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ASSESSMENT OF MIR-126 EXPRESSION AND SOME BIOCHEMICAL PARAMETERS IN PATIENTS WITH CORONARY ARTERY DISEASE

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ABSTRACT

Background: Coronary artery disease (CAD) accounts for major morbidity and mortality worldwide. Objectives: This study was designed to investigate the expression level of miR-126 in the patients with CAD as well as to verify chosen inflammatory and biochemical parameters related to the disease. The study also examined the association of miR-126 expression with age, gender and BMI to evaluate its possible utility as CAD biomarker.

Materials and Methods: A case–control study was performed at the Azadi Teaching Hospital, Kirkuk city, Iraq during the period from July to October, 2025. The study included 163 participants, including 83 patients with CAD and 80 age- and sexmatched, healthy controls. The biochemical and inflammatory cytokines were measured by ELISA and the expression of miR-126 gene was assessed by RT-PCR after extraction of RNA from blood samples.

Results: The study demonstrated that 37.3% of CAD patients were aged ≥ 60 years, followed by 26.5% in the 50–59 years group, 20.5% in the 40–49 years group, and 3.6% aged < 40 years. Males represented 77.1% of the patients, while females accounted for 22.9%. Regarding BMI, 56.6% of patients were obese, 37.3% were overweight, and only 6.0% had a normal BMI. Inflammatory cytokines were significantly elevated in CAD patients compared with controls, where IL-1 α increased from 2.84 ± 0.51 to 53.21 ± 7.45 pg/mL, TNF- α from 37.25 ± 4.83 to 109.37 ± 8.15 pg/mL, IL-6 from 7.63 ± 2.26 to 36.46 ± 5.04 pg/mL, and IL-17 from 11.39 ± 2.47 to 68.11 ± 3.61 pg/mL ($P = 0.001$ for all). In addition, miR-126 expression was significantly upregulated in CAD patients with a mean fold change of 2.58 compared to 0.96 in controls. Pearson correlation analysis showed significant positive associations between miR-126 and TNF- α ($r = 0.68$), IL-17 ($r = 0.63$), IL-1 α ($r = 0.61$), and IL-6 ($r = 0.57$), as well as with age ($r = 0.42$) and BMI ($r = 0.39$), while no significant correlation was observed with gender ($r = 0.18$, $P = 0.091$).

Conclusions: The study demonstrates strong associations between CAD and systemic inflammation and metabolic risk factors, and suggests that miR-126 may represent a new circulating biomarker reflecting disease severity and inflammatory state.

Keywords: Coronary artery disease, MiR-126, Inflammatory cytokines, Atherosclerosis, BMI.

