

Association of PECAM1 Val125Leu and Gly670Arg Polymorphisms With Risk of Atherosclerosis in a Lur Population of Iran

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Abstract- According to importance of PECAM1 in pathogenesis atherosclerosis, we intend to investigate the Val125Leu and Gly670Arg polymorphisms with risk of atherosclerosis in the Lur population of Iran among the individuals who underwent coronary angiography. A genetic association study was performed on 154 approved cases of atherosclerosis and 145 ruled out cases for atherosclerosis based on the expert opinion of interventional cardiologists. Atherogenic indices including atherogenic coefficient (AC), non-HDL-C fraction, Castelli's Risk Indices-I and -II (CRI-I and -II), atherogenic index of plasma (AIP), and Triglycerides-Glucose (TYG) were all calculated. Logistic regression was used and odds ratio (OR) was reported. Genotype Leu/Leu showed an association with increased risk of atherosclerosis. However, this association was not statistically significant (odds ratio [OR]=1.86, $P=0.066$). Allele Leu showed an association with increased risk of atherosclerosis. However, this association was not statistically significant (OR=1.34, $P=0.069$). For polymorphism Gly670Arg, no genotype or allele was associated with risk of atherosclerosis ($P>0.05$). The most important atherogenic index was AC (area under curve=0.719). After adjusting genotype Leu/Leu with AC as well as age, gender and body mass index, a significant harmful effect was observed for this genotype (OR=2.35, $P=0.041$). Susceptibility to atherosclerosis does not have a crude association with Val125Leu and Gly670Arg polymorphisms of PECAM1 gene among the patients with acute chest pain in Lur population of Iran. However, besides AC and some demographic variables, genotype Leu/Leu showed a harmful association. © 2026 Tehran University of Medical Sciences. All rights reserved.

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Introduction

Atherosclerosis—sometimes called as coronary artery disease (CAD)—is a major cause of morbidity and mortality worldwide. Many old aged individuals suffer from atherosclerosis complications (1). Although males are more susceptible, both genders are involved with CAD. Many risk factors are known for CAD. One of the most famous risk factors is smoking which seems to be more dangerous in women (2). Pathophysiologically, atherosclerosis is the basis of CAD and is a progressive

condition consisting of a plaque formed from accumulation of fibers, cholesterol, and elements like calcium (3). Accordingly, an atherosclerotic plaque is formed between intima and media of coronary arteries (4). In addition, thrombosis can be added on the plaques and therefore acute coronary syndrome is occurred (5).

Atherosclerosis is a complex process based on inflammatory events that reactive oxygen species (ROS) are created by endothelium, smooth muscles and immune cells. In addition, ROS plays role in formation of thrombosis, affecting lipoproteins and changing

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bioavailability of nitric oxide (NO) (6-8). Hence, cardiovascular events have multifactorial etiology. One of these etiologies is the inflammatory basis including adhesion molecules expression by endothelial cells. This process results in adhesion and implantation of circulating immune cells on the forming atherosclerosis (9,10). The adhesion molecules are mainly inter-cellular adhesion molecule-1 (ICAM1), vascular adhesion molecule-1 (VCAM1), platelet and endothelial cell adhesion molecule-1 (PECAM1), E-selectin and P-selectin (11,12).

Another risk factor for atherosclerosis is dyslipidemia. In the general population, dyslipidemia is a substantial risk factor for future cardiovascular events (13,14). When lipid parameters including total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), and triglyceride (TG) appear to be normal, studies have shown that lipid fractions such as Castelli's risk index-I (CRI-I), Castelli's risk index-II (CRI-II), the plasma atherogenic index (AIP), atherogenic coefficient (AC), Non-HDL- Cholesterol Index (NHC), and triglyceride glucose index (TYG) can predict the risk of developing cardiovascular events (15,16). Atherogenic indices measure how probable serum lipids are to cause atherosclerotic alterations. They are becoming more widely acknowledged as useful indicators of cardiovascular disease (CVD) risk (17). Atherogenic indices assess the likelihood of serum lipids causing atherosclerotic changes. They are increasingly being recognized as useful indicators of CVD risk (17).

Like many other diseases, genetic basis in atherosclerosis is of great importance. Each of the involving molecules mentioned in the pathogenesis of atherosclerosis has an encoding gene that these genes have in turn single nucleotide polymorphisms (SNPs). According to the genetic association studies, many of these genes have association with risk of atherosclerosis (18). Previously, polymorphisms of vitamin D receptor (19), apolipoprotein E (20), angiotensin converting enzyme (ACE) (21), interleukin 6 (22), and so on have been shown to be associated with atherosclerosis in meta-analyses.

