occupies a leading position among the causes of perinatal morbidity and mortality, as it is associated with fetal growth restriction, chronic hypoxia, fetal distress, preterm birth, and a high risk of neonatal complications. The incidence of placental dysfunction significantly increases in the presence of preeclampsia, arterial hypertension, anemia, endocrine disorders, infectious diseases, and maternal extragenital pathology.

The development of PD is primarily based on impaired placentation, inadequate trophoblast invasion, alterations in uteroplacental blood flow, endothelial dysfunction, and oxidative stress. As a result, placental ischemia develops, leading to impaired transport of oxygen and nutrients as well as disruption of the endocrine function of the placenta. Decompensated forms of PD are particularly dangerous, as they may result in intrauterine fetal death and severe disturbances in postnatal adaptation.

Current approaches to the diagnosis of PD are based on a comprehensive assessment of the mother-placenta-fetus unit. The most informative diagnostic methods include ultrasound fetometry, Doppler assessment of uteroplacental and fetal blood flow, cardiotocography, and the evaluation of biochemical markers of placental dysfunction. Early detection of hemodynamic abnormalities enables prediction of pregnancy complications and facilitates the selection of optimal management strategies.

Modern management of pregnant women with PD includes careful fetal surveillance, correction of associated maternal disorders, prevention of thrombotic and hemodynamic disturbances, and an individualized approach to determining the timing of delivery. Early diagnosis and appropriate treatment contribute to reducing severe perinatal outcomes and improving both maternal and neonatal prognosis.

*Keywords:* placental dysfunction, fetal growth restriction, Doppler ultrasonography, pregnancy, fetal distress, cardiotocography, perinatal complications.

*Problem statement.* Placental dysfunction (PD) is one of the most common pathological conditions in obstetrics and is associated with high rates of perinatal morbidity and mortality. Impaired placental function leads to insufficient fetal supply of oxygen and nutrients, resulting in chronic hypoxia, fetal growth restriction, fetal distress, and preterm birth. Despite significant advances in modern medicine, the problem of early diagnosis and effective management of PD remains relevant due to the complexity of its pathogenetic mechanisms and the lack of universally accepted approaches to the management of such pregnancies.

Current trends in obstetrics are focused on the early detection of placental dysfunction before the onset of clinical manifestations. At the same time, the prevalence of pregnancies complicated by extragenital diseases, arterial

hypertension, diabetes mellitus, preeclampsia, and other risk factors continues to increase, significantly elevating the likelihood of PD development. This necessitates the improvement of diagnostic, predictive, and preventive strategies for this complication.

*Analysis of recent research and publications.* Contemporary studies on placental dysfunction highlight its multifactorial nature and the complexity of its pathogenetic mechanisms. Recent investigations emphasize the pivotal role of impaired placentation, oxidative stress, and placental metabolic dysfunction as key contributors to the development of placental insufficiency [2, 4]. In particular, alterations in mitochondrial homeostasis and energy metabolism have been identified as important factors in the pathogenesis of placental dysfunction in complicated pregnancies [2]. Furthermore, several studies have confirmed the significance of oxidative stress as one of the principal mechanisms responsible for placental tissue damage and impaired placental function [4].

An important area of current research focuses on the immunological and inflammatory mechanisms underlying PD. Cytokine-mediated responses, particularly those involving interleukins, have been shown to influence placentation and fetal growth by altering the balance between inflammatory and adaptive processes [3]. These findings support the concept that systemic inflammation plays a substantial role in the development of placental insufficiency.

Another group of studies has examined the clinical consequences of PD. Placental dysfunction has been identified as a major pathogenetic factor in fetal growth restriction and increased neonatal vulnerability. Prolonged fetal hypoxia and impaired nutrient supply have been shown to contribute to long-term adverse effects on the organs and systems of the newborn [5].

