Paraoxonase-1 Levels and Q192R (rs662) Gene Polymorphism as Risk Factors for Coronary Artery Disease in Patients from Basra-Iraq

Wisal A. Salem[1*], Dawood S. Ali[2] and Hamid J. Abbas[3]

[1]Department of Basic Sciences, College of Dentistry, University of Basrah, Basrah, Iraq
[2]Department of Chemistry, College of Science, University of Basrah, Basrah, Iraq
[3]Al-Faiha'a Teaching Hospital, Al-Zehra'a Medical College, University of Basrah, Basrah, Iraq.

[*]**Corresponding author E-mail:** wisal.salem@uobasrah.edu.iq

https://doi.org/10.29072/basjs.20260107

ARTICLE INFO

Received: 18 January 2026

Accepted: 19 April 2026

Published: 30 April 2026

Keywords:

Paraoxonase-1 ,Q192R polymorphism (rs662); Coronary artery disease; Genetic susceptibility; Oxidative stress

ABSTRACT

Paraoxonase-1 is an antioxidant enzyme within the high density lipoprotein (HDL) that is important in avoiding low-density lipoprotein (LDL) oxidation and lowering the risk of atherosclerosis. The PON1 gene is highly polymorphic, which has a profound effect on the activity of the enzyme as well as the levels of its presence in the bloodstream and its predisposition toward cardiovascular diseases. The purpose of the case-control study was to determine the relationship between the PON1 Q192R (rs662) single nucleotide polymorphism (SNP) and coronary artery disease (CAD) among Iraqi Basra population. The number of patients with CAD was 129 and controls were 66. The technique of polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) was used to perform genotyping. The findings showed a strong difference between the allele distribution in patients and controls ($P < 0.05$). The associations did not exceed the level of statistical significance, whereas odds ratios were higher among patients. Nonetheless, binary logistic regression analysis found out that the carriers of the R allele (QR and RR genotypes) were more likely to develop CAD. These results indicate the significance of PON1 in cardiovascular protection and indicate that the Q192R polymorphism may have an impact on genetic susceptibility to CAD in the Iraqi population.

1. Introduction

Cardiovascular diseases (CV) are also a primary cause of mortality in the world as well as a massive threat to human health [1]. The lack of initial symptoms makes CV risk factors possibly aspirational bodies, and thus, the necessity to detect such CV risk factors at an early stage.s. Even though these early symptoms can be detected and followed with conventional laboratory tests (such as cholesterol screenings), their long-term prognosis and CV depend on lifestyle.. Genetic factors and events are distinct and hence they are good candidates to future CV risk markers [2]. Genetic indicators will be a better option in risk assessment because they remain constant and not affected by environmental factors. The diagnosis and prevention of CV can be assisted with genetic testing [3,4]. Atherosclerosis is a chronic inflammatory disease that is progressive and is characterized by the deposition of lipids in the arterial wall. Excessive oxidative stress is the major driver of this process which causes structural and functional changes in lipids and proteins, eventually leading to endothelial dysfunction and plaque formation [5]. Good cholesterol (high-density lipoprotein; HDL) levels in the blood are associated with an increased risk of atherosclerosis [6-8]. In turn, reactive species sequestered in the arterial wall may cause changes in LDL cholesterol, leading to the development of plaque [9]. Increased oxidative stress exacerbates the buildup of oxidized LDL. This concept (the lipid hypothesis) arose from previous research that claimed LDL is the primary cause of atherosclerosis [10].

Paraoxonase-1 (PON1) is an important antioxidant enzyme for HDL by removing cholesterol from phagocytic cells and inhibiting LDL particle oxidation [11]. This enzyme (PON1) is an HDL-dependent enzyme that protects cells and biomolecules from the damaging effects of LDL by reducing its synthesis in the liver and secreting it into the bloodstream. PON1 is supplied in serum and binds to a blood phosphate-binding protein before being released into the bloodstream. The enzyme primarily binds to HDL, but rarely to very low-density lipoprotein (VLDL) and chylomicrons [12, 13].

