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*Original Article*

# Evaluation of the Coenzyme $Q_{10}$ and Some Biochemical Parameters in Patients with Ischemic Heart Disease

**Ayob Alsaiegh, O. M[1 *], Lateef Husein, A[1], Harith Mohammad, M[2], Dhafer Abdulnafa, Z[2]**

*1. Department of Biochemistry, College of Medicine University of Tikrit, Tikrit, Iraq*
*2. College of Medicine, University of Mosul, Mosul, Iraq*

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Corresponding Author: othmanalsaiegh@gmail.com

**Abstract**

Ischemic heart disease (IHD) is a common diagnosis and a leading cause of death in both males and females. It accounts for 30% of deaths worldwide, including 40% in high-income countries and approximately 28% in developing nations. Several cardiac markers have been used to diagnose and manage cardiovascular diseases. The Coenzyme $Q_{10}$ ($CoQ_{10}$) plays a potential role in the prevention and treatment of cardiovascular diseases by improving cellular bioenergetics. This study aimed to evaluate the role of $CoQ_{10}$ and other biochemical parameters in IHD (angina pectoris and myocardial infarction). A case-control study was conducted at the Intensive Care Unit of Ibn-Sina Teaching Hospital and Al-Salam General Hospital in Nineveh Province, Iraq, for two months, from April 1 to June 1, 2022. It included 90 adult participants divided into case and control groups. The case group included 60 patients admitted to the Intensive Care Unit and diagnosed with IHD (myocardial infarction or angina pectoris), and the control group included 30 healthy participants matched in age and gender with the case group. Subsequent assay of C-reactive protein (CRP), creatine phosphokinase (CPK), troponin level, and serum $CoQ_{10}$. In this study, 81.7% of patients in the case group were diagnosed with myocardial infarction. Means of serum lactate dehydrogenase (LDH), CRP, CPK, and troponin were significantly higher, while those of $CoQ_{10}$ were significantly lower in the case group, compared to the controls. Statistically, a significant moderate negative correlation was detected between $CoQ_{10}$ level and age. Moreover, significant weak correlations were observed between $CoQ_{10}$ level and all serum LDH, CRP, and troponin levels. Patients with IHDs had considerably low serum levels of $CoQ_{10}$, compared to the control group. The highest mean value of lipid profile, except for triglyceride, was observed in patients with IHD, compared to the control group. This explains the role that cholesterol compounds play in the progression of IHD. No significant correlations were found between $CoQ_{10}$ with body mass index and CPK. The $CoQ_{10}$ had a negative correlation with age, serum LDH, CRP, and troponin.

**Keywords:** Biomarkers, Cardiovascular diseases, Coenzyme $Q_{10}$, Iraq, Ischemia

## 1. Introduction

Ischemic heart disease (IHD) is still a significant burden on individuals and healthcare resources worldwide. Despite clinical practice strategies that have evolved to optimize treatment for IHD, the consequences represent a significant burden on human health in terms of mortality and morbidity (1, 2). In 2015, IHD affected 110 million people and resulted in 8.9 million deaths. It is the reason for 15.6% of all deaths, making it the most common cause globally. The risk of death from IHD for a given age decreased between 1980 and 2010, especially in developed countries (1). The IHD has a number of well-determined risk factors, including hypertension, smoking, diabetes mellitus (DM), lack of exercise, obesity, high blood cholesterol, depression, and family history. It should be noted that nearly half of the cases are linked to genetics. Moreover, smoking and obesity

are associated with 36% and 20% of cases, respectively (1, 2).

Coenzyme $Q_{10}$ ($CoQ_{10}$) is an essential human body compound that is synthesized in the mitochondrial inner membrane. The $CoQ_{10}$ has many important functions in the human body. First, it can be named the key component of the electron transport chain in mitochondria necessary for ATP production as the $CoQ_{10}$ transfers electrons from complex-1 to complex-3. Furthermore, it plays a role in the transfer of protons in the inner mitochondrial membrane. This process is called protonmotive Q-cycle (3).

As a result of its important place in the functioning of organisms, there are many diseases and degenerative states associated with the deficiency of $CoQ_{10}$, such as DM, cardiovascular disease (CVD, including atherosclerosis, hypertension, and dyslipidemia), muscular dystrophy, Alzheimer's disease, and Parkinson's disease (4). Since $CoQ_{10}$ is an essential compound of the human body, there is growing evidence that $COQ_{10}$ is tightly linked to cardiometabolic disorders. Its supplementation can be useful in various chronic and acute disorders (5).

