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Research Article

# Association of Interleukin-32 with Progress of Coronary Artery Disease: A Case-Control Study

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## Abstract

**Background:** Coronary artery disease (CAD) is a chronic inflammatory disease usually caused by atherosclerosis. Interleukin-32 (IL-32) is a recently identified inflammatory cytokine associated with an increased risk of cardiovascular diseases in inflammatory conditions. The aim of the study was to evaluate the role of IL-32 in patients with CAD and its relationship with their disease progression.
**Methods:** The present study was a case-control study that included a control group of 50 healthy individuals and 50 CAD patients. Serum was drawn from all participants for the detection of IL32 serum level by the Enzyme-linked immunosorbent assay (ELISA) method.
**Results:** Regarding the IL32 level, there were statistical differences (P =0.004) between the groups under study. In patients, compared to controls, there were increases in the IL-32 serum level. Additionally, patients with CAD showed a non-significant relationship regarding biomarkers, except erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) (p=0.002 and 0.000, respectively), which elevated in CAD patients compared to the healthy control group.
**Conclusions:** The result of this study has shown an elevation in IL32 serum levels in patients with CAD compared to healthy individuals. Therefore, this interleukin may be related to the progression of the disease.
**Keywords:** Coronary Artery Disease, IL32, ELISA

## Introduction

Coronary Artery Disease (CAD) is one of the most lethal conditions, which causes gradual death as a result of cholesterol buildup in the heart's arteries. Plaque is formed in the arteries as a result of cholesterol deposition, which affects the heart muscle (atherosclerosis). Fatty substances, waste materials, cholesterol, calcium, and the hormone fibrin that causes clotting make up plaque [1]. The CAD is among the most prevalent heart conditions, which includes both unstable and stable angina, myocardial infarction, as well as unexpected heart death [2]. It caused death to approximately 17.9 million deaths globally in 2019, accounting for 32% of all deaths. Moreover, a major proportion of these deaths took place in developing countries [3]. CAD is classified into two broad categories: non-modifiable and modifiable risk factors. Non-modifiable risk factors include age, gender, ethnicity, and family history of CAD. Modifiable risk factors include hypertension, hyperlipidemia, diabetes, stress, poor nutrition, smoking, obesity, and a sedentary lifestyle [4]. Modifiable and non-modifiable risk variables were discovered in order to lessen the substantial healthcare cost associated with CAD and the ensuing economic and disease-related impact [5]. Over the past 40 years, CAD death rates in Western nations have dramatically dropped due to the discovery of risk factors and advancements in medical technology [6].

Interleukin-32 (IL-32), a pro-inflammatory cytokine, was initially discovered in 1992, encoded as 27-KD protein called NK4 by a novel human gene, which is expressed in human T cells and activated natural killer (NK) cells [7]. It triggers the production of other cytokines that are involved in inflammation, such as TNF-α, IL-6, and IL-1β [8]. IL32 is constitutively produced by peripheral blood mononuclear (PBMC), epithelial, and endothelial cells. Additionally, it is found in a variety of nonimmune cells, such as fibroblasts, hepatocytes, mesenchymal stromal cells, epithelial cells, and

endothelial cells [9]. Many forms of IL-32 are identified in most mammals, including IL-32α, IL-32β, IL-32γ, IL-32δ, IL-32ε, IL-32ζ, IL-32η, IL-32θ, and IL-32s. It is extensively dispersed throughout the body, and both immune and non-immune cells exhibit its expression [10].

The IL-32 serum levels were elevated in patients with CAD, which supports its involvement in CAD-related inflammatory responses [11]. Moreover, recent reviews have confirmed IL-32's broader impact on vascular inflammation, suggesting its possibility as both a diagnostic marker and therapeutic target in cardiovascular diseases [12]. Malignant cells, such as melanoma cells, colon cancer cells, pancreatic, thyroid cancer tissues, and cell lines have IL-32 expression [13]. Tregs are significant generators of IL-32 in a variety of illnesses, according to single-cell sequence studies [14]. The production of IL-32 is triggered in response to viral infections such as HIV, influenza A, Epstein-Barr virus, human papillomavirus (HPV), and Herpes simplex virus, as well as bacterial infections brought on by Helicobacter pylori and Mycobacterium tuberculosis (MTB) [15]. Numerous autoimmune conditions, including rheumatoid arthritis and inflammatory bowel disorders, have been associated with abnormal IL-32 production. Additionally, a recent study revealed the role of IL-32 in the etiology of type 1 diabetes [16].

