

The Role of G/A Polymorphism of the F7 Factor Gene in the Genesis Unsuccessful IVF

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Ummary: The article presents the results of molecular genetic studies of the G/A polymorphism of the F7 gene in 90 unsuccessful IVF women with thrombophilia. The results of the study showed that allele A and hetero-G/A and homozygous non-functional A/A genotypes of the F7 polymorphism are one of the markers of an increased risk of developing thrombophilia in women of the Uzbek population. And the functional allele G and the functionally favorable genotype G/G are functional genotypes in relation to the development of pathology ($\chi^2=15.48$; $p<0.0004$; OR=0.03; 95%CI 0.00 – 0.46).

Keywords: thrombophilia, genetics, G/A polymorphism of the F7 gene, mutation, heterozygote.

Studying the mechanisms of development of obstetric complications (SPP, SORP, FPN, non-developing pregnancies) taking into account the genetic factors of the body may provide some understanding of the pathogenesis, clinical course and development of preventive measures for the disease. [1,2,4,5,9,16,17] Research conducted by scientists Makatsaria A.D., Bitsadze V.O. (2006) devoted to the role of thrombophilia, in particular, APS, mutation of factor V Leiden, prothrombin G202 10A and MTHFR C677T, showed their extremely high frequency not only in patients with various thromboembolic complications, but also typical obstetric complications, such as recurrent miscarriages, severe gestosis, premature abruption of the normally located placenta (PONDP), intrauterine growth retardation syndrome (IUGR), antenatal fetal death (AFF), etc. At the same time, thrombophilia as a complex integral factor in the pathogenesis of the development of gestosis was found in 80% of patients with gestosis. It was noted that among patients with mild forms of gestosis, thrombophilia was detected in 54%, while in the control group of pregnant women with a physiological course of pregnancy - in 16% ($p < 0.05$). The results of the study showed that the MTHFR C677T mutation was in 56.8% of cases, while in the group of mild forms of gestosis and in patients with a physiological course of pregnancy – 44 and 12%. Whereas, polymorphism of the PAI-1 gene was detected in 49.1%. In the group of pregnant women with mild forms of gestosis, this polymorphism was found in 40%, and in the control group of pregnant women with a physiological course of pregnancy - only 16% ($p < 0.05$). [6,7,8,10,12-46]

Also multigene forms of thrombophilia, which amounted to 72.5%, while in the group of mild forms of gestosis only 14%, in the group of physiological pregnancy - 4%, and it should be noted that in the two control groups multigene thrombophilia was represented only by gene polymorphism or heterozygous MTHFR C677T, but not the FV Leiden or prothrombin G20210A mutation.

Antiphospholipid antibodies were detected in 17.3% in the retrospective group, 16.25% in the prospective group, 10% in the mild gestosis group and 4% in the control group with a physiological course of pregnancy. [6,7,9,13,17,18]

The purpose of our research was to study the detectability of polymorphism of genotypes G/A of the F7 gene in women of the Uzbek population.

Material and research methods. A total of 118 women aged from 20 to 39 years were examined. Among 118 women, the main group consisted of 90 (76.3%) with unsuccessful IVF in a woman with thrombophilia and 28 - a control group of healthy women without thrombophilia.

Based on informed consent, molecular genetic testing of the F7 G/A gene was performed using real-time polymerase chain reaction. All pregnant women underwent general clinical, instrumental, functional (ultrasound), and ELISA studies. Pregnant women were consulted by related specialists. (therapist, neurologist, infectious disease specialist, dermatologist, endocrinologist, etc.) Among 118 patients, the main group consisted of 90 women with an established diagnosis of thrombophilia and 28 women who made up the age-matched control group. Molecular genetic examination of biomaterials (DNA) was carried out on the basis of the clinical laboratory of Genotechnologies LLC. DNA extraction from all biological blood samples was carried out using the Ribo-prep kit (Interlabservice, Russia).

To identify the genotype polymorphism consisting of alleles G>A of the F7 gene, allele-specific primers from the manufacturer were selected from DNA samples. To genotype DNA samples using polymerase chain reaction (PCR), 200 DNA samples were examined. For this purpose, the 96-cell automated amplifier "Applied Biosystems Veriti" was optimized according to the following program: initial denaturation once at 180 sec 94°C, 94°C - 10 sec, 64°C - 10 sec, 72°C - 20 sec in the program we did these specified steps 40 times for the polymerase chain reaction to occur. Statistical analysis of the results was carried out using the statistical software package "OpenEpi 2009, Version 2.3".