## 2. Materials and Methods

This case-control study was conducted at the Intensive Care Unit (ICU) of Ibn-Sina Teaching Hospital and Al-Salam General Hospital in Nineveh Province, Iraq, for two months, from April 1 to June 1, 2022. This study included 90 adult participants divided into two groups:

Case group: Included 60 patients admitted to the ICU and diagnosed with IHDs (myocardial infarction [MI] or angina pectoris).

Control group: Included 30 healthy participants matched in age and gender with the case group.

Diagnosis of IHD was based on symptoms, electrocardiogram, and biochemical markers of myocardial necrosis.

The blood samples were drawn from the vein. After cleaning the venipuncture site with iodine, 5 mm (10 ml) of blood sample was collected from each patient. It should be mentioned that the blood was drawn into a Gel Tube. The specimen for the gel tube was separated by centrifugation at 3,000 rpm for 10 min to get the serum. The separated serum was stored at –20 ℃ for the subsequent assay of

- ✓ Lipid profile, CRP, and CPK by COBAS C 111 technique.
- ✓ Troponin level by COBAS C 411 technique.
- ✓ Serum $CoQ_{10}$ by Enzyme-Linked Immunosorbent Assay Kit.

Verbal permission was obtained from each participant before data collection, and the information was anonymous. Names were removed and replaced by identification codes. All information is kept confidential in a password-secured laptop, and data is used exclusively for research purposes.

Official approval was granted from the Scientific Committee in the Department of Clinical Biochemistry, College of Medicine, Tikrit University, Tikrit, Iraq. Letter of facilitation was obtained from Tikrit College of Medicine to Ibn-Sina Teaching Hospital and Al-Salam General Hospital.

### 2.1. Statistical Analysis

The data were analyzed in the SPSS software (version 26) and presented as mean, standard deviation, and ranges. Frequencies and percentages present categorical data. An independent t-test (two-tailed) was used to compare the continuous variables between study groups. Pearson's correlation test (r) was also used to assess the correlation between the $CoQ_{10}$ marker level with specific parameters. A *P* value of less than 0.05 was considered statistically significant.

## 3. Results

In total, 90 patients participated in this study and were divided into case and control groups. The case group included 60 patients diagnosed with IHD, and the control group included 30 healthy participants.

### 3.1. Sociodemographic Characteristics

The distribution of study groups by sociodemographic characteristics is shown in figure 1 and table 1. Study participants were within the age ranges of 18-100 years

old, with a mean age of 52.37±16.6 years old. The majority of patients in the case group were aged ≥ 60 years (50%), while 50% of the controls were aged < 40 years.

In this study, the majority of participants in the case and control groups were male (70% and 80%, respectively) and employees (46.7% and 70%, respectively). Regarding the body mass index (BMI) level, 50% of the case group was overweight, while 66.7% of the control group had normal BMI levels.