This study aims to investigate whether increased IL-32 serum levels might be used as a biomarker for diagnosing the progression of coronary artery disease.

## Materials and Methods

### Patients

This is a case-control study consisted of 100 participants (48 males, 52 females), who had separated into two groups: 50 patients with coronary artery disease, with age range from 40–70 years old attended to Shaheed Al Muhrab Center for Cardiac Catheterization and Surgery and Imam Al-Sadiq hospital in Babylon City in Iraq from October 2024 to January 2025. The second group included 50 healthy people.

**Inclusion criteria:** All patients with coronary artery disease were diagnosed by a cardiologist based on clinical symptoms and confirmed using other investigations, including electrocardiography (ECG), echocardiography, and coronary angiography [17].

**Exclusion criteria:** Patients who have pneumonia, cancer, any type of infection, or autoimmune diseases were excluded from the study.

### Sample collection

A 5 ml blood sample was drawn from each patient and the control group. EDTA tube used for the biomarkers test, including Erythrocyte Sedimentation Rate (ESR)(normal range=0-20 mm/hr) and complete blood count (CBC). Where gel tube is used for the detection of C-reactive protein (CRP) level and the determination of IL-32 serum level by the ELISA technique.

### Detection of IL-32 levels

Interleukin-32 serum levels were determination by sandwich-ELISA using ELISA kit (Sunlong Biotech, Chain, Catalogue number: SL0994Hu).

### Determination of Body mass index (BMI):

The following formula was used to calculate the participants' BMI:

BMI=weight (kg)/height2 (m)

The normal value for each category: BMI <18.5, normal weight, 18.5-24.9 underweight, 25-29.9 overweight, and 30-34.9 overweight. The classes of BMI were included: class I obesity, 35; class II obesity, 35–39; class III obesity, >40 [18].

### Ethical considerations

An ethical certificate was obtained from the relevant committees at the University of Karbala, No. 24-46, dated 29 September 2024. Approval was obtained from the Babylon health directorate to complete the study on No. 107, dated 29 December 2024. Verbal consent was obtained from all patients before sample collection. The researcher adhered to all ethical principles outlined in the 1964 Declaration of Helsinki or equivalent ethical standards.

### Statistical analysis

The statistical analysis for the current study was conducted utilizing IBM's SPSS software (Statistical Package for the Social Sciences, version 26; Chicago, Illinois, USA) and the Microsoft Excel 2010 program. Descriptive statistics have been done for all participants of each group. Data was analyzed for means, and the standard deviation was computed for the continuous variables, whereas frequency was used for computing the qualitative data. The means of the investigated parameters were compared between the two groups using a t-test. Chi-square test was applied to compare between percentages. Differences among groups were analyzed using one-way ANOVA analysis of variance. The results of all hypothesis tests with p-values <0.05 (two-sided) were considered to be statistically significant.

## Results

Figure 1 displays the distribution of study populations according to their ages. It highlights the age consistency of the study population, with both patients and controls being similarly distributed across all age groups. Figure 2 describes the means and standard deviation for BMI in patients and controls. The results showed that BMI was significantly higher (p=0.022) in CVD patients (23.08±3.19) compared to controls (22.44±2.17), while Figure 3 displays the distribution of patients and controls according to BMI categories. The percentage of patients and controls was 74% and 86%, respectively, within the normal category (18%) and overweight category (14%). 8% of patients were obese, while no control individuals were found in the obesity category.