Research results. The results of molecular genetic studies are presented in the following table. (Table 1)

Table 1. Frequency of distribution of allelic variants and polymorphism of the F7 gene

№	Group	Allele frequency				Genotype distribution frequency					
		G		A		G/G		G/A		A/A	
		n	%	n	%	n	%	n	%	n	%
1.	Main group n=90(180)	138	76,6	42	23,3	55	61,1	28	31,1*	7	7,7
2	Counter. group n=28 (56)	56	100			28	100				

(G/A) in women with thrombophilia and a healthy control group.

N – number of examined patients; *n - number of alleles studied; * - reliability indicator in relation to the control group (P<0.05)

As can be seen from the table, a comparative analysis of the distribution frequencies of alleles and genotypes of the F7 polymorphism (G/A) of the homeostasis system gene among 180 DNA samples in 90 women with unsuccessful IVF in women with thrombophilia, the presence of a functional allele G was 76.6% (138/180) cases, and in the control group this allele was detected in 100% (56/56), which was 1.3 times higher compared to the indicators of the main group. ($\chi^2=15.90$; $p<0.0001$; $OR=0.03$; $95\%CI$ 0.00 – 0.48). While the mutant allele A in the main group was detected in 23.3% of cases (42/180), and in the control group, this allele was not detected in

the studied isolated DNA from healthy women. ($\chi^2=15.90$; $p<0.0001$; $OR=34.68$; $95\%CI2.10 - 573.21$)Общая модель наследования гена F7 (chi-square test, $df = 2$) is presented in the following table (Table 2.)

Table 2. Differences in the frequency of occurrence of alleles and genotypes of the G/T polymorphism of the F7 gene in the group with unsuccessful IVF in women with and without thrombophilia.

Alleles and genotypes	Frequency of occurrence of alleles and genotypes		Statistical difference
	Main group	Control	
Allele G	138	56	$\chi^2=15.90$; $p<0.0001$; $OR=34.68$; $95\%CI2.10 - 573.21$
Allele A	42	0	
Genotype G/G	55	28	$\chi^2=15.48$; $p<0.0004$; $OR=0.03$; $95\%CI0.00 - 0.46$
Genotype G/A	28	0	$\chi^2=15.48$; $p<0.0004$; $OR=25.99$; $95\%CI 1.53 - 440.86$
Genotype A/A	7	0	$\chi^2=15.48$; $p<0.0004$; $OR=5.12$; $95\%CI 0.28 - 92.51$

The results of molecular genetic studies of genotypes of the F7 gene polymorphism showed in the examined patients that the homozygous variant of functional genotypes G/G in the control group of women without thrombophilia was determined in 100% of cases (28/28), and in the main group - 61.1% (55 /90), which was 1.6 times lower than the control group. ($\chi^2=15.48$; $p<0.0004$; $OR=0.03$; $95\%CI0.00 - 0.46$) . Whereas the heterozygous G/A variant of the F7 gene was not detected in the control group. And in the main group of patients, the heterozygous variant G/A of the F7 gene was detected in 31.1% of cases (55/90) ($\chi^2=15.48$; $p<0.0004$; $OR=25.99$; $95\%CI 1.53 - 440.86$) and the mutant homozygous variant A/ A was detected in 7 patients, which amounted to 7.7%. ($\chi^2=15.48$; $p<0.0004$; $OR=5.12$; $95\%CI 0.28 - 92.51$). In the control group of patients, this genotype was not determined.

Analysis of the results obtained indicates that in unsuccessful IVF patients with thrombophilia, the carriage of hetero- and mutant homozygous variants of the F7 gene genotypes was statistically significantly higher. ($P < 0.001$).

According to the literature, the allelic variant c.1238A (heterozygote c.1238G/A and homozygote c.1238A/A) of the F7 gene leads to a decrease in gene expression and a decrease in the level of factor 7 in the blood, and is considered as a protective marker against the development of thrombosis and myocardial infarction.

Taking into account the peculiarities of the physiological adaptation of the hemostatic system to pregnancy, the vast majority of genetic forms of thrombophilia are clinically manifested precisely during the gestational process and, as it turned out, not only in the form of thrombosis, but also in the form of typical obstetric complications.

Studies have established that during this period, a restructuring of the coagulation, anticoagulation and fibrinolytic systems occurs in the mother's body, which leads to an increase in blood clotting factors by 200%. Moreover, in the third trimester, the speed of blood flow in the veins of the lower extremities decreases by half due to partial mechanical obstruction of the venous outflow by the pregnant uterus. The tendency to blood stasis in combination with hypercoagulation during physiological pregnancy predisposes to the development of thrombosis and thromboembolism. And with pre-existing (genetic) TF, the risk of thrombotic and obstetric complications increases tens and hundreds of times, which corresponds to our own research.

