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ASSOCIATION OF THE GENETIC POLYMORPHISM G84A IN NOS1 GENE WITH ATRIAL FIBRILLATION IN PATIENTS OF THE GRODNO REGION

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Background. Genetic factors play a major role as a risk factor of atrial fibrillation (AF). Genome-wide association studies have now identified around 140 genetic loci associated with AF. To date, no clinical studies have examined the relationship between the development of AF and the G84A polymorphism of the NOS1 gene, which explains the relevance of this study, which aimed to investigate the association of the genetic polymorphism G84A in NOS1 gene with AF in patients of the Grodno Region of Belarus.

Material and methods. The study included 91 patients with coronary artery disease who were admitted to the Grodno State Clinical Cardiology Center. 49 patients (53.8%) had paroxysmal AF, while 42 patients (46.2%) had sinus rhythm. All patients underwent instrumental, laboratory and molecular genetic testing, including the determining of the G84A polymorphism of the NOS1 gene using the polymerase chain reaction technique.

Results. Patients with AF were predominantly female and had higher BNP levels and larger linear and volumetric parameters of both atria and left ventricle, as well as higher grades of mitral and tricuspid regurgitation ($p<0.05$). In the group of patients with AF, the recessive allele A of the G84A polymorphism was statistically significantly more common (41.4%) as compared to patients with sinus rhythm (19.3%, $p=0.01$), while the GG genotype was statistically significantly less common compared to patients with sinus rhythm ($p=0.034$). The presence of the recessive allele A in the genotype was associated with an increased risk of AF (RR=1.92, 95% CI 1.16–3.18, $p=0.03$).

Conclusion. A statistically significant predominance of the recessive allele A of the G84A polymorphic variant of the NOS1 gene was found in patients with AF compared to patients with sinus rhythm. This genetic predisposition can be taken into account in differentiated therapy of patients with cardiac arrhythmias.

***Keywords**: atrial fibrillation; NOS1 gene; genetic polymorphism G84A; sinus rhythm.*

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Introduction

Atrial fibrillation (AF) nowadays is the most frequent type of cardiac arrhythmia [1]. It appears due to abnormalities in muscle layer of atria. It is mostly associated with increased heart rate and is classified into paroxysmal and persistent types [1]. Paroxysmal AF presents less than 7 days and persistent presents more than 7 days. In AF due to abnormal rapid contraction of atria, blood stasis occurs which promotes thrombus formation in atria. Multiple social, lifestyle factors and other heart diseases predisposes to AF and genetics too play a major role as a risk factor of AF [2].

Genome wide association studies have now identified around 140 genetic loci associated with AF [2]. Studies into the effects of several loci and their tentative gene targets have identified novel pathways associated with AF development [2]. However further validations of causality are still needed for many implicated genes. Genetic variants at identified loci also help predict individual AF risk and response to different therapies.

The NOS1 gene is located on the long arm of chromosome 12 (12q24.22) and includes 33 exons [3]. To date, more than 100 polymorphisms of the NOS1 gene are known [3]. The polymorphism in exon 1c of the promoter of this gene, which is manifested by the substitution of guanine (G) for adenine (A) in the 84th position of the nucleotide sequence (rs41279104), has been most fully investigated. In all likelihood, this substitution contributes to a decrease in the expression of the NOS1 gene: genotype –84AA and allele –84A are associated with a 30% reduced in vitro and 50% in vivo expression of neuronal NO synthase, which may reduce the effectiveness of its physiological effects in the myocardium [4].

In clinical studies studying this polymorphism, the presence of the recessive A allele was significantly associated with the development of ischemic stroke [5], type 1 diabetes mellitus [6], post-traumatic gonarthrosis [7], and decreased vasodilation in myocardial infarction [8].

To date, no clinical studies have been conducted on the relationship between the development of AF and the G84A polymorphism of the NOS1 gene, which explains the relevance of this study.

Aim of the study was to evaluate association of genetic polymorphism G84A in NOS1 gene in patients with AF in Grodno Region of Belarus.

Material and methods

The study included 91 patients with chronic forms of coronary artery disease who were admitted to the Grodno State Clinical Cardiological Center for treatment. 49 patients (53.8%) had paroxysmal AF, while 42 patients (46.2%) had sinus rhythm.

