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PLACENTAL INSUFFICIENCY IN WOMEN WITH GESTATIONAL HYPERTENSION: THE ROLE OF ENDOTHELIAL DYSFUNCTION

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Resume: The development of gestational hypertension in women in the second half of pregnancy is accompanied by endothelial dysfunction with an increase in the level of von Willebrand factor, the development of hemodynamic disorders, ultrasound signs of placental insufficiency, fetal hypoxia with the development of fetal growth restriction syndrome.

Key words: placental insufficiency, endothelial dysfunction, gestational hypertension.

The pathogenesis of placental insufficiency (PI) and other complications of gestation is associated with dysfunction of the vascular endothelium. Extragenital diseases are one of the significant risk factors, in the presence of which PI develops in 30–45% of pregnant women [1, 2]. The most unfavorable prognosis for pregnancy outcome is observed with arterial hypertension [3, 4].

Complications of hypertensive disorders in pregnant women, such as placental detachment and hemorrhage, acute cerebrovascular stroke, preeclampsia and exlampsia, fetal growth retardation, as well as preterm delivery and the birth of premature newborns, are the main causes of maternal and perinatal mortality. [5, 6].

37 pregnant women aged 20 to 39 years with gestational arterial hypertension (without proteinuria) were included in our work-up. Inclusion criteria: first developed during pregnancy arterial hypertension, voluntary informed consent to participate in the study. The control group consisted of 20 healthy pregnant women. The research methods used by us were the determination of a marker of endothelial dysfunction - von Willebrand factor, ultrasound examination including Dopplerography, cardiotocography.

Statistical analysis was performed using the Statistica package. Quantitative indicators are presented in the form M±SD, where M is the mean value, SD is the standard deviation, qualitative indicators are presented in the form of actual frequencies of observations and their proportion (n, %). Differences at p-level ≤ 0.05 were considered statistically significant.

Verification of gestational arterial hypertension ranged from 22 to 27 weeks (mean 24.6±1.9 weeks). The average blood pressure (BP) values were: systolic blood pressure (SBP) - 145.1±8.7 mmHg versus 115.6±7.2 mmHg in the control group ($p<0.001$), diastolic blood pressure (DBP) — 94.7±8.2 mmHg versus 68.7±6.15 mmHg ($p<0.001$), respectively. The dynamics of increase in SBP, compared with the first trimester, was 22.3% ($p<0.001$), diastolic - 14.9% ($p<0.001$), mean blood pressure - 18.1% ($p<0.05$) and 20.9 mmHg ($p<0.001$).

The marker of endothelial dysfunction, von Willebrand factor, was significantly higher in the group of pregnant women with gestational arterial hypertension - 174.8±28.2% versus 75.1±5.2% in the control group ($p<0.001$).

At 22–26 weeks, ultrasound signs of placental insufficiency (calcifications, expansion of the intervillous space) did not differ significantly in the main and control groups, and at 28–32 weeks of pregnancy they were recorded in 24 (65.0%) women with gestational hypertension ($p <0.001$). An assessment of blood flow velocity curves at 28–32 weeks, according to Doppler ultrasound, indicated that endothelial dysfunction is accompanied by an increase in the value of the resistance index in the uterine arteries in the presence of gestational hypertension - 0.64±0.08 ($p<0.001$) versus 0.45 ±0.05 control group.

In pregnant women with echographic signs of a compensated form of placental insufficiency, an isolated decrease in uteroplacental blood flow was most often observed. The structure of disorders was dominated by changes in hemodynamics of grade IA in 26 (70.3%) women with gestational hypertension ($p < 0.001$), disturbances of grade II were rare - in 1 (2.7%) woman and were statistically insignificant.

Analysis of cardiotocography parameters revealed that the basal rhythm frequency did not differ between groups. In most cases, an undulating type of cardiotocography was observed. A monotonous rhythm was observed in 7 (18.9%) pregnant women with gestational arterial hypertension and was recorded in cases with fetal growth retardation syndrome. Initial signs of fetal hypoxia were detected in 13 (35.1%) cases of gestational hypertension and were absent in the control group ($p < 0.05$). The mean Fisher score was 7.23 ± 0.67 versus 8.42 ± 0.49, respectively ($p<0.001$).

Signs of stage I fetal growth retardation at 32–34 weeks, according to ultrasound examination, were observed in 6 (16.2%) pregnant women with gestational hypertension, stage II - in 1 (2.7%) pregnant woman ($p<0.001$). The average delivery time was 35.0 ± 5.8 weeks for gestational hypertension versus 38.4 ± 0.98 weeks in the control group ($p<0.001$).

Thus, the development of gestational hypertension in women in the second half of pregnancy is accompanied by the development of endothelial dysfunction with an increase in the level of von Willebrand factor, the development of hemodynamic disorders, ultrasound signs of placental insufficiency, fetal hypoxia with the development of fetal growth retardation syndrome.

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