To assess the frequency of occurrence of various genotypes of the polymorphic F7 gene and the potential influence of a number of dynamic factors that determine the genetic structure of the

population, as well as to assess the population risk of developing unsuccessful IVF, we analyzed the expected and observed frequency of genotypes of the polymorphism under study and the correspondence of the frequency distribution to the Hardy-Weinberg equilibrium (HV).

Table 3. Expected and observed frequency of distribution of genotypes for RCV polymorphism F7 in the main group of patients.

Genotypes	genotype frequency		χ^2	P
	Observable	expected		
G/G	61,1	53,01	0.588	0,42
G/A	31,1	39,6	0.358	
A/A	7,8	7,4	0.054	
Total	100,00	100,00	0.64	

Based on the calculation by the CV equation, in the main group the frequency of observed favorable genotypes G/G was 1.2 times higher than the expected frequencies - 61.1% and 53.01%, respectively. The heterozygous variant G/A of the observed frequency of the F7 gene was 31.1%, and the theoretically expected frequency was 39.6%, respectively, which indicates an increase in this indicator by 1.3 times. ($P < 0.05$). The frequency of the observed mutant homozygous variant A/A of the F7 gene was 7.8%, and the expected one was 7.4%, which was 1.05 times higher than the expected values ($P > 0.05$).

Table 4. Expected and observed frequency of distribution of genotypes for RCV polymorphism F7 in the control group of patients.

Genotypes	genotype frequency		P
	Observable	Observable	
G/G	100,0	60,6	1.000
G/A	0	34,51	0.000
A/A	0	4,9	0.000
Total	100,00	100,00	0

The results of the analysis of the expected frequencies of genotypes of the F7 gene in the control group showed that the observed frequency of functional genotypes G/G was 100%, while the expected one was 60.6%, which was 1.4 times lower than the observed values. While the observed frequency of the heterozygous variant G/T and mutant homozygous variants of the F 7 gene was 0, the expected frequency was 34.5 and 4.9%, respectively, which indicates an increase in the determinability of carriage of polymorphism in the association of mutant genotypes.

Analysis of the results obtained indicates that the distribution of all genotypes of the F7 polymorphism in the main and control groups corresponds to RHV, indicating the absence of the influence of systematic or random factors that can change the genetic structure of populations. The study of the genetic structure of this marker revealed a relatively high level of expected heterozygosity and homozygous variants of mutant alleles in the main and control groups of pregnant women (39.6% and 34.5%, respectively). In both groups, the D indicator is to the left of 0, that is, it is negative ($D < 0$). The revealed fact indicates higher frequencies of expected heterozygotes and homozygotes, and not the actual calculated frequencies of genotypes.

When analyzing the frequency distribution of alleles and genotypes of this polymorphism in the group of patients of the main group, significant differences were found compared to the control group. The functionally unfavorable allele A of the F7 gene was more than 20 times statistically significantly more prevalent in the studied alleles in unsuccessful IVF patients with thrombophilia (TF) compared to the control group without TF ($\chi^2=15.90$; $p < 0.0001$; $OR=34.68$; $95\% CI 2.10 - 573.21$).

The frequency distribution of genotypes of this polymorphism also revealed significant differences between the main and control comparison groups in the total sample ($P < 0.05$).

Associations of “functionally unfavorable” genotypes G/A ($\chi^2=15.48$; $p<0.0004$; OR=25.99; 95%CI 1.53 – 440.86) and A/A ($\chi^2=15.48$; $p<0.0004$; OR=5.12; 95%CI 0.28 – 92.51) with the development of TF.

Analyzing the results of molecular genetic studies, we can say that allele A and hetero-G/A and homozygous non-functional A/A genotypes of the F7 polymorphism are one of the markers of an increased risk of developing a non-developing pregnancy during IVF with thrombophilia in women of the Uzbek population. And the functional allele G and the functionally favorable genotype G/G are functional genotypes in relation to the development of pathology ($\chi^2=15.48$; $p<0.0004$; OR=0.03; 95%CI 0.00 – 0.46).

Conclusions: Thus, the data from our study showed the connection between the “T” allele and the heterozygous genotype of the F7 gene polymorphism with the development of thrombophilia in women of the Uzbek population. At the same time, the risk of developing pathology when carrying the “T” allele and the G/A genotype increases more than 20 times (OR=34.68; OR=25.99), respectively. The presence of the wild allele and genotype of the F7 gene polymorphism in patients plays a protective role in relation to the formation of TF. The obtained result also indicates that the mutant variant allele A and the heterozygous G/A genotype of the F7 gene polymorphism predict the risk of developing unsuccessful IVF with TF in women of the Uzbek population.

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