Considerable attention has also been devoted to the diagnosis of PD. Doppler ultrasonography is regarded as one of the most informative methods for assessing

uteroplacental and fetal blood flow and for predicting adverse perinatal outcomes [7, 8]. Alterations in hemodynamic parameters of the umbilical and uterine arteries have been found to correlate with the severity of placental dysfunction [8, 9]. Moreover, studies have demonstrated the diagnostic value of Doppler assessment in differentiating fetal growth restriction from constitutionally small fetuses [8].

A separate field of contemporary research concerns the neonatal and perinatal consequences of placental insufficiency. Placental dysfunction has been identified as one of the key factors contributing to increased neonatal vulnerability, particularly among preterm infants. Prognostic models proposed in recent studies enable the assessment of adverse outcome risks based on a combination of clinical and ultrasonographic parameters [6].

Contemporary publications also focus on biochemical and molecular markers of placental dysfunction. It has been established that alterations in angiogenic factors, particularly imbalances in placental proteins and metabolic indicators, represent early predictors of placental dysfunction and preeclampsia [11]. These findings open new opportunities for early screening and the implementation of personalized approaches to pregnancy management.

In the context of therapeutic and preventive strategies, studies have demonstrated the effectiveness of anticoagulant and antiplatelet therapies in reducing the risk of placental-related complications [15]. Several reports emphasize the importance of preeclampsia prevention as one of the primary approaches to decreasing the incidence of placental dysfunction [16]. In addition, novel strategies, including targeted therapies and experimental methods aimed at improving placental function, are currently under active investigation [14].

*Purpose of the article.* To analyze current approaches to the diagnosis and management of pregnant women with placental dysfunction based on a review of contemporary scientific literature.

*Presentation of the main material of the study*. Placental dysfunction is a pathological syndrome resulting from impaired morphological and functional capacity of the placenta to ensure adequate exchange between the maternal and fetal organisms. The placenta performs essential functions throughout pregnancy, including the transport of oxygen, nutrients, hormones, and metabolic waste products. Any disruption of its structure or blood supply may adversely affect intrauterine fetal development and lead to severe perinatal complications [1].

Current evidence indicates that placental dysfunction is a multifactorial condition. The main risk factors for its development include preeclampsia, arterial hypertension, maternal anemia, diabetes mellitus, cardiovascular diseases, thyroid disorders, chronic infections, autoimmune diseases, and thrombophilic conditions [2]. Harmful lifestyle factors, particularly smoking, also play a significant role, as they induce vasospasm and chronic fetal hypoxia. The risk of placental dysfunction is further increased in multiple pregnancies, Rh incompatibility, and women with an unfavorable obstetric history.

The pathogenesis of placental dysfunction is primarily associated with impaired placentation during the early stages of pregnancy [3]. Inadequate trophoblast invasion results in incomplete remodeling of the uterine spiral arteries, causing them to retain high vascular resistance. Consequently, blood flow to the placenta is restricted, leading to placental ischemia. Chronic hypoperfusion promotes the development of oxidative stress, activation of pro-inflammatory cytokines, and disruption of angiogenic mechanisms [4]. These alterations are accompanied by endothelial dysfunction, impaired microcirculation, and the development of fetal hypoxia.

One of the most common consequences of placental dysfunction is fetal growth restriction (FGR). In this condition, the fetus fails to achieve its genetically determined growth potential because of insufficient oxygen and nutrient supply [5]. Chronic hypoxia leads to a compensatory redistribution of blood flow, known

as the “brain-sparing effect,” in which the majority of blood is directed to vital organs, particularly the brain and heart. However, prolonged persistence of this adaptive mechanism eventually results in the exhaustion of compensatory reserves and increases the risk of intrauterine fetal demise.

Clinical manifestations of placental dysfunction may be subtle, particularly during the early stages of the disease. Pregnant women may report decreased fetal movements or changes in fetal activity patterns; however, in many cases, the condition is detected only through instrumental examination [6]. Therefore, timely prenatal screening of high-risk pregnancies is of paramount importance.