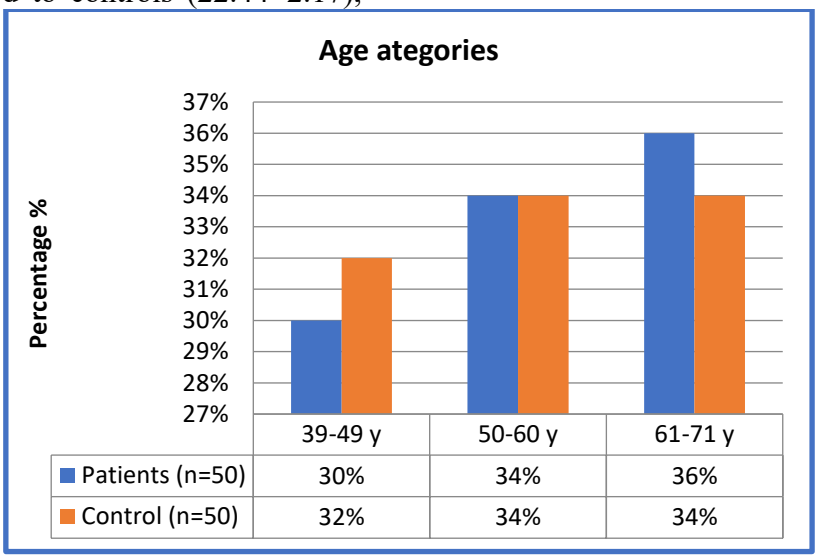


**Figure 1:** Age categories of study population

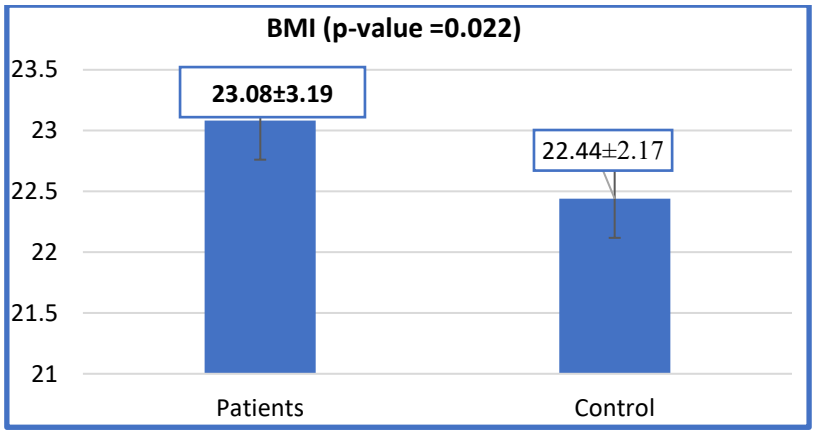


**Figure 2:** Body mass index (BMI) value in study population

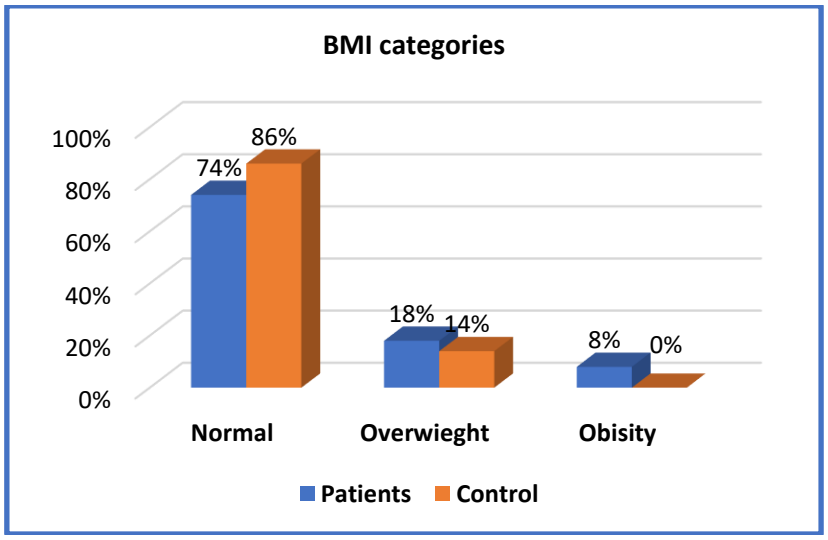


**Figure 3:** Percentage of body mass index categories in study population

Figure 4 shows the distribution of patients according to the nature of their diet into two groups: vegetarians and non-vegetarians, where 42% were vegetarians and 58% were non-vegetarians, without significant differences between these groups (p=0.6892). Table 1 showed the levels of IL-32 in both CVD patients and controls, where the result of statistical analysis revealed a significant (p=0.004) elevation in IL-32 levels in CVD patients compared with control individuals. Figure 5 illustrates the levels of some laboratory markers in CVD patients, where the mean of eosinophils was 0.128, neutrophils was 5.1478, monocytes was 0.5156, basophils was 0.0744, lymphocytes was 3.0062, CRP was 7.202, and ESR was 17.06.