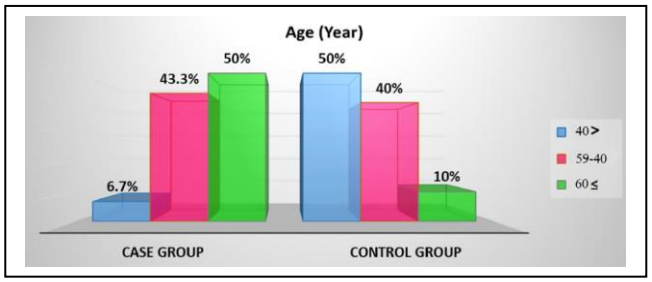


**Figure 1.** Distribution of study groups by age

**Table 1.** Distribution of study groups by sociodemographic characteristics

| Variable | Study group | | Total (%) n= 90 |
|---|---|---|---|
| | Case (%) n= 60 | Control (%) n= 30 | |
| **Gender** | | | |
| Male | 42 (70.0) | 24 (80.0) | 66 (73.3) |
| Female | 18 (30.0) | 6 (20.0) | 24 (26.7) |
| **Occupation** | | | |
| Private work | 10 (16.7) | 4 (13.3) | 14 (15.6) |
| Employee | 28 (46.7) | 21 (70.0) | 49 (54.4) |
| Housewife | 17 (28.3) | 2 (6.7) | 19 (18.9) |
| Student | 4 (6.7) | 0 (0) | 4 (4.4) |
| Retired | 1 (1.7) | 3 (10.0) | 4 (4.4) |
| **BMI Level** | | | |
| Normal | 15 (25.0) | 20 (66.7) | 35 (38.9) |
| Overweight | 30 (50.0) | 10 (33.3) | 40 (44.4) |
| Obese | 15 (25.0) | 0 (0) | 15 (16.7) |

### 3.2. Clinical Information

Table 2 summarizes the distribution of study groups by certain clinical information. It was noticed that 43.4% of the cases and 83.3% of the controls were smokers, and 40% of cases and 53.3% of controls had a positive family history of IHD. Regarding chronic medical diseases, 48.3% of cases had hypertension and DM, while most controls did not have chronic diseases (86.7%).

### 3.3. Diagnosis of Patients in the Case Group

As shown in figure 2, 81.7% of patients in the case group were diagnosed with MI.

**Table 2.** Distribution of study groups by certain clinical information

| Variable | Study group | | Total (%) n= 90 |
|---|---|---|---|
| | Case (%) n= 60 | Control (%) n= 30 | |
| **Smoking status** | | | |
| Current smoker | 26 (43.4) | 25 (83.3) | 51 (56.7) |
| Nonsmoker | 34 (56.6) | 5 (16.7) | 39 (43.3) |
| **Chronic medical disease** | | | |
| No | 8 (13.3) | 26 (86.7) | 34 (37.8) |
| Hypertension | 12 (20.0) | 2 (6.7) | 14 (15.6) |
| Diabetes Mellitus | 8 (13.3) | 2 (6.7) | 10 (11.1) |
| Hypertension+Diabetes | 29 (48.3) | 0 (0) | 29 (32.2) |
| IHD | 3 (5.0) | 0 (0) | 3 (3.3) |
| **Family history of IHD** | | | |
| Positive | 40 (66.7) | 16 (53.3) | 56 (62.2) |
| Negative | 20 (33.3) | 14 (46.7) | 34 (37.8) |

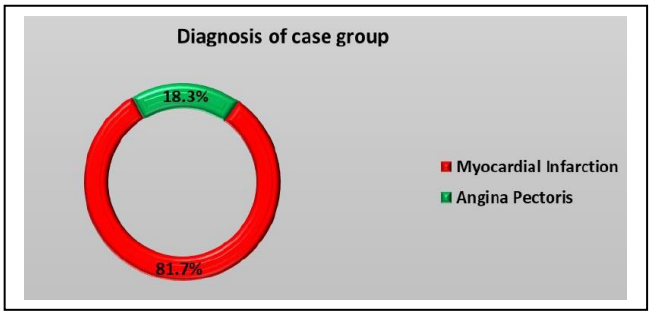


**Figure 2.** Distribution of case group by diagnosis

### 3.4. Biochemical parameters

Table 3 tabulates the distribution of study groups by specific biochemical parameters. In the case group, 71.7%, 100%, 63.3%, and 86.7% had high LDH levels, high CRP levels, high CPK levels, and high troponin levels, respectively. In the group of healthy individuals, 36.7% had high LDH levels, all had normal CRP and CPK, and 3.3% had high troponin levels.

#### 3.4.1. Comparison between Study Groups

Table 4. summarizes the comparison of specific biochemical parameters between study groups. it can be noticed that means of serum LDH, CRP, CPK, and

troponin were significantly higher in the case group, compared to the control group.

