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PPARGC1A Gly482Ser POLYMORPHISM AS A MOLECULAR GENETIC PREDICTOR OF EMBRYONIC LOSS IN WOMEN WITH RECURRENT MISCARRIAGE

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Background. Recurrent miscarriage remains one of the most challenging problems in modern obstetrics and reproductive medicine. Despite advances in diagnostic technologies, the causes of early embryonic loss remain unclear in a substantial proportion of cases. Current evidence suggests that genetically determined disturbances of energy metabolism, hemostasis, folate metabolism, and placental microcirculation may contribute to impaired implantation and early embryonic development. Particular attention has recently been paid to the PPARGC1A gene, which is involved in mitochondrial biogenesis, oxidative metabolism, cellular energy homeostasis, and adaptive responses to metabolic stress. Functional polymorphisms of this gene may influence the metabolic stability of trophoblastic and decidual tissues during early gestation and thereby contribute to pregnancy failure.

Objective. To evaluate the association and prognostic significance of the PPARGC1A Gly482Ser polymorphism in the development of embryonic loss in women at high risk of recurrent miscarriage.

Materials and Methods. The study included women examined and treated at the Andijan branch of the Republican Specialized Scientific and Practical Medical Center for Maternal and Child Health and at maternity institutions in Andijan, Uzbekistan. The study population consisted of women with early pregnancy loss and women with uncomplicated pregnancy serving as controls. Molecular genetic testing was carried out to determine polymorphic variants of PPARGC1A Gly482Ser. In addition, polymorphisms of the F2 20210G/A and MTHFR A1298C genes were evaluated as components of the broader genetic profile associated with embryonic loss. Clinical, laboratory, molecular genetic, morphological, and statistical methods were used.

Results. The PPARGC1A Gly482Ser polymorphism demonstrated a strong association with embryonic loss in women at high risk of recurrent miscarriage. The discriminative ability of this genetic marker was high, with an area under the ROC curve of **AUC = 0.97**. The sensitivity was **0.52**, while specificity reached **0.95**. The odds ratio was **OR = 18.78**, indicating a markedly increased probability of embryonic loss associated with the unfavorable genetic variant. These data support the pathogenetic importance of genetically determined disturbances in mitochondrial regulation and energy metabolism during the earliest stages of pregnancy.

The study also demonstrated that unfavorable polymorphic variants of PPARGC1A, F2, and MTHFR were associated with structural abnormalities of chorionic villi and decidual tissue. The most characteristic morphological findings included avascularization and fragmentation of chorionic villi, pronounced stromal edema, hydropic dystrophy of cytotrophoblasts, fibrinoid deposition, necrotic foci, chronic intervillitis, microcirculatory disorders, and ischemic-inflammatory destruction of the villous tree.

These findings indicate that impaired energy metabolism, genetically determined abnormalities of hemostasis, and folate-cycle disturbances may interact within a single pathogenetic mechanism leading to abnormal placentation, trophoblastic dysfunction, and early embryonic death.

Conclusion. The PPARGC1A Gly482Ser polymorphism demonstrated high prognostic potential for embryonic loss in women with recurrent miscarriage. Its association with a high odds ratio and excellent ROC characteristics suggests that PPARGC1A may be considered a promising molecular genetic marker for early risk stratification. Integration of molecular genetic testing with morphological assessment may improve the prediction of adverse pregnancy outcomes and may contribute to the development of individualized monitoring and preventive strategies in women with recurrent reproductive losses.

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