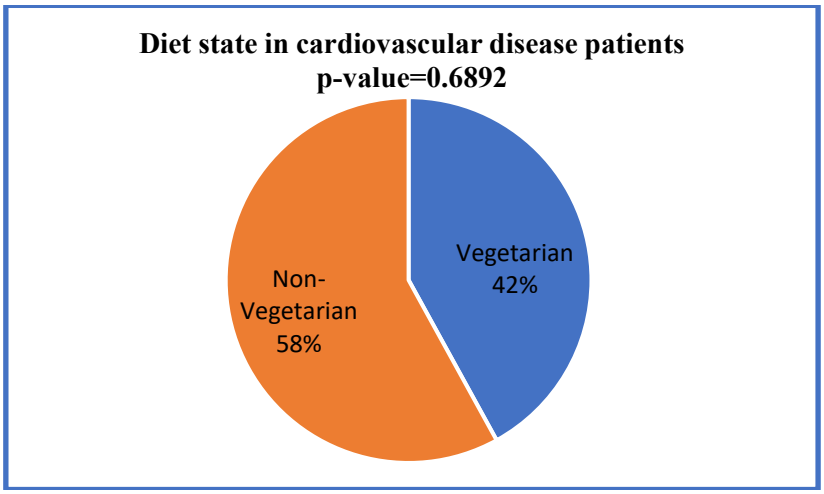


**Figure 4:** Distribution of diet state in cardiovascular disease patients

**Table 1:** Serum levels of IL-32 for study population

| Parameters | Study population Mean ± SD | | p-value |
|---|---|---|---|
| | **Patients (n=50)** | **Control (n=50)** | |
| IL-32 pg/ml | 23.232 ± 7.855 | 15.984 ± 5.168 | 0.004* |

*Significant difference at the 0.05 level by t-test.
SD: standard deviation

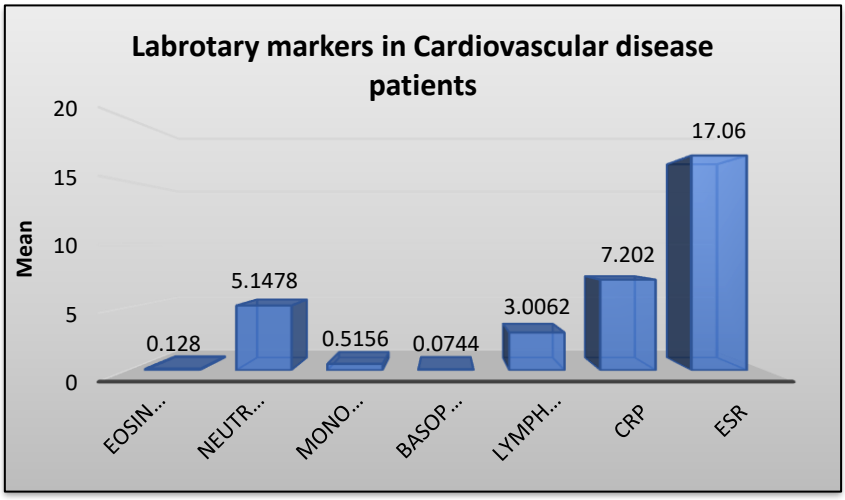


**Figure 5:** Laboratory markers in cardiovascular disease patients
CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate

## Discussion

The current study showed that the distribution of patients and controls according to the age categories was nearly equal, and this is because the selection of samples wasn't randomized. A study conducted by Livenson *et al.* (2020) mentioned that the prevalence of CAD among adults was higher, and the reason was due to an increase in risk factors such as hypertension, lipidemia, and diabetes mellitus [19].

According to the BMI (normal weight) in this study, the distribution of CAD was higher in patients compared to controls, and this may be attributed to the comorbidities risk factor of CAD, like hypercholesterolemia, hypertriglyceridemia, hypertension, and diabetes mellitus. Zhao *et al.* (2021) found an association between BMI and risk of CVD incidence [20]. While the percentage

distribution of the disease within patients and controls was higher in the normal weight category, the overweight category. A study conducted by Dikaiou *et al.* (2021) found that a higher estimation was in normal weight (22.5– < 25 kg/m2) and the lowest among the categories of those who are obese or extremely fat. Patients with a low-normal body mass index (BMI) of around 22 kg/m2 were the least at risk [21].

The study has shown a non-significant relationship within the patient group regarding the nature of their dietary state. A similar result was mentioned by Szczepańska *et al*. (2023), who found 42% of patients consumed vegetables several times in their weekly mails. However, 57% of them consumed both red and white meat [22]. Recently, Sapala *et al.* (2025) declared that unusual dietary patterns were apparent despite the fact that there was no elevated risk of malnutrition, especially with regard to inadequate consumption of fruits, vegetables, legumes, whole grains, nuts, dairy products, and seafood. An unbalanced dietary status might increase the risk of body weight, hypertension, and body fats, which increases the risk of cardiovascular disease [23].

