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Study of the participation of *Lipoprotein Lipase* gene polymorphism in coronary artery disease

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The study designed to assess the association of *Lipoprotein Lipase* gene polymorphism with CAD. The study consists of two groups, the first group includes 93 patients with CAD and the second group includes 81 subjects with no sign, no symptom and no history of CAD. Genotyping of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was done by PCR-RFLP. The results of present study revealed study show that TT and TG genotypes of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was found to be non-significantly increase the risk of CAD with respect to those of the GG genotypes of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] respectively. Moreover, the outcomes of the study show that a significant increase of serum triglycerides concentration in the TT genotype of *LPL* gene polymorphism [HindIII T↔G (SNP)] when compared with GG genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] in patients with CAD. This study conclude that the *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was not associated with CAD and the TT genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] associated with increase triglycerides in patients with CAD.

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Introduction

Coronary artery disease (CAD) is the greatest popular form of cardiac disease; it represents the most popular reason of death in several countries [1,2]. It happens when the coronary arteries which afford the muscle of heart by blood (oxygen) are narrowed or obstructed through deposits of plaque. This plaque deposits decrease the flow of blood through coronary arteries and the heart muscle becomes starve and causes chest pain. The heart attack occurs after a complete blockage of blood flow. The causes of CAD is known to be a multifactorial, having both genetic and environmental contributes disease [3,4].

The concentration of serum lipids are influenced by both lifestyle factors (such as diet, physical activity, obesity) and genetic factors [5]. Any of these factors cause defect in lipid metabolism that leads to atherosclerosis. Atherosclerosis is the essential cause of CAD, which is mainly categorized by excessive deposition of lipid in the endothelium of the vascular tree walls. Individuals with abnormal metabolism of lipid and lipoprotein, including the increase in cholesterol, triglyceride, and LDL-Cholesterol levels besides decreased levels of HDL-Cholesterol, are more inclined to the development of CAD [6]. Lipoprotein lipase (LPL) enzyme plays an important role in the lipoprotein metabolism. It catalyzes the hydrolysis of triglycerides in chylomicrons and VLDL. Because of its essential role in lipoprotein metabolism, LPL is possibly of

high function in the atherosclerosis development [7,8,9].

The main actions of this enzyme are the plasma triglycerides hydrolysis in chylomicrons and VLDL lipoproteins into free fatty acids and glycerol, converts VLDL to LDL, and enhances lipoprotein-receptor interactions [10]. The fatty acids that resulted from triglyceride hydrolysis are esterified and stored in adipose and muscle tissues [11]. The LPL also plays a vital role in the regulation of HDL-Cholesterol levels because the hydrolysis of triglyceride rich lipoproteins contributes to HDL-Cholesterol metabolism. A decrease in LPL activity can effect on plasma lipid concentrations, i.e. it causes either insulated hypertriglyceridemia or in association with hypercholesterolemia [10,12].

The *LPL* gene is situated on chromosome 8p22, straddling about 30 kilobases. It contains ten exons separated by nine introns and this gene encodes a 475-amino acid [13,14]. The genetic studies have revealed nearly one hundred mutations and single nucleotide polymorphisms (SNP) in *LPL* gene and indicate that the variations in this protein can impact on the development of atherosclerotic plaque [15]. This study is proposed to assess the association of *LPL* Gene polymorphism [*HindIII* T↔G (SNP)] with CAD.

Materials and methods

The study includes two groups, the first group consists of 93 patients with CAD, whose ages range between 41 and 74 years (mean age 53.9±7.59), they were diagnosed by cardiologist and chosen from cardiology center in Babylon Province. The second group includes 81 subjects, whose ages range between 35 and 69 years (mean age 51.62±8.32), they were selected with no sign, no symptom and no history of CAD.

From all subjects, 5 mL of blood was withdrawn by vein puncture and then separated into two parts, the first part of blood (2 mL) was collected in EDTA containing tube and it stored at -70 until used for gene analysis i.e. *LPL* gene polymorphism [*HindIII* T↔G (SNP)]. The second part of blood (3 mL) was collected in the plain tubes; sera were obtained and stored at -20˚C until used for estimating lipid profile concentrations.