Exclusion criteria from the study were: acute myocardial infarction, unstable angina, myocarditis, chronic rheumatic heart disease, valvular pathology of the heart requiring surgical correction, prosthetic heart valves, coronary artery bypass grafting, or coronary angioplasty (less than 3 months before

enrollment in the study), oncological diseases and severe concomitant extracardiac pathology.

All patients underwent clinical, laboratory, and instrumental studies, including the determination of the G84A polymorphism of the NOS1 gene using the polymerase chain reaction technique. Venous blood was used as the test material for studying the polymorphism. Isolation of human genomic DNA was carried out using the DNA-Extran-1 reagent kit («Synthol», Russian Federation). Each polymorphic variant of was identified using the corresponding reagent kit manufactured by «Litekh» (Russian Federation). DNA amplification was carried out on a Rotor Gene-Q amplifier («Qiagen», Germany). The distribution of alleles and genotypes in the studied groups corresponded to the Hardy-Weinberg equilibrium (p>0.05).

Echocardiography was performed on Phillips iE33 device with a multi-frequency sensor (frequency 2.5-5.0 MHz). The examination was performed with the patient lying on his left side with his back to the researcher or on his back. The study protocol included the following indicators: left atrium (LA) and right atrium (RA) diameter in 2-chamber and 4-chamber mode, end-systolic diameter and end-diastolic diameter (mm) of the left ventricle (LV), LVEF; assessment of the state of the valvular apparatus of the heart, degree of regurgitation on the valves.

Statistical analysis was performed using the STATISTICA 12.0 software package with a preliminary check for normal distribution using a distribution histogram. Quantitative data, the distribution of which was not normal, were given as a median, 25% and 75% quartiles. Since most of the quantitative characteristics did not obey the normal distribution law, non-parametric methods were used for comparison. The Mann-Whitney test was used to assess differences in quantitative traits between two independent groups. At a significance level of p less than 0.05, it was believed that the studied indicator in the compared groups had statistically significant differences. The study was performed in accordance with Good Clinical Practice standards and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to inclusion in the study.

Results

Clinical characteristics of the patients are presented in Table 1.

Patients with AF and sinus rhythm were comparable in age (p>0.05). In AF group female gender was prevalent, while patients with sinus rhythm were predominantly male (p<0.001). It is interesting that patients with AF had significantly higher body mass index (32.7 [29; 36] vs 29.4 [27; 31] kg/m2, p=0.008) and more often had obesity (51% vs 19%. p=0.001) than patients with sinus rhythm. Patients of both groups had no difference in prevalence of hypertension (91% vs 92%, p>0.05) and diabetes mellitus (8% vs 7%, p>0.05). Patients with sinus rhythm more often had stable angina (64% vs 26%, p=0.002), however myocardial infarction prevalence in both groups was comparable (15% vs 12%, p>0.05). However, patients of both groups had no differences in heart failure NYHA Class (p>0.05).

Table 1 – Clinical characteristics of patients

Parameters	Atrial fibrillation (n=49)	Sinus rhythm (n=42)	p
Male gender, n (%)	37 (75.5%)	16 (38.1%)	**<0.001**
Age, years	61.6 [54; 68]	60.7 [53; 66]	0.425
Body mass index, kg/m2	32.7 [29; 36]	29.4 [27; 31]	**0.008**
Obesity, n (%)	25 (51%)	8 (19%)	**0.002**
Hypertension, n (%)	45 (91.8%)	39 (92.9%)	0.856
Stage 1, n (%)	11 (22.4%)	10 (23.8%)	0.878
Stage 2, n (%)	34 (69.4%)	27 (64.3%)	0.606
Stage 3, n (%)	0 (0%)	2 (4.8%)	0.408
Stable angina, n (%)	13 (26.5%)	27 (64.3%)	**<0.001**
Class 1, n (%)	4 (8.2%)	6 (14.3%)	0.352
Class 2, n (%)	8 (16.3%)	19 (45.2%)	**0.003**
Class 3, n (%)	1 (2%)	2 (4.8%)	0.469
Myocardial infarction history, n (%)	6 (12.2%)	6 (14.9%)	0.775
Diabetes mellitus, n (%)	4 (8.2%)	3 (7.1%)	0.856
Heart failure NYHA Class	-	-	
Class 1, n (%)	3 (6.1%)	2 (4.8%)	0.777
Class 2, n (%)	37 (75.5%)	36 (85.6%)	0.340
Class 3, n (%)	9 (18.4%)	4 (9.6%)	0.368

Laboratory parameters of patients are presented in Table 2.

