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**PATHOGENESIS OF ENDOTHELIAL DYSFUNCTION**

**IN PREGNANT WOMEN WITH PREECLAMPSIA**

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**Relevance**. Preeclampsia occupies an important place among numerous types of obstetric pathology, being one of the most serious complications of pregnancy and childbirth. The problem of preeclampsia is determined by its incidence (7-16%) and the fact that this complication of pregnancy ranks second among causes of maternal mortality, and amounts for 17 to 40% of its obstetric causes. Preeclampsia is associated with high rates of perinatal loss. Currently, most researchers consider preeclampsia and its various clinical manifestations in terms of systemic endothelial dysfunction, which is accompanied by activation of platelet and vascular homeostasis, damage and dysfunction of erythrocytes, microcirculation disorders, vasoconstriction and impairment of regional, particularly utero-placental blood flow.

**Purpose**. To identify pathogenic features of endothelial dysfunction in pregnant women with preeclampsia.

**Materials and methods.** The study was conducted at the clinical base of the Department of Obstetrics, Gynecology and Pediatric Gynecology. The study involved 200 pregnant women who were treated in the municipal healthcare facility “Kharkiv city hospital No. 1”. The main group consisted of 50 pregnant women with mild preeclampsia, 50 women with moderate and 50 women with severe preeclampsia. Control group consisted of 50 women with physiological pregnancy. All the women underwent comprehensive clinical and laboratory examination. Additional studies included determination of the level of Willebrant factor (WF), endothelin-1 (ET-1) and S-nitrosothiol.

**Results and their discussion**. In pregnant women with mild preeclampsia the content of WF increased 1.2 times, with moderate preeclampsia - 1.5 times, with severe - 1.8 times. The increase in WF is always associated with activation of coagulation and an increase in platelet factor secretion. Assessment of coagulation in pregnant with preeclampsia showed increased activity of coagulation (as compared to the control group) and the presence of thrombocytopenia. The determined damage of the endothelium (increased level of WF) triggers endothelial dysfunction, which may be a leading factor of preeclampsia. Endothelial dysfunction was studied by determination of ET-1 and S-nitrosothiols (stable metabolite of endothelium-secreting NO) levels. The study of S-nitrosothiols content showed that in preeclampsia (even in mild severity) it was significantly reduced, especially in severe degree (5 times) (p <0.01). Assessment of ET-1 content showed that its level slightly increased in mild preeclampsia, in moderate preeclampsia it was 1.5 times higher and 1.8 times higher in severe preeclampsia as compared to the control group (p <0.01).

**Conclusions**. Pregnant women with preeclampsia were found to have endothelial damage, which caused its dysfunction.