Genotyping of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was done by PCR-RFLP (16). The DNA were extracted (from frozen blood) by using genomic DNA mini kit (Favorgen). The PCR technique were applied for DNA amplification and this technique done by using specific primers [the forward primer is (5'GATGTCTACCTGGATAATCAAAG'3) and the reverse primer is (5'CTTCAGCTAGACATTGCTAGTGT'3)]. The reaction of PCR was done by using Maxim PCR Pre-Mix Kit (20 µL) which containing i-Taq DNA Polymerase, mixture of dNTP, reaction buffer, and so in one tube for one PCR reaction. For each tube, we added 2 µL of genomic template DNA, 1 µL (10 pmoles/µL) of both forward and reverse primers, and then complete the volume to 20 µL by adding 16 µL of nuclease free water. The PCR thermocycler program which gave the best results of amplification include, initial denaturation (at 94° C for 5 min), then thirty cycles include, denaturation (at 94° C for 1 min), annealing (at 55° C for 1 min), extension (at 72° C for 1 min), and then, a final extension (at 72° C for 7 min). The products of PCR (350 bp) were analyzed on agarose gel (2%) electrophoresis.

The PCR products were digested with *HindIII* restriction enzyme (Biolabs, New England). In *LPL* gene polymorphism [*HindIII* T↔G (SNP)] comprises the replacement of a thymine (T) with a guanine (G) base at position +495 in intron 8 and the cleavage of *HindIII* restriction enzyme abolished by converting the recognition sequence form AAGCTT into AAGCGT, i.e., it cleaves T, not G. The RFLP program which gave the best results include 10 µL of PCR product, 1 µL (10 U) of *HindIII* restriction enzyme, 5 µL of buffer and complete the volume 50 µL of nuclease free water and incubated overnight at 37 °C. The

digestion product of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] include three genotypes, the non-protective genotype (TT) is digested into two fragments (140 and 210 bp) whereas the homozygous protective genotype (GG) remains uncut (350 bp) while the heterozygous genotype (TG) contained three fragments (140, 210 and 350 bp). The digested product was detected by agarose gels (2%) electrophoresis.

Serum total cholesterol, HDL-Cholesterol, and triglycerides concentrations are determined by spectrophotometric kit. Serum LDL-Cholesterol concentration is measured by Friedewald equation [17]. The SPSS was used for statistical analysis. The P-value less than 0.05 was considered as a statistically significant.

Results

The general characteristics of patients with CAD and controls groups are indicated in Table 1. The result of lipid profile measurements show a significant increase ($p < 0.001$) in total cholesterol, triglycerides, the LDL-Cholesterol levels and a significant decrease in HDL-Cholesterol level ($p < 0.001$) in patients with CAD when compared to those of the control group are demonstrated in Table 1.

The genotypes distribution and frequency of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] are depicted in Table (1-3) and Figure 1. The results analysis indicate that the *LPL* gene polymorphism [*HindIII* T↔G (SNP)] genotype frequencies of TT, TG and GG were 54.8%, 35.5%, and 9.7% in patients with CAD and 48.1% 38.3%, and 13.6% in controls group, respectively. The TT and TG genotypes of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was found to be non-significantly increase (OR=1.598, CI 95% 0.603-4.235, P=0.345), (OR=1.301, CI 95% 0.474-3.565, P=0.608) the risk of CAD with respect to those of the GG genotypes of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] which are revealed respectively in Table 4.

The allele distribution and frequency of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] are illustrated in Table 3 and Figure 1. The allele frequencies of T and G in *LPL* gene polymorphism [*HindIII* T↔G (SNP)] were found to be 72.6% and 27.4% in patients with CAD and 67.3% and 32.7% in the controls group, respectively. The minor allele frequencies (G) of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] in patients with CAD and control groups were found to be 27.4% and 32.7%, respectively. It was non-significantly change ($P > 0.05$) in patients with CAD and control groups as mentioned in Tables 1-4.

The outcomes of the current study indicated that a significant increase ($P < 0.05$) of serum triglycerides concentration is obvious in the TT genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] when compared with GG genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] in patients with CAD, while there are non-significant variations of other serum lipid types among all genotypes of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] in patients with CAD demonstrated in Table 5.