The present study aimed to evaluate the association between serum Paraoxonase-1 (PON1) levels and the Q192R (rs662) gene polymorphism with the risk of coronary artery disease (CAD) among patients from Basra, Iraq, while also investigating the role of PON1 as an oxidative stress-related biomarker and determining whether genetic variation in the PON1 gene contributes to increased susceptibility and progression of CAD in the studied population.

2. Materials and methods

2.1 Study Design

The study included 129 patients (47 females and 82 males), aged 45-75 years, diagnosed with acute coronary syndrome (ACS)/myocardial infarction (MI) based on coronary angiography, medical and clinical examinations, and laboratory findings at the Al-Sadder Teaching Hospital, Basra Specialized Centre for Cardiac Diseases and Surgery, and Al-Fayhaa Teaching Hospital. Data were compared with those apparently healthy individuals matched for age and sex. The control group did not have any known disorder. It is a condition characterized by severe chest pain and changes in electrocardiogram that is suggestive of myocardial ischemia and elevated cardiac biomarkers [14].

2.2 Laboratory Tests

The routine biochemical assays and immunoassay analysis were measured on automated biochemical analyzers Cobas Integra 400 Plus (Roche Diagnostics, Germany), with the Cobas e411 used to measure the biochemical parameters and the fasting blood glucose, blood urea, serum creatinine, total cholesterol, triglycerides, HDL, LDL These measures were calculated using standardized enzymatic and immunoassay methods with the result of enzymatic reactions being the quantification of glucose, lipids, and renal functional markers, which is achieved by using specific substrate-enzyme interactions to produce a measurable colorimetric product, and hs-TnI was measured using electrochemiluminescence immunoassay (ECLIA), which involved the interaction of DNA was extracted using the mini-DNA extraction kit from Geneaid Company (Ver. 06.22.16). Polymerase chain reaction (PCR) was performed using the following primers [15]:

Primer	Sequence	Product Size(bp)
Forward	5′-TAT TGT TGC TG GGG ACC TGA G-3′	238bp
Reverse	5′-CCT GAG AAT CTG AGT AAA TCC ACT-3′	238bp

On 2% agarose [15]. PON1 genotypes were identified using the PCR and restriction fragment length polymorphism (PCR–RFLP) method, with the restriction enzyme AlwI (Bio Lab/USA). On a 2% (w/v) agarose gel, three banding pattern genotypes were characterized: QQ (238 bp), QR (238 bp, 175 bp, and 63 bp), and RR (175 bp and 63 bp).

2.3 Statistical analysis

Statistical analyses were performed using SPSS V26. Continuous variables were analyzed and expressed with means and standard deviations. An odds ratio (OR) with a 95% confidence interval (CI) was used to evaluate the relationship between the two groups, alleles, and genotypes. Classical analysis of PON1 gene data included the t-test. The genotype frequencies followed the Hardy-Weinberg equilibrium. Moreover, the DeLong method of area under the receiver operating characteristic (ROC) curves derived the best cut-off values of risk markers of acute coronary syndrome (ACS)/myocardial infarction (MI) in relation to the severity of ACS. Multivariable logistic regression was conducted to adjust for potential confounders of ACS.

3. Results & Discussion

Table 1 summarizes the baseline patient demographics and laboratory data. The results showed no difference between patients and controls for age, Body Mass Index, Fasting blood glucose, urea, creatinine, and HDL. There were significant differences for cholesterol, triglyceride, LDL, VLDL, troponin, and PON1 levels.

Table 1: Basic and biochemical characteristics of the study population

Variables	CAD Patients (N=129) Mean±SD	Controls (N=66) Mean±SD	P. Value
Age (Years)	56.42±8.11	54.62±7.73	0.638
BMI (Kg/m²)	27.98±4.22	26.98±3.28	0.096
Sex Females	46 (35.7%)	36 (54.5%)	0.011
Males	83 (64.3%)	30 (45.5%)	
FBS (mg/dl)	94.866±3.336	94.013±3.975	0.143
Urea (mg/dl)	33.927±6.880	33.722±5.279	0.481
Creatinine (mg/dl)	0.825±0.213	0.776±0.230	0.159
Cholesterol (mg/dl)	199.457±36.263	184.472±40.283	0.003
Triglyceride (mg/dl)	190.613±67.399	122.365±42.778	<0.001
HDL (mg/dl)	40.09±7.165	43.652±6.394	0.001
LDL (mg/dl)	122.545±35.997	71.875±27.818	<0.001
VLDL (mg/dl)	38.580±14.867	33.069±13.174	0.035
Troponin hs (pg/ml)	535.108±486.247	2.799±3.495	0.001
PON1 (nmol/L)	30.59±25.44	81.51±39.55	0.001