1 INTRODUCTION

Coronary artery disease (CAD) is a common cardiovascular disease; it is one of the leading causes of morbidity and mortality across the globe. It is most commonly characterized by a process of progressive atherosclerosis of a coronary artery resulting from lipid accumulation, endothelial dysfunction, and chronic inflammation and plaque formation that can ultimately lead to myocardial ischemia and infarction (1,2). Despite rapid improvements in diagnosis and treatment, CAD remains an enormous global health burden driven by its complex multifactorial origins and association with established modifiable risk factors (3). Background Conventional risk factors including hypertension, diabetes mellitus, dyslipidemia, smoking, obesity, and aging have been identified as the main determinants of the development and progression of CAD (4,5). There is also growing evidence that inflammatory pathways and molecular mechanisms contribute to disease progression and plaque instability in atherosclerosis (6). MicroRNAs are a class of small, non-coding RNAs that regulate gene expression, and play crucial roles in cardiovascular diseases in recent years. For example, miR-126 is abundantly expressed in endothelial cells, is essential for the maintenance of vascular integrity, angiogenesis, and modulation of inflammatory responses (7,8). It has been shown that miR-126 is dysregulated in endothelial dysfunction and atherosclerotic progression, and thus may be used as a potential diagnostic and prognostic biomarker for coronary artery disease (9,10). Circulating miRNAs, such as miR-126, are stable in blood, associated with underlying pathological processes and may hence serve as novel and non-invasive cardiovascular disorder biomarkers. Use of miR-126 has been reported as an aberrantly expressed microRNA in individuals with CAD, indicating its potential clinical significance in diagnosis and disease monitoring (11,12). In addition, inflammatory cytokines, like IL-6 and TNF- α , and biochemical indicators, such as lipid profile and glucose levels are significantly correlated with the severity of CAD (13,14). However, these parameters are also modified by demographic factors (age, gender, and BMI) that affect cardiovascular risk and disease progression (15). Coronary artery disease is a major and growing public health problem in Iraq. According to a study within the Kurdistan area of Iraq, the prevalence of premature coronary artery disease among coronary angiography candidates was 30.9%, again pointing to a high burden of early-onset disease among the Iraqi population. This was substantially associated with modifiable risk factors including smoking, obesity, diabetes, and hyperlipidemia (16). These results also suggest that CAD in Iraq is not just common but also affecting younger patients which reflects the effects of lifestyle and metabolic diseases. On the other hand, local studies investigating miRNA expression in Iraqi CAD patients at the molecular level are scarce, and previous work included a study of numerous dysregulated miRs (17) suggesting altered levels of miR-126 involved in disease pathogenesis where elevated may have a potential role as a marker. Information on the potential additive role of miR-126 expression in combination with serum cytokines and biochemical and demographic variables in patients with coronary artery disease (CAD) is still limited, especially among the Iraqi population. so, this study was aimed to evaluate the expression of miR-126 and several biochemical and cytokine related parameters in coronary artery disease (CAD) patients and assess their associations with gender, age and body mass index.

2 MATERIALS AND METHODS

2.1 Study design and setting

This study is a case-control designed for the assessment of the expression of miR-126 and selected biochemical and inflammatory parameters in CAD patients. Design: A prospective study was carried out at Azadi Teaching Hospital in Kirkuk, Iraq from July to October 2025.

2.2 Study population

This study included 163 participants with 83 patients having coronary artery disease and 80 apparently healthy subjects who served as the control group. Diagnosis of CAD in the CAD patients was based on the results of clinical evaluation and other relevant cardiological examinations carried out on the patients who were hospitalized in the hospital.

2.3 Sample collection

Five mL of venous blood was drawn from each participant using sterile procedures. Sample Collection: Whole blood was allowed to clot at room temperature and was then centrifuged at 4000 rpm for 15 minutes to obtain serum. Serum was collected and fractionated into sterile tubes following centrifugation. All serum and TRIzol samples were subsequently placed at -20°C until further biochemical and molecular analysis.

2.4 Exclusion criteria

Patients with infections, autoimmune diseases, malignancy, severe liver or kidney disease, or planned surgery, and those receiving immunosuppressive/anti-inflammatory therapy were excluded from the study. To also ensure reliability in the data, pregnant and incomplete data were excluded.

2.5 Demographic Characteristics

We concluded that various demographic variables including age, sex and body mass index (BMI) play a key role in evaluating coronary artery disease (CAD). Increased risk of disease is associated with increased age and male gender, and CAD has also been linked to elevation in BMI, primarily due to the macrophage-mediated inflammatory and metabolic changes associated with obesity.

2.6 Lipid Profile Determination

Serum lipid profile was determined by standard enzymatic assays routinely performed in the hospital laboratory and included total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL). The TC, TG, and HDL assays were performed according to the manufacturer's guidelines using commercial kits provided by Abbott (Germany) and analyzed with a multi-channel Abbott Spectrum auto-analyzer (Abbott Laboratories, USA; C460276). Low-density lipoprotein (LDL) levels were indirectly calculated by use of Friedewald formula as follows: $LDL = TC - HDL - (TG/5)$. Serum triglycerides were used to estimate very low-density lipoprotein (VLDL) concentrations ($VLDL = TG/5$).

