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TITLE

RECURRENT PREGNANCY LOSS ASSOCIATED WITH HEREDITY THROMBOPHILIA

AUTHOR/S

Davidenko N (UA) [1], Loskutova T (UA) [2]

ABSTRACT

Context. Implantation, trophoblastic invasion and subsequent functioning of the placenta are a multi-step process of hemostasis system and endothelial interactions. The role of genetic thrombophilia in the genesis of RPL has not been fully understood.

Objective: To determine the genotype-aggressors that play roll in the development of RPL.

Methods. It was prospective, case-control study. With PCR were detected genetic polymorphisms of coagulation factors and fibrinolysis (1691 G > A F5 Leiden, 20210 G > A prothrombin gene (F2), -675 5G/4G PAI-1, -455 G > A fibrinogen ?). Statistical analysis was performed, p ? 0.05.

Patient(s): 109 pregnant women with RPL and 34 healthy pregnant women in 1st trimester of pregnancy (control group) were examined and tested for the presence of genes polymorphisms.

Result(s): F2 heterozygous variant GA 20210 was determined only in RPL group (p<0,001, OR = 26,47; 95% CI 1,6-445,7), homozygous GG 20210 F2 has protective properties for RPL (p<0,001, OR = 0,03, 95% CI 0,002-0,58). Heterozygous variant 1691 GA F5 Leiden in 3.8 times more often were observed in RPL group (p<0,05, OR = 5,3; 95% CI 1,5-18,5), GG genotype 1691 in 1,4 times - in control group (p<0,05, OR = 0,18; 95% CI 0,05-0,63). PAI-1 genotype 5G/5G has protective properties for the development RPL and occurs in 3,4 times more frequently in pregnant women of control group (p<0,001, OR= 0,16, 95% CI 0,07-0,36). Genotype PAI-1 -675 4G/4G was registered 5.4 times more often in control group (p<0,05, OR=7,5; 95% CI 1,7-33,39). Carriers of genotype - 455 AA FGB in 8,55 times more frequent were registered in the RPL group (p <0,001, OR = 9,7, 95% CI 1,3-74,16).

Conclusions: genes-aggressor of RPL are: heterozygous polymorphisms of F2 20210 G>A and F5-Leiden 1691 G> A, and homozygous polymorphisms of -675 5G/4G PAI-1 and -455 G>A fibrinogen ?.

INSTITUTE

[1] Public Institution "Dnepropetrovsk Center of Primary Health Care N 3", [2] State Establishment "Dnipropetrovsk Medical Academy, of Health Ministry of Ukraine"