Biomarker performance was assessed using the (ROC) and Area Under the Curve (AUC). After troponin I hs (100%), PON1 (72.8) was the second-best predictor of coronary, as shown in Table 2 and Figure 1.

Table 2: Receiver-operating characteristic (ROC) and area under the curve (AUC) analyses of serum biomarkers for the diagnosis of CAD.

Variables	Area under the ROC curve (AUC)	p-value	Cut off	Sensitivity (%)	Specificity (%)	Efficacy
Troponin hs	1.0	0.0001	36.8	100	100	100
PON1	0.685	0.0001	34.9	69.8	78.8	72.8

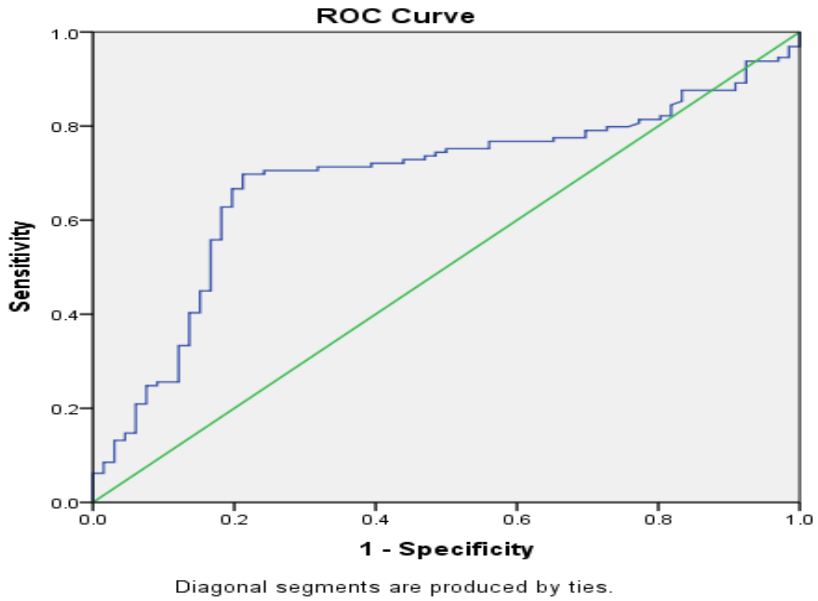

Figure 1: Receiver-operating characteristic (ROC) curve for PON1.

In Table 3, the logistic regression model was used to determine the risk of acute coronary disease (CAD) in patients. The odds ratios were troponin 1.678 and PON1 1.007, although the P values were not significant.

Table 3: Identification of risk of incident CAD by multivariable logistic regression analysis for all Patients

Variables	Regression coefficient	Standard error	Wald	P Vale.	Odds ratio	95% confidence Limits
Troponin I hs	0.518	11.922	0.002	0.965	1.678	0.0-34.0
PON1	0.007	18.410	0.000	1.000	1.007	0.0-10.0

The relationship between PON1 Q192R genetic polymorphism (SNP rs662) and patients' biochemical markers. Creatinine, triglycerides, HDL, LDL, Troponin I hs, VLDL, and PON1 revealed significant statistical differences between the three genotype groups; other measures showed no significant difference, as indicated in Table 4. The arterial wall damage and subsequent (LDL) oxidation initiate and promote atherosclerosis. In contrast, HDL can prevent

the progression of atherosclerosis by inhibiting LDL oxidation. Plasma HDL concentrations are inversely related to the incidence of CAD, indicating HDL's role as an atherosclerosis inhibitor [23, 24]. Lipid profile testing is important for screening in most standard populations and has been used in risk assessment systems (such as the Framingham Risk Scores) for over 30 years. Genetically predisposed dyslipidemia may be linked to CAD. Lower HDL levels are a significant cause of atherosclerotic plaque development [25, 26].