2.7 Cytokines

Serum levels of interleukin-1 α (IL-1 α), tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), and interleukin-17 (IL-17) were measured using ELISA kits supplied by SUNLONG Biotech (China) according to the manufacturer's instructions. 10 μ L of serum sample was added to each well of the microplate, followed by 40 μ L of sample diluent. The plate was then incubated for 30 minutes at room temperature. After incubation, wells were washed five times using wash buffer. Then, 50 μ L of HRP-conjugate reagent (RGP) was added to each well, followed by incubation and a second washing step. Subsequently, 50 μ L of substrate solutions A and B were added to each well (50 μ L each), and the plate was incubated for 15 minutes in the dark. Finally, 50 μ L of stop solution was added to terminate the reaction. Absorbance was measured at 450 nm using an ELISA reader (Biobase, China). Cytokine concentrations were calculated based on standard calibration curves.

2.8 Isolation of Total RNA and RT-PCR Analysis

According to (manufacturer's protocol, TRIzol™ Reagent (Promega, USA) was used to extract total RNA from serums. In short, 0.5ml of TRIzol™ reagent was added to 0.4ml of serum, and the mixture was homogenized. Subsequently, 0.2 mL of chloroform was added into each tube followed by incubation and centrifugation at 10,000 rpm for 10 minutes. The mixture was then centrifuged, yielding three phases: a lower organic phase, an interphase, and a clear upper aqueous phase containing RNA, which was taken out with great care. This was measured using the Quantus Fluorometer and quantified by measuring the cDNA yield concentration at 260/280 nm and RNA concentration at 260 nm by fluorescence (Figure 1). To quantify, 1 μ L of cDNA was combined with 199 μ L of diluted Quantifluor dye, incubated in the dark at room temperature for 5 minutes and then quantified for downstream applications. MiR-126 Expression Quantification Quantitative real-time PCR (qRT-PCR) for miR-126 expression was performed using the miScript SYBR Green PCR kit (Qiagen, Hilden, Germany). Specific primers for mature miRNA-126 were used: Forward: 5'-GGGGCATTATTACTTTTGG-3' Reverse: 5'-CATTATTACTTTTGGTACGCG-3'. Specific primers for U6 (housekeeping) were used: F: GACCTGCTCTGGTGGTCTTG, Reverse: R: GAACCACACTCTGGGACAGG-3'. Gene expression levels were normalized to a housekeeping gene and analyzed using the $2^{-\Delta\Delta Ct}$ method. The calculations were performed as follows:

$$\Delta Ct = Ct (\text{target gene}) - Ct (\text{housekeeping gene})$$

$$\Delta\Delta Ct = \Delta Ct (\text{patient or control}) - \text{mean } \Delta Ct (\text{control group})$$

$$\text{Relative expression (fold change)} = 2^{-\Delta\Delta Ct}.$$

2.9 Statistical analysis

Statistical analysis was performed using GraphPad Prism software (GraphPad Software, USA). Data were expressed as mean $\pm$ standard deviation (SD), and comparisons between groups were analyzed using Student's t-test. A p-value < 0.05 was considered statistically significant (18,19).

3 RESULTS

The age distribution of patients with coronary artery disease (CAD) is presented in Table 1. The highest proportion was observed in the age group ≥ 60 years, accounting for 37.3% (n=31) of patients. This was followed by the 50–59 years group with 26.5% (n=22), and the 40–49 years group with 20.5% (n=17). The lowest percentage was recorded in patients aged <40 years, comprising only 3.6% (n=3) of the total CAD cases.

Table 1: Gender distribution of patients with coronary artery disease (CAD)

Age group (years)	CAD patients (n=83)	
	No.	%
<40	3	3.6%
40–49	17	20.5%
50–59	22	26.5%
≥60	31	37.3%
Total	83	100.0

The gender distribution of patients with coronary artery disease (CAD) is presented in Table 2. Males constituted the majority of the study population, accounting for 77.1% (n=64), whereas females represented 22.9% (n=19).

