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MORPHOFUNCTIONAL CHANGES OF THE PLACENTA IN PREGNANT WOMEN WITH HYPERHOMOCYSTEINEMIA AND ENDOTHELIAL DYSFUNCTION

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Introduction:

Hyperhomocysteinemia is one of the most significant metabolic disorders contributing to endothelial damage and placental insufficiency during pregnancy. The present study investigated the morphofunctional alterations in placental tissue among women with elevated plasma homocysteine levels and signs of endothelial dysfunction.

Materials and Methods:

The study included 36 women aged 20–40 years: 18 with laboratory-confirmed hyperhomocysteinemia (study group) and 18 healthy pregnant women (control group). Venous blood samples were analyzed for total homocysteine, nitric oxide metabolites, and endothelial markers (VCAM-1, ICAM-1). Placental biopsies obtained immediately after delivery were examined histologically using hematoxylin-eosin, PAS, and Mallory trichrome stains. Morphometric measurements included villous diameter, capillary density, and syncytial knot index.

Results:

Placental examination revealed pronounced structural alterations in women with hyperhomocysteinemia. The most common findings included focal fibrinoid necrosis, vascular wall thickening, perivillous fibrin deposition, and a reduction in functional capillaries. The average villous diameter was decreased by 18% compared with the control ($p < 0.05$). Endothelial cell swelling and basal membrane thickening were observed in 70% of the study group samples. Immunohistochemical analysis revealed a twofold increase in VCAM-1 expression ($p < 0.01$), indicating chronic endothelial activation. The syncytial knot index was elevated by 1.8 times, suggesting compensatory response to placental hypoxia.

Discussion:

The findings confirm that hyperhomocysteinemia disrupts endothelial homeostasis and triggers morphofunctional remodeling of placental tissue. Elevated homocysteine levels impair nitric oxide synthesis and induce oxidative stress, leading to microthrombosis and reduced perfusion. These mechanisms underlie chronic fetoplacental hypoxia, which contributes to intrauterine growth restriction and preeclampsia. Similar changes have been reported in studies by Steegers-Theunissen et al. (2013) and Di Simone et al. (2003), supporting the hypothesis of homocysteine-induced vascular injury as a key etiological factor in placental pathology.

Conclusion:

Hyperhomocysteinemia in pregnancy causes distinct morphofunctional changes in placental structure, characterized by villous ischemia, endothelial degeneration, and impaired vascularization. The combination of biochemical and morphological assessment of placental function provides an effective tool for early detection of hypoxic disorders in high-risk

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pregnancies. Timely correction of homocysteine metabolism and antioxidant therapy may help improve maternal and fetal outcomes.

References:

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