Paraoxonase enzyme activity is influenced by both genetic and environmental factors. The paraoxonase is distinguished by activity due to biological and environmental factors; because the Q192R polymorphism is the most influential in cardiovascular disorders, researchers have focused on variations that potentially modify the active site and its characteristics [27]. Polymorphism techniques are used to account for mutations and allele differences within distinct gene loci, as well as the variety among individuals of a species or populations. These methods can be used to identify SNPs in genes associated with disease and disorder [28, 29].

Table 4: Relationship between the PON1 Q192R SNP (rs662) of the gene and parameters studied in patient groups

Parameters	PON1 SNP genotypes						P. Value
	QQ (n=64) Mean ± SD		QR (n=54) Mean ± SD		RR (n=11) Mean ± SD		
FBS (mg/dl)	94.45	3.12	95.38	3.55	94.72	3.36	0.15
Urea (mg/dl)	33.95	7.79	33.73	5.75	34.73	6.81	0.74
Creatinine (mg/dl)	0.84	0.20	0.77	0.22	0.93	0.14	0.05
Cholesterol(mg/dl)	203.49	39.36	195.29	29.96	1926.36	45.88	0.56
Triglyceride (mg/dl)	199.76	72.44	175.21	61.10	212.94	54.14	0.08
HDL (mg/dl)	42.34	9.67	49.75	43.88	37.02	5.18	0.02
LDL (mg/dl)	123.76	36.89	115.81	33.64	148.48	31.87	0.01
VLDL (mg/dl)	39.64	14.21	39.52	15.19	27.74	13.86	0.008
Troponin hs (pg/ml)	513.03	458.07	380.51	342.50	1422 .45	290.54	0.001
PON1(nmol/L)	42.51	29.64	21.34	11.81	6.59	2.83	0.001

At position 192 of PON1, a single amino acid, glutamine (the Q allele) or arginine (the R allele), represents a crucial variation. As a result, glutamine provides more oxidative protection in the form of oxidation [16]. Position 192, which signals the PON1 active site, is known to stabilize PON1 on HDL particles. The polymorphism at Q192R, known as the allele rs662, influences substrate specificity. The Q192R rs662 paraoxonase-1 polymorphism is one of the most advanced markers for inhibiting coronary artery stenosis [17]. PON1 participates in both

oxidative stress defense and atherogenesis inhibition. Oxidized lipoprotein levels have been known to be increased for decades. PON1 is essential for antioxidant function because it protects LDL from lipid peroxidation, hence slowing atherosclerosis progression [17-19]. PON1 protein levels are at least largely determined by HDL plasma concentrations. HDL produces a hydrophobic environment that shields the amino-terminal region of PON1, which is required for substrate binding. PON1 activity may be influenced by a variety of variables, including lipoproteins and their metabolism, macromolecules (such as medicines), and lifestyle factors.

Paraoxonase-1 is a liver enzyme that enters the bloodstream and binds to HDL, but also in other CAD And also diabetes, hypercholesterolemia, and metabolic syndrome, all of which influence the etiology of chronic diseases, particularly in the elderly [20-22].

Table 5: Risk of CAD associated with PON1 genotype according to different models of inheritances

Genetic Model	Comparison	Patients (N=129)	Controls (N=66)	Unadjusted OR (95% CI)	P-Value	Age Adjusted OR (95% CI)	P-Value
Codominant	QQ (Reference)	64 (49.6%)	30 (45.5%)	1.00 (Reference)	—	1.00 (Reference)	—
	QR vs QQ	54 (41.9%)	32 (48.5%)	1.33 (0.39–4.52)	0.53	1.15 (0.33–3.98)	0.823
	RR vs QQ	11 (8.5%)	4 (6.1%)	1.29 (0.38–4.38)	0.773	1.44 (0.42–5.00)	0.562
Dominant Model	(QR + RR) vs QQ	65 (50.4%)	36 (54.5%)	0.90 (0.49–1.62)	0.65	1.23 (0.50–3.50)	0.70
Recessive Model	RR vs (QQ + QR)	11 vs 118	4 vs 62	1.44 (0.44–4.73)	0.777	1.34 (0.40–4.40)	0.62
Allelic Model	Q allele	70.50%	69.70%	Reference	—	—	—
	R allele	29.50%	30.30%	0.96 (0.61–1.52)	0.907	0.95 (0.60–1.50)	0.88