According to importance of PECAM1 in pathogenesis atherosclerosis and CAD, we intend to investigate the Val125Leu and Gly670Arg polymorphisms with risk of atherosclerosis in the Lur population of Iran among the individuals who underwent coronary angiography. Atherogenic indices were also investigated.

Materials and Methods

Study design

As a case-control study, a genetic association study was conducted on the blood samples of a previous project (23). The samples had been taken by approval of the ethics committee of Lorestan University of Medical Sciences with registration number IR.LUMS.REC.1396.314 along with informed consent from the patients regarding any reasonable laboratory study on their blood samples.

Samples and groups

The samples consisted of 154 approved cases of atherosclerosis and 145 ruled out cases for atherosclerosis in Shahid Madani center of cardiovascular disease in Khorramabad, west of Iran. The diagnosis was through coronary angiography. The patients were those who had acute chest pain and were subjected to coronary angiography. Accordingly, the cases and the controls were selected based on the expert opinion of an interventional cardiologist. The exclusion criteria had been malignancies, congenital heart disease, chronic kidney disease, pulmonary obstruction, rheumatology diseases and treatment with corticosteroids or any other confounding drug. The base line characteristics had been reported in the mentioned study.

Atherogenic indices calculation

Atherogenic indices were calculated by lipid profile parameters and fasting blood sugar (FBS) based on the information of the previous study (23). The following formulas were used to calculate the atherogenic indices (24,25):

$CRI-I = TC / HDLc$, $CRI-II = LDL / HDLc$, $AIP = \log TG / HDLc$, $NHC = TC - HDLc$, $TYG = \ln((TG \times FBS) / 2)$, $AC = (TC - HDL) / HDLc$

Laboratory study

From each patient, genomic DNA had been extracted from weight blood cells (WBC) and stored at -20 °C. The polymorphisms of Val125Leu and Gly670Arg PECAM1 gene were studied by polymerase chain reaction with sequence specific primers (PCR-SSP). Dihydrofolate reductase (DHFR) was used as internal control. The primer sequences and product sizes are shown (Table 1).

Genotyping

Since polymorphisms were resulted in amino acid substitution (i.e. missense mutation), the alleles and genotypes were named by the abbreviations of their amino acids. Accordingly, for Val125Leu polymorphism, the genotypes were Val/Val, Val/Leu and Leu/Leu, and

for Gly670Arg polymorphism, the genotypes were Gly/Gly, Gly/Arg and Arg/Arg. To count alleles, both copies were regarded (two-fold of sample size).

Statistical analysis

Hardy-Weinberg equilibrium (HWE) was calculated with chi-square. Simple logistic regression was used to study the association of alleles and genotypes with the outcome, and odds ratios (OR) and 95% confidence intervals (CI) were reported. Predictive role of atherogenic indices for atherosclerosis was studied by receiver operating characteristics (ROC) analysis and area under the curve (AUC). One-way analysis of variance (ANOVA) was used to study association of atherogenic indices with genotypes. Multiple logistic regression was regarded if applicable. *P* less than 0.05 was considered as the significance level in SPSS 24 software (IBM, NY, US).

Results

Demographic characteristics

A total of 154 cases and 145 controls were studied. Among the cases, there were 42.76% males, while among the controls, there were 52.6% males (*P*=0.089). Age wise, the average age was 63.57±10.65 years in cases and 55.57±9.98 years in controls (*P*<0.001). BMI was significantly higher in the cases (29.35±4.96) compared with the controls (26.79±5.07) (*P*<0.001). No significant difference was observed for systolic blood pressure (133.07±15.66 [cases] vs 130.86±13.59 [controls] mmHg) and diastolic blood pressure (82.09±10.11 [cases] vs 80.39±10.15 [controls] mmHg) (*P*>0.05).

The genotype and allele frequencies

For polymorphism Val125Leu, HWE was established for all samples, case group and control group (*P*>0.05). Genotype Leu/Leu showed an association with increased risk of atherosclerosis. However, this association was not statistically significant (OR=1.86, *P*=0.066) (Table 2). Allele Leu showed an association with increased risk of atherosclerosis. However, this association was not statistically significant (OR=1.34, *P*=0.069) (Table 3).

For polymorphism Gly670Arg, HWE was established for all samples, case group and control group (*P*>0.05). No genotype was associated with risk of atherosclerosis (*P*>0.8) (Table 4). In addition, no allele was associated with risk of atherosclerosis (*P*>0.8) (Table 5).

The results of gel electrophoresis are shown for Val125Leu and Gly670Arg polymorphisms (Figure 1).

Atherogenic indices

The most important atherogenic index was AC (AUC=0.719) followed by TYG (AUC=0.71) (Table 6). The atherogenic indices were compared between the genotypes using one-way ANOVA. Hereby, no significant association was found (Tables 7 and 8).