**Table 3.** Distribution of study groups by certain biochemical parameters

| Variable | Study group | | Total (%) n= 90 |
|---|---|---|---|
| | Case (%) n= 60 | Control (%) n= 30 | |
| **CRP** | | | |
| High | 60 (100.0) | 0 (0) | 60 (66.7) |
| Normal | 0 (0) | 30 (100.0) | 30 (33.3) |
| **CPK Level** | | | |
| High | 38 (63.3) | 0 (0) | 38 (42.2) |
| Normal | 22 (36.7) | 30 (100.0) | 52 (57.8) |
| **Troponin Level** | | | |
| High | 52 (86.7) | 1 (3.3) | 53 (58.9) |
| Normal | 8 (13.3) | 29 (96.7) | 37 (41.1) |

**Table 4.** Comparison of certain biochemical parameters between study groups

| Variable | Study group | | *P*-value |
|---|---|---|---|
| | Case Mean ± SD | Control Mean ± SD | |
| CRP (mg/L) | 32.7 ± 23.6 | 1.98 ± 1.2 | 0.001 |
| CPK (U/L) | 611.03 ± 733.0 | 94.73 ± 29.5 | 0.001 |
| Troponin (ng/ml) | 18.25 ± 22.7 | 0.016 ± 0.016 | 0.001 |

### 3.5. $CoQ_{10}$ Marker Level

The comparison in $CoQ_{10}$ Level between study groups is shown in figure 3 and table 5. In the case group, 65% of patients had low $CoQ_{10}$ levels, while all controls had normal levels. The mean value of the $CoQ_{10}$ level was significantly lower in the case group, compared to the control group (6.07 versus 12.41 mg/L, *P*=0.001).

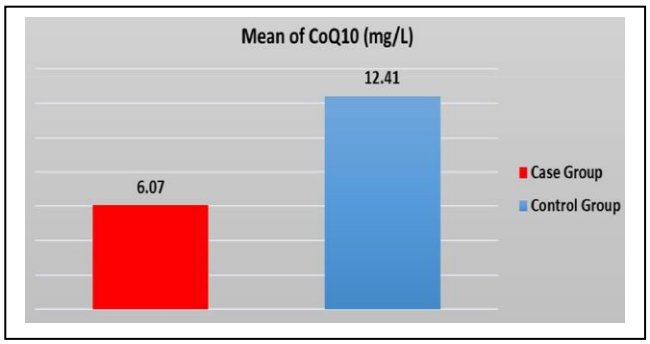


**Figure 3.** Mean of $CoQ_{10}$ in study groups

**Table 5.** Comparison in $CoQ_{10}$ Level between study groups

| $CoQ_{10}$ Level (mg/L) | Study group | | Total (%) n= 90 |
|---|---|---|---|
| | Case (%) n= 60 | Control (%) n= 30 | |
| Normal | 21 (35.0) | 30 (100.0) | 51 (56.7) |
| Low | 39 (65.0) | 0 (0) | 39 (43.3) |
| | **Mean ± SD** | **Mean ± SD** | ***P* - Value** |
| | 6.07 ± 1.6 | 12.41 ± 6.4 | 0.001 |

### 3.6. Correlation between $CoQ_{10}$ Marker and Specific Parameters

As shown in table 6 and figures 4, 5, 6, and 7, a statistically significant moderate negative correlation was detected between $CoQ_{10}$ level and age (r=-0.409, *P*=0.001). However, significant weak negative correlations were found between $CoQ_{10}$ level and all of the serum LDH (r=-0.216, *P*=0.04), CRP (r=-0.337, *P*=0.001, and troponin level (r=-0.235, *P*=0.026). The $CoQ_{10}$ level had no statistically significant correlations with BMI, serum cholesterol, triglyceride, HDL, and CPK.

**Table 6.** Correlation between $CoQ_{10}$ level and certain biological parameters

| Variable | $CoQ_{10}$ level (mg/L) | |
|---|---|---|
| | r | ***P* - Value** |
| Age (Year) | - 0.409 | 0.001 |
| BMI (kg/m$^2$) | - 0.142 | 0.244 |
| S. LDH (IU/L) | - 0.216 | 0.04 |
| CRP (mg/L) | - 0.337 | 0.001 |
| CPK (U/L) | - 0.201 | 0.057 |
| Troponin (ng/ml) | - 0.235 | 0.026 |