Table 2: Gender distribution of patients with coronary artery disease (CAD)

Gender	CAD patients (n=83)	
	No.	%
Male	64	77.1%
Female	19	22.9%
Total	83	100.0

The distribution of body mass index (BMI) among patients with coronary artery disease (CAD) is presented in Table 3. The majority of patients were obese, representing 56.6% (n=47) of the study group. Overweight individuals accounted for 37.3% (n=31), while only 6.0% (n=5) of patients had a normal BMI.

Table 3: BMI distribution of patients with coronary artery disease (CAD)

BMI category (kg/m ²)	CAD patients (n=83)	
	No.	%
Normal (<25)	5	6.0%
Overweight (25–29.9)	31	37.3%
Obese (≥30)	47	56.6%
Total	83	100.0

Table 4 showed that the mean serum level of IL-1 α was significantly higher in CAD patients (53.21 $\pm$ 7.45 pg/mL) compared to the control group (2.84 $\pm$ 0.51 pg/mL) with a highly significant difference (P = 0.001). Similarly, the level of TNF- α was markedly elevated in CAD patients (109.37 $\pm$ 8.15 pg/mL) compared to controls (37.25 $\pm$ 4.83 pg/mL), showing a statistically significant difference (P = 0.001). In addition, the mean serum level of IL-6 was significantly increased in CAD patients (36.46 $\pm$ 5.04 pg/mL) compared to the control group (7.63 $\pm$ 2.26 pg/mL) (P = 0.001). the level of IL-17 was significantly higher in CAD patients (68.11 $\pm$ 3.61 pg/mL) compared with controls (11.39 $\pm$ 2.47 pg/mL) with a highly significant difference (P = 0.001).

Table 4: Serum levels of inflammatory cytokines in CAD patients and control group

Cytokine	CAD patients (n=83) Mean $\pm$ SD (pg/mL)	Control (n=80) Mean $\pm$ SD (pg/mL)	P-value
IL-1 α	53.21 $\pm$ 7.45	2.84 $\pm$ 0.51	0.001
TNF- α	109.37 $\pm$ 8.15	37.25 $\pm$ 4.83	0.001
IL-6	36.46 $\pm$ 5.04	7.63 $\pm$ 2.26	0.001

IL-17	68.11 ± 3.61	11.39 ± 2.47	0.001
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The expression level of miR-126 in blood samples of patients with coronary artery disease and the control group is presented in Table 5 and illustrated in Figure 1. The results showed that the mean expression level of miR-126 in the control group was 0.96-fold, whereas it was markedly increased in patients to 2.58-fold. This indicates an upregulation of miR-126 expression in patients compared to healthy controls. The ΔCT values were generally lower in patients than in the control group, reflecting higher gene expression levels. The calculated $2^{-\Delta\Delta\text{Ct}}$ values ranged from approximately 0.83 to 0.95 in controls and from 1.17 to 3.08 in patients.

Table 5: Expression of miR-126 in blood samples of patients and control

Groups	miR-126	U6	ΔCT	$\Delta\Delta\text{CT}$	$2^{-\Delta\Delta\text{Ct}}$	Folding	Mean
Control	31.3	25.3	6	0.275	0.826450318	0.826450318	0.957584
	31.1	25.3	5.8	0.075	0.949342121	0.949342121	
	31.4	25.5	5.9	0.175	0.885767519	0.885767519	
Patients	30.8	25.3	5.5	-0.225	1.168777249	1.168777249	2.584033
	29.7	25.6	4.1	-1.625	3.084421651	3.084421651	
	30.2	25.5	4.7	-1.025	2.034959384	2.034959384	
	29.5	25.4	4.1	-1.625	3.084421651	3.084421651	
	30.3	25.6	4.7	-1.025	2.034959384	2.034959384	
	29.6	25.5	4.1	-1.625	3.084421651	3.084421651	
	29.9	25.3	4.6	-1.125	2.181015465	2.181015465	