Table 2 – Laboratory parameters of patients (Me [25%;75%])

Parameters	Atrial fibrillation (n=49)	Sinus rhythm (n=42)	p
RBC, 10^{12}/L	4.8 [4.3; 5.2]	4.5 [4.3; 4.8]	0.053
Hemoglobin, g/L	142.7 [133.5; 159.2]	141.1 [132; 149.5]	0.061
WBC, 10^9/L	9.9 [5.6; 7.6]	6.1 [4.8; 7.2]	0.160
ESR, mm/h	11.7 [4; 14.5]	14.8 [6; 20.25]	0.069
Urea, mmol/L	6.6 [5.3; 7.6]	5.6 [4.7; 6.3]	**0.017**
Creatinine, µmol/L	106.8 [85; 123]	91.8 [78; 101.3]	**0.044**
eGFR, ml/min/1.73m^2	67.2 [51; 82]	72.5 [57; 86]	0.383
Cholesterol, mmol/L	4.9 [3.8; 5.9]	5.6 [4.5; 6.9]	0.113
Glucose, mmol/L	6.6 [5.2; 6.5]	5.8 [5.4; 6.2]	0.531
Sodium, mEq/L	142.6 [140.9; 145]	143.6 [142; 145.7]	0.428
Potassium, mEq/L	4.5 [4.2; 4.7]	4.3 [4.1; 4.5]	0.141
BNP, pg/mL	1287.2 [571; 1376]	322 [322; 322]	**0.004**

Abbreviations: RBC – red blood cells; WBC – white blood cells; ESR – erythrocyte sedimentation rate; eGFR – estimated glomerular filtration rate; BNP – brain natriuretic peptide.

Laboratory markers of patients in both groups had no significant differences except for urea (p=0.017), creatinine (p=0.044) and BNP levels (p=0.004). Complete blood count parameters in both grours were comparable.

Echocardiographic parameters of patients are presented in Table 3.

According to the results of trannsthoracic echocardiography, patients had significant differences in volumes in diameters of both atria and ventricles (p<0.001). Also, patients with AF had lower LVEF (p=0.001) and higher grades of both mitral and tricuspid regurgitation (p<0.05), which can be explained by cardiac remodeling induced by AF development.

The distribution of genotype and allele frequencies for the G84A polymorphism of the NOS1 gene is presented in Table 4. Thus, it was found that in the studied sample, the dominant allele G was found in 70.3% of cases and the recessive allele A was found in 29.7% of cases. The distribution corresponded to the Hardy-Weinberg equilibrium ($\chi 2$=1,37, p=0,69).

Table 4 – Frequency distribution of genotypes and alleles of the G84A polymorphism of the NOS1 gene (abs./%)

Parameter	Frequency (number / %)	
	Number	%
Genotype (n=91)		
GG	47	51.6
GA	34	37.4
AA	10	11.0
Allele (n=182)		
G	128	70.3
A	54	29.7

Table 3 – Echocardiographic parameters of patients (Me [25%;75%])

Parameter	Atrial fibrillation (n=49)	Sinus rhythm (n=42)	p
LA diameter (2 chamber), mm	44.1 [42; 45]	36.8 [34; 39]	**<0.001**
LA diameter (medial to lateral), mm	43.1 [40; 45]	37 [34; 41]	**<0.001**
LA diameter (front to back), mm	60.5 [56; 65]	50.6 [47; 54]	**<0.001**
RA diameter (medial to lateral), mm	41.1 [38; 44]	35.2 [33; 38]	**<0.001**
RA diameter (front to back), mm	57 [52; 61]	47.6 [45; 50]	**<0.001**
LV ESD, mm	36.9 [49; 55]	32.4 [30; 35]	**<0.001**
LV EDD, mm	53.1 [49; 55]	50.5 [48; 54]	0.084
M-mode	-	-	-
LV ESV, ml	73.8 [46.75; 65.75]	44.4 [35; 51]	**<0.001**
LV EDV, ml	142 [117; 151.5]	148.3 [108; 144]	0.061
LVEF, %	57.2 [56; 60]	64.6 [62; 68]	**<0.001**
B-mode	-	-	-
LV ESV, ml	73.8 [48.75; 69.75]	41.6 [29.75; 51;75]	0.025
LV EDV, ml	147.2 [116; 155]	117 [87; 132]	0.260
LVEF, %	52.6 [49; 58]	64.2 [60; 66]	**0.001**
Septal thickness (systolic), mm	17 [15; 19]	16.6 [16; 18]	0.833
Septal thickness (diastolic), mm	13.3 [12; 15]	12.3 [12; 13]	**0.034**
Posterior wall thickness (systolic), mm	17.4 [16; 19]	16.2 [15; 18]	0.081
Posterior wall thickness (diastolic), mm	12.1 [11; 13]	11.2 [10; 12]	**0.015**
Right ventricle diameter, mm	26.3 [24; 28]	24.5 [23; 26]	**0.010**
Contractility index	1.1 [1; 1]	1 [1; 1]	0.383
MR grade 1, n (%)	12 (24.5%)	25 (59.5%)	**0.002**
MR grade 2, n (%)	34 (69.4%)	17 (40.5%)	**0.011**
MR grade 3, n (%)	2 (4.1%)	0 (0%)	0.778
TR grade 1, n (%)	11 (22.4%)	25 (59.5%)	**<0.001**
TR grade 2, n (%)	35 (71.4%)	16 (38.1%)	**0.003**
TR grade 3, n (%)	2 (4.1%)	0 (0%)	0.778
AR grade 1, n (%)	16 (32.7%)	6 (14.3%)	**0.042**
AR grade 2, n (%)	8 (16.3%)	4 (9.5%)	0.519