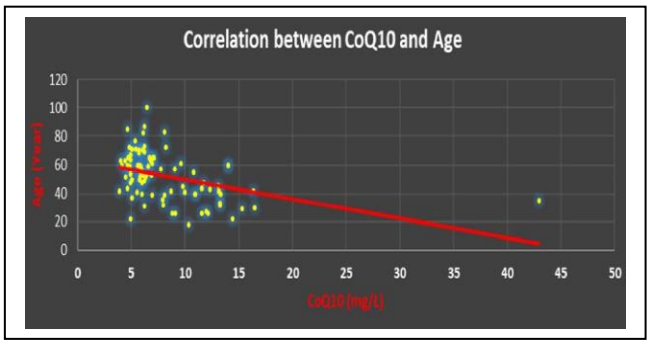


**Figure 4.** Correlation between $CoQ_{10}$ and age

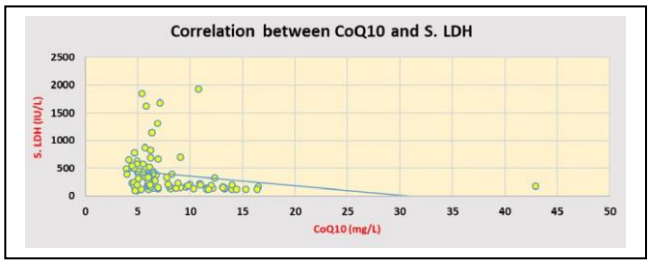


**Figure 5.** Correlation between $CoQ_{10}$ and S. LDH

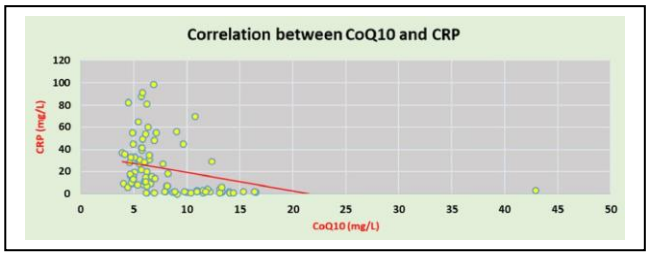


**Figure 6.** Correlation between $CoQ_{10}$ and S. LDH

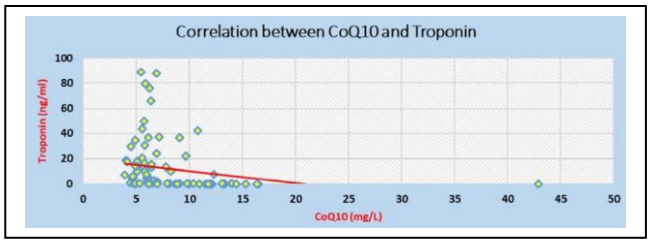


**Figure 7.** Correlation between $CoQ_{10}$ and troponin

## 4. Discussion

The current study was performed on 90 patients who were divided into two groups. The case group included 60 patients diagnosed with IHD, and the control group included 30 healthy participants.

Based on the findings, 43.4% of cases and 83.3% of controls were current smokers, and 40% of cases and 53.3% of controls had a positive family history of IHD. Regarding chronic medical diseases, 48.3% of cases had hypertension and DM, while most controls did not have chronic diseases (86.7%).

Results of a study conducted by Khandelwal, Kapoor (6) in 2022 were inconsistent with those of the present study. They studied 284 participants, 135 (47%), 98 (34%), and 82 (29%) of whom had arterial hypertension, DM, and smoking habits, respectively. Another study with different results was published by Bouzidi, Messaoud (7) in 2020, in which 48.4% of patients had DM, 47.1% of them had hypertension, and smoking was reported in 41.0% of them. In a study performed by Shabana, Shahid (8) in 2020, DM, hypertension, smoking, and family history were observed in 64.3%, 59.9%, 29.4%, and 36% of the patients, respectively.

Hypertension, DM, and smoking are closely interlinked as the risk factors of IHD due to similar risks, such as endothelial dysfunction, vascular inflammation, arterial remodeling, atherosclerosis, dyslipidemia, and obesity. The CVD complications, namely DM and hypertension, substantially overlap with those of microvascular and macro-vascular diseases. Common mechanisms, such as upregulation of the renin-angiotensin-aldosterone system, oxidative stress, inflammation, and immune system activation, likely contribute to the close relationship between diabetes and hypertension (9).