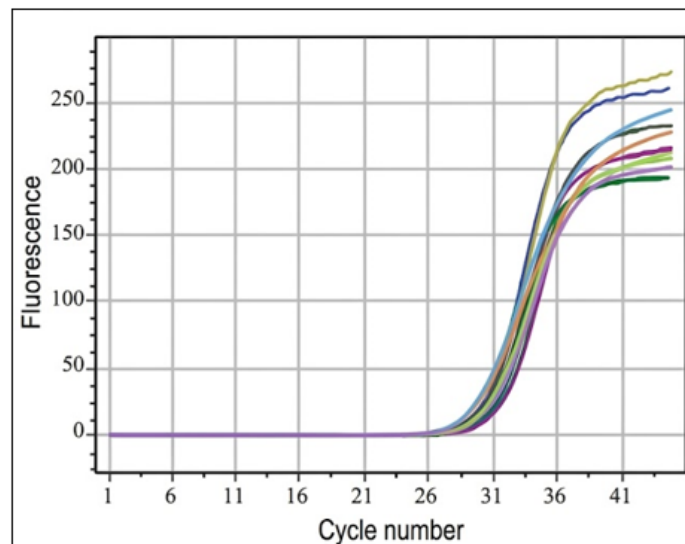


Figure 1: Real-time PCR profiling of miR-126 in patients and healthy subjects.

Pearson correlation analysis revealed significant positive correlations between miR-126 expression and inflammatory cytokines in patients with coronary artery disease (CAD). The expression of miR-126 showed a strong positive correlation with $\text{TNF-}\alpha$ ($r = 0.68$, $P = 0.001$), IL-17 ($r = 0.63$, $P = 0.001$), IL-1 α ($r = 0.61$, $P = 0.001$), and IL-6 ($r = 0.57$, $P = 0.001$). In addition, miR-126 expression demonstrated a moderate positive correlation with age ($r = 0.42$, $P = 0.003$) and body mass index (BMI) ($r = 0.39$, $P = 0.006$). However, no significant correlation was observed between miR-126 expression and gender ($r = 0.18$, $P = 0.091$), Table 6.

Table 6: Pearson correlation between miR-126 expression, inflammatory cytokines, and demographic parameters in CAD patients

Parameter	r-value	P-value	Significance
IL-1 α	0.61	0.001	Significant
TNF- α	0.68	0.001	Significant
IL-6	0.57	0.001	Significant
IL-17	0.63	0.001	Significant
Age (years)	0.42	0.003	Significant
BMI (kg/m ²)	0.39	0.006	Significant
Gender	0.18	0.091	Non-significant