Abbreviations: LA – left atrium; RA – right atrium; LV – left ventricle; ESD – end-systolic diameter; EDD – end-diastolic diameter; ESV – end-systolic volume; EDV – end-diastolic volume; LVEF – left ventricular ejection fraction; MR – mitral regurgitation; TR – tricuspid regurgitation; AR – aortic regurgitation.

When studying the frequency of distribution of genotypes and alleles of the G84A polymorphism among patients of the study groups (Table 5), we found that in the group of patients with AF, the recessive allele A was significantly more common (41.4%) compared to patients with sinus rhythm (19.3%, p=0.01). In the group of patients with AF, GG genotype was significantly less common compared to patients with sinus rhythm (p=0.034), while AA genotype was more common in AF group compared to patients with sinus rhythm (p=0.012).

When assessing the relative risk of AF development depending on the polymorphic variant of the NOS1 gene, the following results were obtained. The presence of the recessive allele A in the genotype was associated with an increased risk of AF (RR=1.92, 95% CI 1.16–3.18, p=0.03). At the same time, the presence of the G allele in the genotype reduced the risk of AF development (RR=0.77, 95% CI 0.64–0.93, p=0.008), as did the presence of the GG genotype (RR=0.64, 95% CI 0.43–0.95, p=0.01).

Discussion

According to literature data, the frequency of the dominant G allele of the G84A polymorphism of the

Table 5 – Frequency distribution of genotypes and alleles of the G84A polymorphism of the NOS1 gene among patients with sinus rhythm and atrial fibrillation

Parameter	Frequency (number / %)				p
	Atrial fibrillation (n=49)		Sinus rhythm (n=42)		
	Number	%	Number	%	
Genotype					
GG	19	40.5	29	66.0	**0.034**
GA	21	44.6	12	27.2	0.138
AA	7	14.9	3	6.8	**0.012**
Allele					
G	59	58.6	71	80.7	**0.010**
A	35	41.4	17	19.3	
Hardy-Weinberg equilibrium correspondence	$\chi2$=0.29, p=0.58		$\chi2$=0.12, p=0.73		-

NOS1 gene in the global population is about 88%, while the frequency of the recessive A allele is 12% [3]. However, in the European population, this ratio is 80% to 20%, while in the African and Latin American populations it is 93% to 7% [3].

In patients without cardiac arrhythmias who formed the control group in our study, the distribution of allele frequencies corresponded to the literature data (80.7% – allele G and 19.3% – allele A). While in patients with AF, the frequency of the A allele was significantly higher (41.4%, p=0.01) than in the general population.

The clinical significance of the G84A polymorphism of the NOS1 gene has been widely covered in international scientific studies related to various branches of medicine, which demonstrates the crucial role of the nitric oxide synthesis system in the functioning of all organs and systems of the body.