Figure 2, shows the electrophoretic pattern of the PON1 Q192R (rs662) polymorphism gene. Three genotypes were identified by three unique banding patterns: QQ (238 bp), QR (238 bp, 175 bp, and 63 bp), and RR (175 bp and 63 bp), which were seen on a 2% (w/v) agarose gel with ethidium bromide staining.

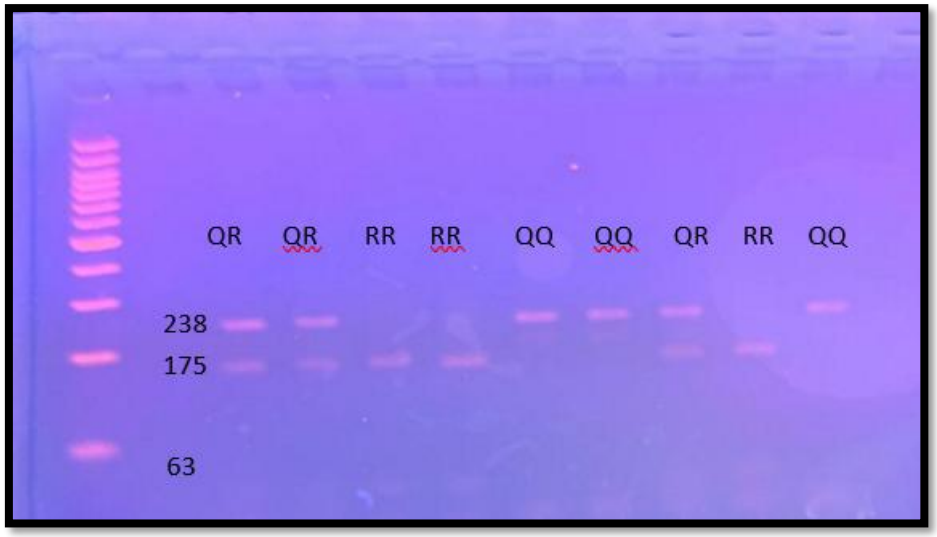

Figure 2: Electrophoretic pattern of the PON1 Q192R (rs662) polymorphism gene.

Restriction fragment length polymorphism polymerase chain reaction (RFLP-PCR) is a molecular technique for detecting changes within homologous DNA sequences known as polymorphisms [30]; RFLP-PCR indicated genetic variability in the PON1 gene, which was more common in CAD patients compared to healthy people. A study of CAD patients and age-matched controls in North India found that the Q192R variation in the PON1 gene was significantly associated with CAD [31]. Furthermore, as reported by S.T. Raza et al., [15], the RR homozygous genotype of the Q192R polymorphism in the PON1 gene was identified as having a notable relationship with CAD in Indian people from West India and is believed to be a risk phenotype when compared to other phenotypes. A separate study found that the RR genotype was associated with an increased frequency of cardiovascular events such as MI, as well as higher mortality and stroke rates [31]. A genetic risk factor of CAD in Mexican women was found by Ochoa-Martinez et al. Q192R polymorphism [32]. Moreover, the QQ192R polymorphism of the QR gene is heterozygous and strongly associated with the development of CAD among Mexican patients who have cardiovascular diseases [32, 33]. An investigation to Chinese population showed that the mutant allele RR of PON1Q192R gene is highly related with high risk of CAD in comparison with other genotypes [33]. A recent study of CAD patients in an Iranian society revealed that possession of the allele 192R increased the risk of CAD [34]. In this investigation, genetic and plasma PON1 levels were considered by the same researchers in their research analysis of a research cohort of 3,668 persons without acute coronary syndromes who underwent coronary angiography. Participants were monitored for three years for serious adverse cardiac events. The 192RR PON1 genotype significantly impacted plasma PON1 levels, although there was no correlation with the 3-year incidence of serious adverse

cardiac events [27]. In the Turkish population, we discovered that the PON1 RR genotype is associated with a higher frequency of CAD [35]. The current study confirmed the previous findings, which found that the 192RR PON1 polymorphism was linked to CAD in Iraq, implying that the RR homozygous genotype may increase the risk of CAD in the diseased population compared to healthy controls.

