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MODERN VIEWS ON PATHOGENESIS OF THE PREECLAMPSIA IN RELATION TO ENDOTHELIAL DYSFUNCTION

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Relevance. A major source of maternal and perinatal morbidity and mortality is preeclampsia, a dangerous complication of the second half of pregnancy marked by arterial hypertension and proteinuria. Endothelial dysfunction, which arises in the setting of preeclampsia, is a crucial component in the pathophysiology of this illness. It is thought that the creation and subsequent application of efficient techniques for treating endothelial dysfunction in clinical practice will greatly enhance the progression of pregnancy and raise the likelihood of positive results for preeclamptic patients. In this regard, a more thorough comprehension of the mechanisms behind the emergence of endothelium dysfunction is essential.

Aim: to examine and organise recent research on the pathophysiology of endothelial dysfunction in pregnancy**.**

Materials and methods. Current perspectives on the pathophysiology of endothelial dysfunction in the context of preeclampsia were the topic of a thorough search, review, and analysis of published literature, both domestically and internationally.

Results and their discussions. Atypically superficial extravillous trophoblast penetration into the mother's spiral arteries is a hallmark of preeclampsia. These arteries' retained elastic and resistant qualities cause peripheral vascular resistance to rise, which in turn reduces placental blood flow. Significant levels of proinflammatory and antiangiogenic factors are generated during intraplacental ischaemia and hypoxia, which starts the development of systemic endothelial dysfunction.

Soluble Fms-like tyrosine kinase-1 (sFlt-1), which is produced in excess during hypoxia, is one of the most significant pathogenetic antiangiogenic agents. By attaching to the proper receptors on the surface of endothelial cells, this protein efficiently prevents placental growth factor (PlGF) and vascular endothelial growth factor (VEGF) from interacting, hence decreasing angiogenesis. In clinical practice, measuring the levels of placental growth factor (PlGF), soluble Fms-like tyrosine kinase-1 (sFlt-1), and their ratio (sFlt-1/PlGF) is actively utilised for early preeclampsia diagnosis and prognosis (many weeks prior to manifestation).

A rise in Th17 lymphocytes and decreased Treg cell function are also strongly correlated with the development of endothelial dysfunction in pregnancy. Additionally, under the impact of miR-155, a decrease in placental vascularization is seen when angiogenic inducer 61 (CYR61) expression is inhibited. Polymorphisms in the TNF-α gene, particularly at position -308, are more common in preeclamptic women. These SNPs are associated with increased expression of TNF-α, which destroys endothelial cells and causes inflammation. Reduced nitric oxide generation or bioavailability, overexpression of the methylenetetrahydrofolate reductase gene, which raises homocysteine levels, and elevated levels of soluble endoglin (sEng) are all linked to endothelial dysfunction. The internal organs of the mother and foetus may suffer greatly from vasoconstriction and immunological disorders brought on by the combination of these harmful causes.

Conclusions. Because of its high rates of maternal and neonatal morbidity and mortality, preeclampsia continues to be one of the most dangerous obstetric issues. The evolution of endothelial dysfunction, which leads to haemodynamic abnormalities and ultimately multiple organ failure, is closely linked to the development of this condition. Nevertheless, focused repair of endothelial dysfunction is not included in current pathogenetic therapy for preeclampsia, underscoring the critical need for a thorough investigation of its pathophysiology and the creation of suitable therapeutic strategies.