### 4.1. Diagnosis of Patients in the Case Group

In the present research, 81.7% of patients in the case group were diagnosed with MI. This rate is higher than that found in a study performed by Khandelwal, Kapoor (6) in 2022, in which 70% of patients had MI. Moreover, in the current study, high cholesterol levels, high triglyceride levels, low HDL levels, high LDH levels, high CRP levels, high CPK levels, and high troponin levels were found in 3.3%, 51.7%, 76.7%, 71.7%, 100%, 63.3%, and 86.7% of patients in the case group, respectively. Regarding the healthy individuals in this study, 33.3%, 40%, 53.3%, 6.7%, 100%, and 3.3% had high cholesterol levels, high triglyceride levels, low HDL levels, high LDH levels, normal CRP and CPK, and high troponin levels, respectively. In the present study and by comparison between study groups, mean values of serum LDH, CRP, CPK, and troponin were significantly higher ($P$<0.05) in the case group, compared to the control group.

### 4.2. $CoQ_{10}$ Marker Level

In the case group of the present study, 65% had low $CoQ_{10}$ levels, while all controls had normal levels.

Accordingly, the mean value of the $CoQ_{10}$ level was significantly lower in the case group (*P*=0.001). Moreover, a significant moderate negative correlation was detected between $CoQ_{10}$ level and age (r=-0.409, *P*=0.001). In addition, $CoQ_{10}$ had significant weak negative correlations with serum LDH (r=-0.216, *P*=0.04), CRP (r=-0.337, *P*=0.001), and troponin level (r=-0.235, *P*=0.026).

Low serum levels during the acute phase of IHD were associated with long-term mortality in patients, suggesting the utility of low serum $CoQ_{10}$ levels as a predictor and potential therapeutic target (10). Accordingly, in their study, Yalcin, Kilinc (11) explored the relationship between low plasma $CoQ_{10}$ concentration and coronary artery disease. They observed that plasma $CoQ_{10}$ concentrations in patients with IHD and controls were 0.77 and 0.41 μmol/l, respectively, with a significant relationship (*P*<0.01) (10).

It has been reported that $CoQ_{10}$ has a wide range of therapeutic effects; however, the mechanism behind these therapeutic benefits is not yet fully understood. In addition to showing potential as an antioxidant and functioning as a cofactor in the mitochondrial respiratory chain, it has been suggested to have gene regulatory properties that might account for its effects on overall tissue metabolism (12).

Three out of four patients with CVD have low levels of $CoQ_{10}$. It has been noticed that plasma levels of $CoQ_{10}$ in those with IHD and dilated cardiomyopathy are much lower than that in healthy individuals. Depending on the severity of the cardiac injury, the circulating level of $CoQ_{10}$ decreases in direct proportion to disease progression (13). There are several theories about the mechanism of action of $CoQ_{10}$ in CVD. First, regarding its antioxidant effect, as mentioned above, ubiquinone should be reduced to ubiquinol to ultimately show its anti-oxidative function. Reactive oxygen species (ROS) can lead to severe cellular damage by means of reacting with cell membranes, DNA, and protein centers (14).

Besides, the products of oxidative stress and cytokines may cause hypertrophy since they trigger the growth of myocytes. Ubiquinol (a reduced form of $COQ_{10}$) stops the initial formation of lipid peroxyl radicals. This is why $COQ_{10}$ is considered a very potent antioxidant against ROS and free radicals in biological membranes (15).

Secondly, it plays a significant role in the energetic needs of the heart. For example, the contraction of the cardiac, which involves the release of $Ca^{2+}$ from the sarcoplasmic reticulum, and the subsequent activation of the contractile proteins requires energy. There is a theory that reduced energy production may cause myocardial failure in mitochondria. Therefore, as it was mentioned earlier, $CoQ_{10}$ is the main component in the transport of electrons necessary for ATP production (5). Furthermore, its anti-inflammatory effect should be considered since CVDs, such as heart failure, are related to a chronic pro-inflammatory state, supposing increased cytokine levels and adhesion molecules (16). Some new studies have established anti-inflammatory properties of $CoQ_{10}$, possibly by means of the regulation of nitric oxide, and that mechanism may be effective in heart failure treatment. Therefore, the secretion of cytokines and chemokines would not induce myocardial fibrosis and lead to Heart Failure development (17).