Adjusted model

According to the borderline effect of Leu/Leu genotype in crude models, an adjusted model was performed consisting of age, gender, body mass index as well as AC as the most important atherogenic index. Accordingly, Leu/Leu genotype was a significant risk factor for susceptibility to atherosclerosis (OR=2.35, *P*=0.041) (Table 9).

Table 1. The list of primers used in the study

Primer name	Sequence	TM	Size (bp)
125Leu forward	AACCACTGCAGAGTACCAGC	55.18	310
125Val forward	AACCACTGCAGAGTACCAGG	55.18	
Val125Leu reverse	GACAGAGTGAGACTCCATCAA	51.41	
760Gly forward	TAGGTCACAATGACGATGTCG	53.69	220
670Arg forward	TAGGTCACAATGACGATGTCA	51.41	
Gly670Arg reverse	AGCAGCTAGCTACCTTCATTG	52.77	
DHFR forward	GCATGTCTTTGGGATGTGGA	57.30	280
DHFR reverse	GGAATGGAGAACCAGGTCTT	57.30	

Table 2. Frequency of Val125Leu genotypes and their association with CAD

Genotype	CAD	Control	Total	Odds ratio	95% CI	<i>P</i>
Val/Val	33 (21.4%)	43 (29.7%)	76 (25.4%)	Reference		
Val/Leu	81 (52.6%)	74 (51.0%)	155 (51.8%)	1.42	0.82 – 2.47	0.208
Leu/Leu	40 (26.0%)	28 (19.3%)	68 (22.7%)	1.86	0.96 – 3.61	0.066
Total	154 (100%)	145 (100%)	299 (100%)			

Table 3. Frequency of Val125Leu alleles and their association with CAD.

Allele	CAD	Control	Total	Odds ratio	95% CI	P
Val	147 (47.7%)	160 (55.2%)	307 (51.3%)	Reference		
Leu	161 (52.3%)	130 (44.8%)	291 (48.7%)	1.34	0.98 – 1.86	0.069
Total	308 (100%)	290 (100%)	598 (100%)			

Table 4. Frequency of Gly670Arg genotypes and their association with CAD.

Genotype	CAD	Control	Total	Odds ratio	95% CI	P
Gly/Gly	35 (22.7%)	34 (23.4%)	69 (23.1%)	Reference		
Gly/Arg	81 (52.6%)	77 (53.1%)	158 (52.8%)	1.02	0.58 – 1.79	0.940
Arg/Arg	38 (24.7%)	34 (23.4%)	72 (24.1%)	1.08	0.56 – 2.10	0.807
Total	154 (100%)	145 (100%)	299 (100%)			

Table 5. Frequency of Gly670Arg alleles and their association with CAD

Allele	CAD	Control	Total	Odds ratio	95% CI	P
Gly	151 (49.0%)	145 (55.2%)	307 (50.0%)	Reference		
Arg	157 (51.0%)	145 (44.8%)	291 (50.0%)	1.04	0.75 – 1.43	0.812
Total	308 (100%)	290 (100%)	598 (100%)			

Table 6. ROC curve analysis using atherosclerosis markers to distinguish the different groups in Control and Patients group

	Area	Standard Error	P	95% CI	
Atherogenic index				Lower Bound	Upper Bound
AC	0.719	0.025	<0.001***	0.670	0.768
TYG	0.711	0.025	<0.001***	0.662	0.761
NHC	0.693	0.026	<0.001***	0.642	0.744
CRI-II	0.653	0.027	<0.001***	0.600	0.706
CRI-I	0.653	0.027	<0.001***	0.600	0.705
AIP	0.651	0.027	<0.001***	0.599	0.704

Note: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. TYG: triglyceride glucose index; CRI-I: Castelli's Risk Index, AC: atherogenic coefficient, AIP: Atherogenic index of plasma; NHC: Non-HDL-Cholesterol

Table 7. Relationship between PECAM1 Val125Leu genotypes and atherogenic markers

Variable	PECAM1 Val 125 Leu			P
	Val/Val genotype (N=76) (mean ± SD)	Val/Leu genotype (N=155) (mean ± SD)	Leu/Leu genotype (N=68) (mean ± SD)	
AIP	0.15 ± 0.24	0.17 ± 0.21	0.16 ± 0.25	0.89
CRI-1	3.9 ± 2.8	3.8 ± 2	3.9 ± 2.3	0.96
CRI-2	2.2 ± 0.89	2.3 ± 0.95	2.4 ± 1	0.39
NHC	122.6 ± 60	122.1 ± 50	128.5 ± 45	0.67
AC	3.32 ± 1.7	3.4 ± 1.5	3.4 ± 1.4	0.85
TYG	8.7 ± 0.63	8.7 ± 0.54	8.7 ± 0.62	0.76