It is reported that the presence of the recessive allele A of the G84A polymorphism of the NOS1 gene in the genotype of patients increased the risk of type 1 diabetes by 1.49 times (p=0.016) [7]. S.D. Yoo et al. found that a two-locus combination of the allele A of the G84A polymorphism of the NOS1 gene and the allele G of the 3238C/G polymorphism of the ApoC3 gene was associated with the risk of ischemic stroke in the Korean population (RR=4.34, 95% CI 2.71–6.95) [5]. At the same time, N. Shnayder et al. described that no statistically significant differences were found in the distribution of genotypes and alleles of the G84A polymorphism in patients with hypertension and migraine, and no relationship was found between the genotypes of patients and the severity of the diseases studied [6].

D. Galimberti et al. stated that no differences were found in the distribution of alleles and genotypes of the G84A polymorphism in patients with Alzheimer's disease and in healthy individuals [9]. T. Okumura et al. showed that no relationship was found between the presence of the recessive allele A and the development of schizophrenia in the Japanese population [10], which was subsequently confirmed in a meta-analysis conducted by S. Ahmed et al. [11]

Of great interest is the study by O. Nobrega et al., which investigated the relationship between the distribution of alleles and genotypes of the G84A polymorphism of the NOS1 gene and the concentration of NO in blood plasma in patients with myocardial infarction [12]. Thus, according to the data obtained, patients with AA genotype had the lowest concentration of NO on the fifth day after infarction compared with other genotypes (p<0.05), as well as the smallest percentage of flow-mediated dilation of the coronary arteries (p=0.009), which indicates an insufficient degree of NO production by the endothelium [12].

Conclusion

1. Patients with AF had higher BNP levels and larger linear and volumetric parameters of both atria and left ventricle, as well as higher class of mitral and tricuspid regurgitation (p<0.05).

2. In patients with paroxysmal and persistent forms of AF, the recessive allele A and genotype AA of the G84A polymorphism of the NOS1 gene was significantly more common compared to patients with sinus rhythm (p<0.05).

3. Patients with the recessive allele A of the G84A polymorphism of the NOS1 gene have an increased risk of AF development (RR=1.92), which can be taken into account in the differentiated therapy of patients with cardiac arrhythmias.

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АССОЦИАЦИЯ ГЕНЕТИЧЕСКОГО ПОЛИМОРФИЗМА G84A В ГЕНЕ NOS1 У ПАЦИЕНТОВ С ФИБРИЛЛЯЦИЕЙ ПРЕДСЕРДИЙ В ГРОДНЕНСКОЙ ОБЛАСТИ

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Введение. Генетические факторы играют важную роль в качестве фактора риска фибрилляции предсердий (ФП). Полногеномные исследования в настоящее время выявили около 140 генетических локусов, связанных с ФП. На сегодняшний день не проводилось клинических исследований связи между развитием ФП и полиморфизмом G84A гена NOS1, что объясняет актуальность данного исследования, целью которого было изучение ассоциации генетического полиморфизма G84A гена NOS1 у пациентов с ФП в Гродненской области Республики Беларусь.

Материал и методы. В исследование включен 91 пациент с ишемической болезнью сердца, находившийся на лечении в Гродненском государственном клиническом кардиологическом центре. У 49 пациентов (53,8%) была пароксизмальная форма ФП, у 42 пациентов (46,2%) – синусовый ритм. Всем пациентам проводились инструментальные, лабораторные и молекулярно-генетические методы исследования, в том числе определение полиморфизма G84A гена NOS1 методом полимеразной цепной реакции.

Результаты. Пациенты с ФП были преимущественно женского пола, имели более высокий уровень BNP и большие линейные и объемные параметры как предсердий, так и левого желудочка, а также более высокую степень митральной и трикуспидальной регургитации ($p<0,05$). В группе пациентов с ФП рецессивный аллель А полиморфизма G84A встречался статистически значимо чаще (41,4%) по сравнению с пациентами с синусовым ритмом (19,3%; $p=0,01$), тогда как генотип GG встречался статистически значимо реже по сравнению с пациентами с синусовым ритмом ($p=0,034$). Наличие рецессивного аллеля А в генотипе ассоциировалось с повышенным риском ФП (RR=1,92; 95% ДИ: 1,16–3,18; $p=0,03$).

Выводы. Выявлено статистически значимое преобладание рецессивного аллеля А полиморфного варианта G84A гена NOS1 у пациентов с ФП по сравнению с пациентами с синусовым ритмом. Данную генетическую предрасположенность можно учитывать при дифференцированной терапии пациентов с нарушениями сердечного ритма.

Ключевые слова: *фибрилляция предсердий, ген NOS1, генетический полиморфизм G84A, синусовый ритм*

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