Moreover, $CoQ_{10}$ has been reported to have a wide range of therapeutic effects. However, the mechanism behind these therapeutic benefits is not fully understood yet. In addition to showing potential as an antioxidant and functioning as a cofactor in the mitochondrial respiratory chain, $CoQ_{10}$ has been suggested to have gene regulatory properties that might account for its effects on overall tissue metabolism. Despite reports about its safety, efficacy in different disease states, and deficiency in many conditions, $CoQ_{10}$ supplementation is not widely prescribed in clinical practices. Potential reasons for this issue include a lack of understanding about the critical role of $CoQ_{10}$, ignorance of the detrimental effects of $CoQ_{10}$ deficiency, and the fact

that $CoQ_{10}$ is a nutraceutical rather than a patentable drug (12).

This was proved in a study performed by Kumar, Kaur (13), which reported a significant improvement in clinical and hemodynamic parameters and exercise tolerance in patients who received adjunctive $CoQ_{10}$ in 60-200 mg doses on a daily basis. The above-mentioned study was performed on patients with heart failure, hypertension, IHD, and other cardiac illnesses.

Results of another randomized study that involved diabetic patients with IHD supports those of the present study regarding the anti-inflammatory effect of $COQ_{10}$. However, no improvement was observed in the cardiometabolic markers in the aforementioned study. They concluded that $CoQ_{10}$ intake after 8 weeks among diabetic patients with stable IHD had beneficial effects on serum IL-6 levels but did not alter other cardiometabolic markers (18).

Despite these recommendations, findings of the research on $CoQ_{10}$ absorption and bioavailability differ and depend on the type of used $CoQ_{10}$ preparation. Many formulations have been developed to improve $CoQ_{10}$ solubility in the organism. Recent new formulations for $CoQ_{10}$ are based on enhancing its water-solubility, as in the cases of Q-Ter or Ubisol-$Q_{10}$. Ubisoft-$Q_{10}$ is a nano-micelle formulation that appears to be water-soluble containing $CoQ_{10}$, where solubilization is achieved due to the amphipathic properties of polyethyleneglycol-derivatized-tocopherol, which allows for the formation of stable and water-soluble nano-miscelle (19). Q-Ter is a supplement consisting of copovidone that acts as a carrier, $CoQ_{10}$, and glycine that works as a catalyst. This composition makes Q-Ter 200 times more soluble than pure $CoQ_{10}$ (20).

### 4.3. Sociodemographic Characteristics

The mean age of the participants was 52.37±16.6 years old, ranging from 18 to 100 years. The majority of participants in the case group were aged ≥ 60 years old (50%); while 50% of controls were aged < 40 years old. Regarding gender, the highest proportion of both groups were males (70% in the case and 80% in the control group). In terms of BMI level, 50% of the case group was overweighed, while 66.7% of controls had normal BMI levels.

Regarding the comparison with other studies, Biradar and Rangaswamy (21) conducted a study in 2022 on 80 patients. In the aforementioned study, the majority of participants were male (n=62, 77.5%) with a male-to-female ratio of 3.4:1. Moreover, the participants were above 18 years old with a mean age of 55.98±13.47 years old (21). Similar results were observed in a study carried out by Shabana, Shahid (8) in 2020, which was performed on 1,000 subjects. The mean age of patients with IHD was 59.1±12.7 years old, with a slight male predominance, as they constituted 58.2% of the participants. In the above-mentioned research, the male-to-female ratio was 1.3:1. Moreover, the mean value of the BMI among participants was 22.4±6.7 $Kg/m^2$ (8).

Different results were observed in a study performed by Thabet NI (22) on 273 patients who were admitted to the coronary care unit with a final diagnosis of IHD. In the aforementioned study, there were 160 (58.6%) males and 113 (41.4%) females with a male to a female ratio of 1.4:1. Moreover, the mean age of participants was 58.9+11.3 years old (range: 27-87 years old). Besides, their mean BMI value was 25.2±5 $kg/m^2$ (range: 15-41 $kg/m^2$). In that research, 108 (39.6%), 89 (32.6%), and 86 (31.5%) patients were associated with hypertension, DM, and stroke, respectively (22).

Different sample sizes in each study as well as socioeconomic, ethnic, environmental, educational, urbanization, physical activity, and drugs, can lead to the observed differences. Another important factor determining the difference was gender, as female participants were more diabetic, hypertensive, obese, hypertriglyceridemic, less or passive smokers, and older, compared to male participants. Meanwhile, the male participants smoked more and were thinner and younger than females, due to cultural habits.