4.Conclusion

Paraoxonase-1 (PON1) 192RR genotype potentially is a more powerful predictor of atherogenesis and may be a cause of an increased risk of coronary artery disease in the Iraqi population. In addition, PON1 activity and genetic variability are closely related to the cardiovascular risk profile in community-based and clinical context, which emphasizes the possible role of the PON1 activity as a disease susceptibility biomarker.

Acknowledgements

The authors express gratitude to the participants of this study.

Ethical Approval

The Ethical Committee of the Department of Chemistry, College of Sciences, University of Basrah, and the Training and Human Development Centre at the Basrah Health Directorate approved the research protocol on January 11, 2023, number 836. This study was conducted according to the Declaration of Helsinki.

Conflict of interest

The authors assert that they possess no conflicts of interest.

Funding

The authors assert that this research did not receive any external support.

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مستويات الباراكسوناز-1 وتعدد أشكال الجين Q192R (rs662) كعوامل خطر للإصابة بمرض الشريان التاجي لدى المرضى العراقيين

وصال عبد الرحمن سالم[1]، داود سلمان علي[2*]، وحامد جدوع عباس[3]

[1] فرع العلوم الأساسية، كلية طب الأسنان، جامعة البصرة، البصرة، العراق
[2] قسم الكيمياء، كلية العلوم، جامعة البصرة، البصرة، العراق .
[3]مستشفى الفيحاء التعليمي، كلية الزهراء الطبية، جامعة البصرة، البصرة، العراق.

المستخلص:

يُعد إنزيم Paraoxonase-1 (PON1) أحد الإنزيمات المضادة للأكسدة المرتبطة بالبروتينات الدهنية عالية الكثافة (HDL)، حيث يلعب دورًا مهمًا في منع أكسدة البروتينات الدهنية منخفضة الكثافة (LDL) وتقليل خطر التصلب العصيدي. تؤثر تعددات الأشكال الجينية لجين PON1 بشكل كبير على نشاط الإنزيم ومستوياته في الدم، إضافةً إلى قابليته للإسهام في الإصابة بأمراض القلب والأوعية الدموية. هدفت هذه الدراسة من نوع الحالات والشواهد إلى تقييم العلاقة بين تعدد الأشكال الجيني Q192R (rs662) في جين PON1 وخطر الإصابة بمرض الشريان التاجي لدى المرضى العراقيين في البصرة. شملت الدراسة 129 مريضًا مصابًا بمرض الشريان التاجي و66 فردًا سليمًا كمجموعة ضابطة. تم تحديد الأنماط الجينية باستخدام تقنية تفاعل البوليميراز المتسلسل–تعدد أشكال طول القطع المقيدة .(PCR-RFLP) أظهرت النتائج وجود اختلاف معنوي في توزيع الأليلات بين المرضى والأصحاء .($P < 0.05$) وعلى الرغم من أن نسب الأرجحية كانت أعلى لدى المرضى، إلا أن هذه الفروق لم تصل إلى مستوى الدلالة الإحصائية. ومع ذلك، أظهر تحليل الانحدار اللوجستي الثنائي أن حاملي الأليل R الأنماط الجينية QR و RR لديهم احتمالية أعلى للإصابة بمرض الشريان التاجي. تشير هذه النتائج إلى الدور الحيوي لإنزيم PON1 في الحماية القلبية الوعائية، وتدعم أن تعدد الأشكال Q192R قد يسهم في زيادة القابلية الوراثية للإصابة بالمرض لدى السكان العراقيين.

الكلمات المفتاحية: باراوكسوناز 1، تعدد أشكال جين PON1 Q192R.