TABLE 1 General characteristics of the patients with CAD and controls groups

Characteristic			Controls	Patients
Number			81	93
Sex (Male/Female)		No.	49/32	67/26
		%	60.49/39.51	72.04/27.96
Diabetes Mellitus	**Diabetic**	No.	11	59
		%	13.58%	63.44%
	Non-diabetic	No.	70	34
		%	86.42%	36.56%

Hypertension	**Hypertensive**	No.	19	55
		%	23.45%	59.14%
	Normotensive	No.	62	38
		%	76.55%	40.86%
Smoking	**Smoker**	No.	17	44
		%	20.99%	47.31%
	Non-smoker	No.	64	49
		%	79.01%	52.69%

TABLE 2 Serum lipid profiles levels of the patients with CAD and controls groups

Parameter	Group	Mean ± SD	P-value
Total Cholesterol (mg/dL)	Controls	184.34 ± 24.72	P < 0.001
	Patients	220.61 ± 27.00	
Triglycerides (mg/dL)	Controls	128.23 ± 39.40	P < 0.001
	Patients	165.69 ± 54.76	
HDL-Cholesterol (mg/dL)	Controls	55.28 ± 7.96	P < 0.001
	Patients	42.08 ± 10.84	
LDL-Cholesterol (mg/dL)	Controls	107.55 ± 28.25	P < 0.001
	Patients	148.88 ± 34.97	

TABLE 3 Genotypes and Alleles distribution of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] in patients with CAD and the controls groups

			Controls	**Patients**
Genotype	**TT**	**No.**	39	51
	TG	**No.**	31	33
	GG	**No.**	11	9
	Total	**No.**	81	93
			Controls	**Patients**
Allele	**T**	**No.**	109	135
	G	**No.**	53	51
	Total	**No.**	162	186

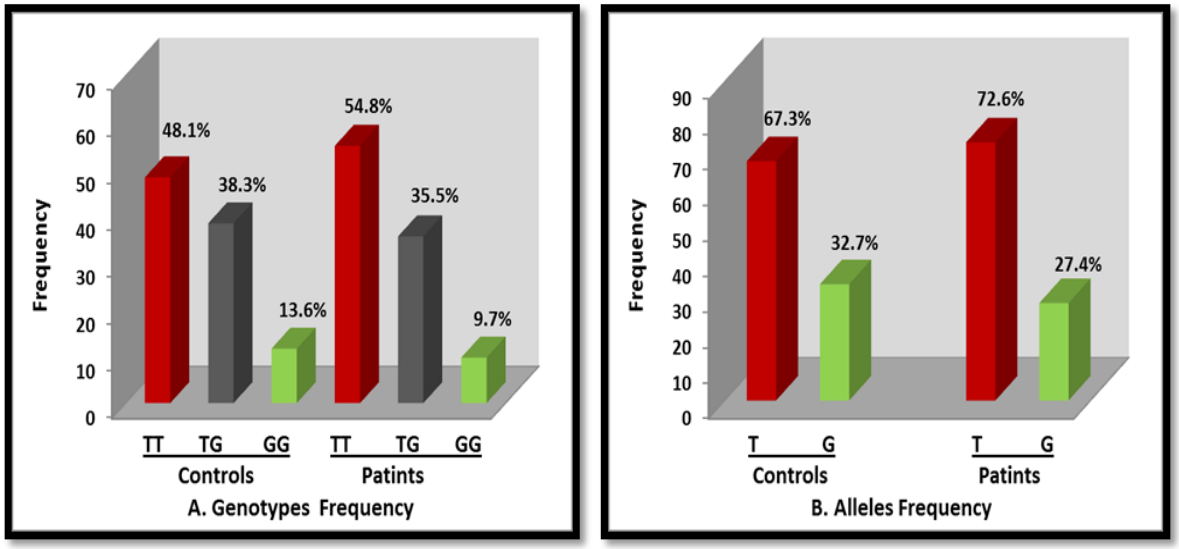

FIGURE 1 Genotypes (A) and alleles frequency (B) of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] in patients with CAD and the controls groups

TABLE 4 Odd ratio (OR), confidence interval (CI) 95% and P-value of genotypes and alleles of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] in patients with CAD and the controls groups