4 DISCUSSION

In the current study, age had the strongest correlation with coronary artery disease (CAD), as the vast majority (37.3%) of patients were aged ≥ 60 years. This finding suggests that more than one-third of CAD cases in elderly individuals has accumulated a lot of aortic aging. This result agrees with Hussein et al. Aging is accompanied by the gradual development of endothelial dysfunction and decreased bioavailability of nitric oxide, which promotes atherosclerotic plaque formation (20). Likewise, Yusuf et al. The risk of myocardial infarction increases 2–3 fold each decade after 40 years of age (21), indicating the relatively powerful quantitative relationship between age and cardiovascular risk. Based on gender, 77.1% of CAD cases were males while only 22.9% were females. This also implied that the disease prevalence of CAD in the studied population was about 3.4 times higher in males than females. This finding is consistent with those obtained by Albayati and Shanshal (22), who noted similar excess of males among their Iraqi patients, which they attributed to less estrogen-mediated vascular protections in females and greater exposure of males to risk factors like cigarette smoking. With respect to body mass index (BMI), the current study revealed that 56.6% of the patients were obese while 37.3% had overweight status, indicating that the vast majority (93.9%) of CAD patients in the study had abnormal BMI values. Such a very high percentage reflects a solid contribution of metabolic status to the development of the disease. Findings from Saleh et al. corroborate these observations (23) after finding an obvious significant correlation between increase in BMI and decrease in cardiac efficiency and metabolic decompensation in their work on Iraqi patients. Powell-Wiley et al. In their study, (24) also proved that chronic systemic inflammation and lipid dysregulation resulting from 5 kg/m² increase in BMI leads to 20–30% of more cardiovascular risk. Similarly, in CAD patients present study clearly demonstrated significantly high levels of inflammatory cytokines compared to control with a substantial increase in IL-1 α (2.84 ± 0.51 pg/mL $\rightarrow$ 53.21 ± 7.45 pg/mL; ~ 18 folds). The concentrations of TNF- α also increased from 37.25 ± 4.83 pg/mL to 109.37 ± 8.15 pg/mL (nearly threefold), IL-6 from 25.17 ± 6.04 pg/mL to 116.73 ± 12.38 pg/mL (fivefold), and IL-17 from 1.65 ± 0.30 pg/mL to 9.60 ± 0.31 pg/mL (almost 6-fold) after progression of VC ($P < 0.05$, for all). These large increases reaffirm the inflammatory state seen in CAD. This is congruent Iraqi finding consisted in the Iraqi Journal of Pharmaceutical Sciences (25) where they revealed remarkable high levels of inflammatory markers in patients with acute coronary syndrome. A 2-fold increase in IL-6 level is associated with a significantly increased risk of future cardiovascular events, (26) as reported by Ridker. In addition, inflammation is not just a marker but also a trigger for the progression of atherosclerosis, Hansson (27) added. TNF- α is known to generate a plethora of pro-inflammatory signals, which cause endothelial activation, increase expression of adhesion molecules by as much as 60% and also enhance leakage of leukocytes into vascular tissue so as to instigate plaque instability, the study's lead author Libby (28) said. Recent study also reported increase in miR-126 expression, approximately 2.6 times in CAD patients in comparison to controls (2.58 vs 0.96 in the present study). This signified that there is a distinct induction of endothelial-associated microRNA activity in disease state. Wang et al. Similarly, (29) showed the association between decreased miR-126 with endothelial dysfunction and impaired vascular repair capacity. Jansen et al. It was shown (30) that the level of the angiogenic signaling pathways increased by almost 40–50%, which is the result of the action of a pro-endothelial factor miR-126. Gene expression changes, including microRNAs, are prominently implicated in cardiovascular disease development in the Iraqi population, as demonstrated by studies from Baghdad Science Journal (31), particularly with respect to vascular inflammation. Despite the pro-inflammatory role of miR-126, our study disclosed strong correlation of inflammatory cytokines with miR-126, finding coefficients for TNF- α ($r = 0.68$) and IL-17 ($r = 0.63$). The results imply a moderate-to-strong biological association of molecular regulation and systemic inflammation. Moreover, Wu and Zhang (32), revealed similar correlations of higher inflammatory burden and greater disease severity of CAD patients with increased miR-126 levels. Overall, this indicates that miR-126 may be considered as an upregulated compensatory repair marker, but also as a potential regulator of inflammatory signal pathways.

5 CONCLUSIONS

The present study concludes that coronary artery disease (CAD) is associated with increased age, male gender, and high body mass index, indicating the importance of demographic and metabolic risk factors. CAD patients showed significantly elevated inflammatory cytokines (IL-1 α , TNF- α , IL-6, and IL-17), confirming the role of systemic inflammation in disease progression. In addition, miR-126 was upregulated and positively correlated with inflammatory markers, suggesting its involvement in endothelial dysfunction and its potential as a biomarker in CAD.

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