There was a significant relationship within the study groups regarding IL-32 serum level, which proved an elevation in the serum level of the patients compared to healthy controls. Tomasi *et al.* (2023) demonstrated this finding by showing an elevation between circulating IL32 levels and by revealing that circulating IL32 levels with associated with impaired blood pressure regulation in individuals at risk of cardiovascular disease [24]. Mohammad-Rezaei *et al.* (2021) mentioned that IL-32 was 2.7 times higher in obstructive CAD compared to non-obstructive CAD, independently related to the presence of obstructive CAD, while HDL levels were not. Therefore, obstructive CAD was satisfactorily predicted by the blood level of this cytokine [25]. In addition, Kaymaz *et al.* (2025) found a noticeable rise in IL32 serum levels in Behçet's disease patients [26]. Such an increase might be connected to vascular involvement. Moreover, IL-32 has a role in other diseases. Choi *et al.* (2019) had revealed that serum IL-32 levels were elevated in patients with endometriosis [27]. Furthermore, Yao *et al.* (2019) indicated that there was a positive correlation between IL-32 mRNA expression and the prevalence of Graves' disease and thyroid function. IL-32 association with the pathophysiology of Graves' disease was demonstrated. Uncertainty remains regarding the precise mechanism of IL-32 in GD [28].

The current study showed a noticeable rise in some biological parameters' mean. This finding was in the same line with Matei *et al.* (2022), who found a significant elevation in ESR levels within CVD patients [29]. As ESR is not a disease-specific marker, increased values of this parameter may be observed in several conditions, such as inflammatory diseases, infections, or tumors. Also, Matei *et al.* (2022) observed an elevation in CRP levels as an indicator of early inflammation [29]. CRP is widely recognized for its function as a measure of chronic inflammation and in cardiovascular disorders due to its relationship with tissue fibrosis in a number of cardiovascular disorders, and elevated CRP levels are linked to the formation of atherosclerotic disorders. Moreover, Wang *et al.* (2023) reported a significant increase in neutrophil counts in CAD patients, which was agree with present study results [30].

The current study showed a non-significant relationship to the distribution of biological markers regarding age category. ESR and CRP were the only markers that had shown noticeable rises within the 60-70 age category. Dugani *et al.* (2021) mentioned that all the inflammatory biomarkers examined showed positive associations (by approximately 1.2 to 1.8-fold per SD) with incident CAD [31]. As the incidence rates of CAD increased with age and were approximately 10-fold higher for CAD onset at age 75 years or older vs. younger than 55 years, consistent with age being a substantial risk factor.

The BMI was associated with CAD in this study. Buschmann *et al.* (2020) reported that the BMI and the biomarker levels are not related to the systemic inflammation markers that are possibly responsible for the worse prognosis and high cardiovascular event rate [32].

The study revealed Non-significant differences in the distribution of inflammatory biomarkers regarding the diet in CAD patients. A study conducted by Ghanavati *et al.* (2021) observed a non-significant difference in the concentration of CRP within study groups according to the diet [33].

The current study finding was reported that patients with hypertension had no significant relationship with inflammatory markers. This result was in agreement with a study done by Bisaria *et al.* (2020) that had shown a non-significant statistical difference between CRP level and hypertension among the patient group [34]. While there was a significant increase in eosinophil levels. This result was contrary to what Gao *et al.* (2019) who reported a significant lowering in eosinophil levels. The results showed CAD patients exhibited lower

eosinophils than non-CAD patients. This result may be attributed to the extensive thrombus formation, which could have induced the decreased eosinophil count [35]. A study of Wang *et al.* (2023) had supported this idea by proven the same results [30]. In contrast, Tanaka *et al.* (2012) found a significant rise in peripheral blood eosinophil counts in individuals with unstable angina pectoris as compared to control subjects because eosinophils produce and release bioactive mediators like leukotriene C4, which is a strong vasoactive and smooth muscle contraction stimulator [36].

## Conclusions

The result of this study has shown an elevation in IL32 serum levels in patients with CAD. Therefore, this interleukin may be related to the progression of the disease. Further research with large sample sizes is required to demonstrate the role of this interleukin in patients with CAD.

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