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VASCULAR DISORDERS IN IUGR - CLINICO-MORPHOLOGICAL APPROACHES

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Abstract:
Intrauterine growth restriction (IUGR) is associated with substantive perinatal morbidity and mortality rates. In the structure of perinatal mortality more than 10% are the share of IUGR, and among stillborn - more than 50%. Pathogenesis of IUGR isn't studied yet that doesn't allow to develop pathogenetic reasonable approaches to forecasting, diagnostics and management. The objective of study was to identify pathogenetic mechanisms of IUGR based on studying clinicopathological characteristics of this syndrome. In this study 30 women with pregnancy complicated with IUGR were observed. The patients were divided into three groups according Doppler flow study results. The first group included 10 patients with normal materno-placental circulation, the second - 9 patients with abnormal umbilical Doppler velocimetry, the third - 11 patients with abnormal Doppler velocimetry in uterine arteries; the pregnants with combined disorders were excluded. The study included clinical, sonographic methods, Doppler velocimetry, etc. Examination of placentas was performed by macro- and micromorphometric analysis, immunohystochemical analisys of VEGF (vascular endothelial growth factor) expression. In groups 2 and 3 maternal floor placental infractions were found statistically more often then in group 1 (55,6%, 90,9%, 10% in groups 2,3 and 1). Extent of decrease in weight of a placenta was in 2,3 and 2,8 times higher at patients with abnormal umbilical and uterine Doppler velocimetry ($p<0,05$). Villus maldevelopment was found in 55,6% placentas in group 2 and in 72,7% in group 3 and in 20% in group 1 ($p<0,05$). In placentas of patients with normal haemodynamics , increased quantity of terminal villi, a lot of syncytial knotts (Tenney-Parker changes), predominance of branching angiogenesis were found. Group 2 was characterized by narrowing of the arterioles of the villi up to complete their obliteration, which was accompanied by dilation of venules, so the c volume of the vascular bed in the group most (0,25 ± 0,04 compared to 0,17 ± 0, 04 in group 1 and 0,15 ± 0,03 in group 3). In group 3, there was a decrease in the volume of the vascular bed, poorly developed terminal villy, sclerosis of the villous stroma, a lot of intervillous fibrinoid. The volume of the villi in the placenta third group was the lowest - 0,37 ± 0,05, compared to 0,46 ± 0,07 in group 1 and 0,50 ± 0,04 in group 2, which corresponds to the predominance of non-branching angiogenesis. In placentas tissue from patients with normal haemodynamics strong expression of VEGF was observed. The expression of VEGF in the endothelium of the villi in group 2 was moderate and mild, and in group 3, the expression of this growth factor was absent in all cases. Discussion. The study revealed a number of significant differences in patients with IUGR according Doppler flow study. Our results suggest that VEGF and placental angiogenesis are important components of the IUGR pathogenesis. The study shows correlation of abnormal Doppler flow velocimetry with villous maldevelopment, prevalence of non-branching angiogenesis, decreased VEGF expression and placental compensatory potential reduction. Thus, patients with IUGR need to be approached differentially with the account of Doppler flow study.