



THE ROLE OF BIOMARKERS AND CLINICAL AND ANAMNESTIC FACTORS IN PRE-ECLAMPSIA

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ABSTRACT

Preeclampsia is one of the most dangerous complications of pregnancy, being a significant cause of maternal and perinatal mortality. Modern methods of predicting this condition are based on the analysis of clinical and anamnestic data, as well as on the assessment of specific biomarkers. Further studies of pathogenesis and the development of optimal prognostic algorithms will help to improve the accuracy of identifying high-risk groups and make screening more effective and accessible. This paper analyzes the scientific literature on the problem of early diagnosis of preeclampsia. The key risk factors influencing the development of pathology, and modern hypotheses about the mechanisms of its occurrence are considered. Particular attention is paid to the role of biochemical markers in predicting the disease, as well as the most promising predictors and their combinations that can improve the quality of early detection of high-risk patients.

Introduction. According to the 2021 clinical guidelines "Preeclampsia. Eclampsia. Edema, proteinuria, and hypertensive disorders during pregnancy, labor and postpartum period", pre-eclampsia is defined as a complication of pregnancy, labor and postpartum period characterized by an increase in blood pressure (CAP >140 mmHg and/or PA >90 mmHg) after the 20th week of gestation, irrespective of the initial BP level, combined with proteinuria or at least one sign of multi-organ failure. The main risk factors include a history of pre-eclampsia, chronic hypertension, gestational diabetes, antiphospholipid syndrome, obesity, age, chronic kidney disease, and the use of assisted reproductive technologies. In addition, there is a confirmed association between pregnancy by a fetus with trisomy 13 and an increased likelihood of developing this complication. Genetic predisposition to pre-eclampsia is also being actively studied.

Although the disease remains the subject of intensive research, its pathogenesis has not yet been fully elucidated. The concept of multifactorial origin of the pathology, where the state of the placenta plays a key role, is generally accepted. The main hypothesis suggests that immune disharmony between the mother and fetus occurs already in the early stages of

pregnancy. This leads to pathological changes that develop in two stages. In the first stage, the placentation process is disrupted, which is due to insufficient proliferation, migration, and invasion of cytotrophoblast cells. This entails a defect in the remodeling of spiral arteries, decreased blood flow in the placenta, the formation of hypoxia, and oxidative stress. Under conditions of chronic hypoxia, apoptosis and necrosis processes intensify in the placental tissue, which aggravates the pathological process. At the second stage of preeclampsia development, endothelial function is disrupted, accompanied by an imbalance of angiogenic and antiangiogenic factors, which leads to systemic endotheliosis. These changes cause the clinical symptoms of the disease. However, this hypothesis has recently been criticized, as more and more studies indicate that the pathogenesis of preeclampsia is not limited exclusively to trophoblastic disorders. In some cases, constitutional features of the mother's body, genetic predisposition, and obesity may be of decisive importance.

In recent decades, scientific research has been actively focused on finding biochemical markers that can predict the development of preeclampsia in early pregnancy. They are aimed at studying the pathogenetic mechanisms of the disease and identifying the correlation between the levels of various biological molecules involved in these processes. To date, the most studied predictors of preeclampsia are vascular endothelial growth factor (VEGF), placental growth factor (PlGF), and soluble FMS-like tyrosine kinase-1 (sFlt-1), the main source of which is the placenta. These factors play a key role in angiogenesis, as well as in maintaining the viability and functional activity of trophoblast cells. Studies have revealed a link between elevated endoglin levels and the development of preeclampsia, as well as its correlation with the severity of the disease. P-selectin is a cell adhesion molecule from the selectin family, produced by endothelial cells and platelets. It is involved in inflammatory processes and has procoagulant activity. Recent studies have shown that an increase in the concentration of soluble P-selectin in the serum before the 20th week of pregnancy precedes the appearance of clinical manifestations of preeclampsia. However, a combined analysis of its level together with Activin-A demonstrated low sensitivity (59%), which calls into question its prognostic value.

Pregnancy-associated plasma protein-A (PAPP-A), like ADAM12, is a metalloproteinase synthesized by trophoblast and decidua cells. This protein plays a key role in placentation, regulating the processes of trophoblast proliferation and migration, hormonal secretion, and the transport of glucose and amino acids. In clinical practice, PAPP-A is more often used for biochemical screening of trisomies, but its low level in the first trimester is associated with a high risk of preeclampsia and fetal growth retardation. Due to its affordability, this biomarker is often included in combined screening.

Modern studies are considering a wide range of potential biomarkers of preeclampsia, including adiponectin, resistin, chemerin, cystatin C, leptin, and others. Some of them show promise but require further study to confirm their clinical significance. Many experts emphasize the importance of the combined use of biochemical markers with the analysis of clinical and anamnestic risk factors.

Conclusion. Thus, at the moment, the studied biomarkers cannot be widely introduced into clinical practice due to their limited availability or insufficient predictive value in predicting pre-eclampsia. Currently, screening for this complication is based on risk

assessment based on clinical and anamnestic data and determination of placental growth factor (PlGF) levels.

Further study of the pathogenetic mechanisms of pre-eclampsia, as well as the search for optimal combinations of biomarkers and clinical predictors, will improve the effectiveness of screening programs. This, in turn, will ensure more accurate identification of high-risk groups and make the diagnosis of the disease more affordable

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