	OR	95% CI	P-value
TT vs. GG	1.598	0.603-4.235	0.345
TG vs. GG	1.301	0.474-3.565	0.608
T vs. G	1.287	0.812-2.038	0.282

TABLE 5 Genotypes correlation of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] with serum lipid profiles levels in patients with CAD group

Parameter	Genotype	Mean ± SD
Total Cholesterol (mg/dL)	**TT**	223.21 ± 28.21
	TG	216.61 ± 25.24
	GG	219.99 ± 26.43
Triglycerides (mg/dL)	**TT**	171.62 ± 59.89 (*)
	TG	162.32 ± 44.65
	GG	152.71 ± 53.16
HDL-Cholesterol (mg/dL)	**TT**	41.90 ± 11.37
	TG	41.73 ± 9.83
	GG	43.65 ± 10.58
LDL-Cholesterol (mg/dL)	**TT**	147.99 ± 33.32
	TG	149.07 ± 35.27
	GG	148.74 ± 34.76

(*) Means significant value ($P < 0.05$) when compared with GG genotype group
(**) Means significant value ($P < 0.05$) when compared with TG genotype group

Discussion

The LPL enzyme plays a central role in the lipoproteins metabolism; it hydrolyzes triglycerides in VLDL and chylomicrons into free fatty acids and the glycerol molecule. The abnormal enzyme activity is associated with

hyperlipidemia and atherosclerosis, and consequently can increase the CAD risk [18].

Genetic variations (polymorphisms or mutations) in the *LPL* gene could influence lipid metabolism and transport that consequently modulate an individual's susceptibility to atherosclerosis [19]. Numerous studies have stated that the *LPL* gene variants directly influence on lipid and lipoprotein metabolism, and then on the risk of CAD [20,21,22]. Increased plasma LPL activity could alter lipid traits, such as decreasing triglycerides and increasing HDL levels, generating a profile associated with protection against atherosclerosis, while the decreased plasma LPL activity has been recognized to play an opposite role [23,24].

There are several polymorphisms in the coding and non-coding regions of *LPL* gene which alter the LPL activity and change serum lipid concentrations and then the CAD risk [25,26,27]. The *HindIII* (rs320) polymorphism is one of the most common polymorphisms in *LPL* gene which includes replacement of a thymine (T) with a guanine (G) in intron 8 at position +495. Allele T (presence of cutting site) is associated with the decreased LPL activity, while allele G (absence of cutting site) is associated with the increased LPL activity [28,29,30].

This study evaluates the association of *lipoprotein lipase* gene *HindIII* polymorphism with CAD; previous studies are controversial about *HindIII* polymorphism of *LPL* gene [31]. Some studies found an association between TT genotype (T allele) and increase in CAD severity [20], while other studies reported that TT genotype (T allele) was associated with the decreased severity [21,22].

The results of present study revealed that TT and TG genotypes of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was found to be non-significantly increase the CAD risk with respect to those of the GG genotypes of *LPL* gene polymorphism [*HindIII* T↔G (SNP)], also the alleles of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was non-significantly change in patients with CAD and control groups. i.e., the *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was not associated with CAD and this result was in consistence with the results of Zeinab A., *et al.* [32], Mahdieh I., *et al.* [33], and Mohamed S., *et al.* [34] studies in which the *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was not associated with CAD. Conversely this results differed from those described by Amer A., *et al.* [35] study who did find an association between the *LPL* gene polymorphism [*HindIII* T↔G (SNP)] and CAD.

In this study, serum triglycerides concentration were significantly increase in the TT genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] when compared with GG genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] in patients with CAD, this result in compatible with the results of Mahdieh I., *et al.* [33] study in which the TT genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] is associated with high levels of triglycerides. This increase of serum triglycerides concentration in the TT genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] resulted from the decreasing the activity of LPL enzyme i.e., decrease the hydrolysis of triglycerides in VLDL and chylomicrons which lead to hyperlipidemia and atherosclerosis, and therefore can increase the CAD risk.

This study concludes that the *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was not associated with CAD and the TT genotype of *LPL* gene polymorphism [*HindIII* T↔G (SNP)] was associated with increasing triglycerides in patients with CAD.

Author contributions

All the authors had similar share in design, conducting, analyzing, writing and revising.

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