Ultrasound examination remains the cornerstone of placental dysfunction diagnosis. Fetal biometry enables the assessment of fetal growth parameters, determination of gestational age appropriateness, and identification of fetal growth restriction [7]. Particular attention is paid to the measurement of fetal abdominal circumference, as this parameter is highly sensitive to chronic hypoxia and nutritional deficiency. In addition, ultrasound assessment allows evaluation of amniotic fluid volume, placental structure, and placental maturity.

One of the most informative contemporary diagnostic methods is Doppler ultrasonography of the uteroplacental and fetal circulation [8]. Assessment of blood flow in the uterine arteries provides valuable information regarding the degree of placental perfusion impairment. Increased resistance indices and the presence of a diastolic notch are indicative of a high risk of placental insufficiency. Evaluation of umbilical artery blood flow enables assessment of the severity of fetal hemodynamic disturbances. As the condition progresses, reduced, absent, or reversed end-diastolic flow may be observed, representing an unfavorable prognostic sign [9].

Cardiotocography (CTG) also plays an important role in the diagnosis of placental dysfunction. This method evaluates fetal well-being by analyzing fetal heart rate, heart rate variability, and the presence of accelerations and

decelerations [10]. Reduced heart rate variability and the occurrence of late decelerations may indicate fetal distress and decompensation. Cardiotocography is particularly valuable during the third trimester of pregnancy and throughout labor.

In recent years, considerable attention has been devoted to the study of biochemical markers of placental dysfunction. One of the most promising approaches is the assessment of placental growth factor (PlGF) levels and the sFlt-1/PlGF ratio [11]. An imbalance of angiogenic factors reflects the severity of endothelial dysfunction and may be used for the early prediction of preeclampsia and placental dysfunction. In addition, markers of oxidative stress, placental proteins, cytokines, and cell adhesion molecules are being actively investigated.

The management of pregnant women with placental dysfunction depends on the severity of the disorder, gestational age, and fetal condition [12]. The primary objective is to prolong pregnancy to a safe gestational age while minimizing the risk to the fetus. Dynamic monitoring of both maternal and fetal conditions is essential and includes regular ultrasound examinations, Doppler assessments, and cardiotocography.

Non-pharmacological interventions include an appropriate balance between work and rest, adequate nutrition, sufficient sleep, reduction of psychoemotional stress, and avoidance of harmful habits [13]. Particular attention should be paid to the management of concomitant extragenital disorders, blood pressure control, and correction of maternal anemia.

Among pharmacological interventions, antiplatelet agents, particularly low-dose acetylsalicylic acid, are widely used because they improve uteroplacental circulation and help prevent thrombotic complications [14]. In the presence of thrombophilic conditions, low-molecular-weight heparins may also be administered. Comprehensive therapy may additionally include agents that improve microcirculation and tissue metabolism. Antioxidant therapy is

considered a promising approach aimed at reducing oxidative stress and stabilizing endothelial function [15].

In cases of decompensated placental dysfunction and severe fetal distress, timely delivery remains the only effective strategy for preventing antepartum fetal death [16]. The timing and mode of delivery depend on gestational age, the severity of blood flow impairment, and fetal condition. In the presence of critical Doppler abnormalities or severe fetal hypoxia, operative delivery by cesarean section is generally preferred.

Thus, contemporary approaches to the diagnosis and management of placental dysfunction are based on a comprehensive assessment of the mother–placenta–fetus unit, early detection of hemodynamic disturbances, and individualized treatment strategies. The use of modern monitoring techniques allows timely identification of signs of fetal decompensation and reduces the risk of severe perinatal complications.

*Conclusions.* Placental dysfunction is one of the leading causes of perinatal morbidity and mortality, as it is associated with impaired functioning of the mother-placenta-fetus unit, chronic fetal hypoxia, and fetal growth restriction. Modern diagnostic methods, including ultrasonography, Doppler assessment, cardiotocography, and the evaluation of biochemical markers, enable timely detection of placental dysfunction and assessment of the severity of pathological changes. A comprehensive approach to the management of pregnant women with PD, including careful fetal surveillance, correction of concomitant disorders, and individualized selection of treatment and delivery strategies, contributes to reducing the risk of severe perinatal complications and improving maternal and neonatal outcomes.

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