Table 8. Relationship between Gly670Arg genotypes and atherogenic markers

Variable	PECAM1 Gly670Arg			P
	Gly/Gly genotype (N=69) (mean ± SD)	Gly/Arg genotype (N=158) (mean ± SD)	Arg/Arg genotype (N=72) (mean ± SD)	
AIP	0.19 ± 0.24	0.16 ± 0.22	0.14 ± 0.23	0.44
CRI-1	4.3 ± 3.0	3.7 ± 2.1	3.6 ± 1.9	0.22
CRI-2	2.4 ± 0.95	2.4 ± 0.96	2.2 ± 0.95	0.30
NHC	116.42 ± 45	126.16 ± 55	125.31 ± 49	0.40
AC	3.2 ± 1.4	3.4 ± 1.4	3.4 ± 1.9	0.78
TYG	8.7 ± 0.61	8.7 ± 0.59	8.7 ± 0.54	0.99

Table 9. Multiple logistic regression model to show adjusted effect of Val125Leu genotypes

Genotype	Odds ratio	95% CI	P
Genotype			
Val/Val	Reference		
Val/Leu	1.51	0.77 – 2.95	0.233
Leu/Leu	2.35	1.03 – 5.32	0.041*
Other covariates			
AC	1.72	1.39 – 2.12	<0.001***
Age (1 year)	1.09	1.06 – 1.12	<0.001***
Gender (male)	3.50	1.92 – 6.36	<0.001***
Body mass index (1 Kg/m ²)	1.13	1.06 – 1.20	<0.001***

Note: * $P<0.05$, ** $P<0.01$, *** $P<0.001$

Discussion

The present study was aimed to investigate two missense mutations of PECAM1 gene including Val125Leu and Gly670Arg polymorphisms in patients with and without atherosclerosis. In spite the change in amino acid in such an adhesion molecule, no significant association was found for Val125Leu polymorphism ($0.05<P<0.2$) and there was no association between Gly670Arg polymorphism and risk of atherosclerosis ($P>0.8$). These lacks of associations along with established HWE indicated lack of role for the polymorphisms in atherosclerosis among the studied ethnicities. According to the borderline effect of Leu/Leu genotype in crude models, an adjusted model was performed in which Leu/Leu genotype was a significant risk factor for susceptibility to atherosclerosis.

To date, some polymorphisms of PECAM1 have been studied and summarized as meta-analyses. Xia *et al.*, in a meta-analysis studied Val125Leu polymorphism in 3696 cases and 3940 controls. No significant pooled association was found in this meta-analysis. There was no Iranian study among the individual studies (26). Our study investigated this polymorphism in an Iranian population and there was a trend of significant association toward increase in risk of atherosclerosis for Leu allele and Leu/Leu genotype in comparison to Val allele and Val/Val genotype, respectively ($0.05<P<0.1$).

In a meta-analysis conducted by Sahebkar *et al.*, for outcome myocardial infarction (MI), other than Val125Leu polymorphism, the two other polymorphisms Gly670Arg and Ser563Asn were studied. Briefly, Gly/Gly was associated with reduced risk of MI (27). In our study, this polymorphism did not have an association with atherosclerosis. Another polymorphism of PECAM1 (rs1867624) was studied in a meta-analysis. No significant pooled association was found between this polymorphism and risk of MI or CAD. However, this

polymorphism was associated with lipid profile (28).

Our findings demonstrated that atherosclerosis disorder was associated with all atherosclerosis indices, and atherogenic values were higher in the atherosclerosis disorder group than in the control group. AC and TYG were worthy prediction biomarkers that may be beneficial in predicting atherosclerosis disease based on the ROC curve, and high levels of AIP were associated with an increased risk of atherosclerosis. All of these variables, according to previous studies, are significant risk factors for cardiovascular disease (29,30). according to our research, diabetes, is a strong risk factor for cardiovascular events. The findings are comparable to those of Fu *et al.*, (31). Diabetes patients have two to four times higher risk of cardiovascular events than non-diabetic people, based on this study.

The strength of this study was to investigate these functional polymorphisms in an Iranian population. However, the limitation of this study was that sampling was for another study in which there were some limitations.

Susceptibility to atherosclerosis (CAD) was not significantly associated with Val125Leu and Gly670Arg polymorphisms of *PECAM1* gene among the patients with acute chest pain in Lur population of Iran. Such functional polymorphisms of adhesion molecules may have other roles in pathogenesis of atherosclerosis. However, besides AC and some demographic variables, genotype Leu/Leu showed a harmful association.

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Ethical Considerations

The samples had been taken by approval of the ethics committee of Lorestan University of Medical Sciences

with registration number IR.LUMS.REC.1396.314.

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