This study confirmed the role of $CoQ_{10}$ in IHD, where the patients had considerably low serum levels of

$CoQ_{10}$, compared to the control group. The $CoQ_{10}$ had no significant correlations with BMI and CPK. However, a negative correlation was found between $CoQ_{10}$ with age, serum LDH, CRP, and troponin.

## Authors' Contribution

Study concept and design: O. M. A. A.
Acquisition of data: A. L. H.
Analysis and interpretation of data: M. H. M.
Drafting of the manuscript: Z. D. A.
Critical revision of the manuscript for important intellectual content: O. M. A. A.
Statistical analysis: A. L. H.
Administrative, technical, and material support: M. H. M.

## Ethics

Official approval was granted from the Scientific Committee in the Department of Clinical Biochemistry, College of Medicine, Tikrit University, Tikrit, Iraq. Letter of facilitation was obtained from Tikrit College of Medicine to Ibn-Sina Teaching Hospital and Al-Salam General Hospital.

## Conflict of Interest

The authors declare that they have no conflict of interest.

## References

1. Wang H, Bhutta ZA, Coates MM, Coggeshall M, Dandona L, Diallo K, et al. Global, regional, national, and selected subnational levels of stillbirths, neonatal, infant, and under-5 mortality, 1980–2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet. 2016;388(10053):1725-74.
2. Kivimäki M, Nyberg ST, Batty GD, Fransson EI, Heikkilä K, Alfredsson L, et al. Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis of individual participant data. Lancet. 2012;380(9852):1491-7.
3. Ayer A, Macdonald P, Stocker R. CoQ10 function and role in heart failure and ischemic heart disease. Annu Rev Nutr. 2015;35:175-213.
4. Garrido-Maraver J, Cordero MD, Oropesa-Ávila M, Vega AF, De La Mata M, Pavón AD, et al. Coenzyme q10 therapy. Mol Syndromol. 2014;5(3-4):187-97.
5. Zozina VI, Covantev S, Goroshko OA, Krasnykh LM, Kukes VG. Coenzyme Q10 in Cardiovascular and Metabolic Diseases: Current State of the Problem. Curr Cardiol Rev. 2018;14(3):164-74.
6. Khandelwal V, Kapoor A, Kazmi D, Sinha A, Kashyap S, Khanna R, et al. Exploring the association of fibrinogen and CRP with the clinical spectrum of CAD and periprocedural outcomes in patients undergoing percutaneous coronary interventions. Ann Card Anaesth. 2022;25(1):34-40.
7. Bouzidi N, Messaoud MB, Maatouk F, Gamra H, Ferchichi S. Relationship between high sensitivity C-reactive protein and angiographic severity of coronary artery disease. J Geriatr Cardiol. 2020;17(5):256-63.
8. Shahid SU, Sarwar S. The abnormal lipid profile in obesity and coronary heart disease (CHD) in Pakistani subjects. Lipids in health and disease. 2020;19(1):1-7.
9. Petrie JR, Guzik TJ, Touyz RM. Diabetes, Hypertension, and Cardiovascular Disease: Clinical Insights and Vascular Mechanisms. Can J Cardiol. 2018;34(5):575-84.
10. Shimizu M, Miyazaki T, Takagi A, Sugita Y, Ouchi S, Aikawa T, et al. Low coenzyme Q10 levels in patients with acute cardiovascular disease are associated with long-term mortality. Heart Vessels. 2021;36(3):401-7.
11. Yalcin A, Kilinc E, Sagcan A, Kultursay H. Coenzyme Q10 concentrations in coronary artery disease. Clin Biochem. 2004;37(8):706-9.
12. Potgieter M, Pretorius E, Pepper MS. Primary and secondary coenzyme Q10 deficiency: the role of therapeutic supplementation. Nutr Rev. 2013;71(3):180-8.
13. Kumar A, Kaur H, Devi P, Mohan V. Role of coenzyme Q10 (CoQ10) in cardiac disease, hypertension and Meniere-like syndrome. Pharmacol Ther. 2009;124(3):259-68.
14. Bergamini C, Cicoira M, Rossi A, Vassanelli C. Oxidative stress and hyperuricaemia: pathophysiology, clinical relevance, and therapeutic implications in chronic heart failure. Eur J Heart Fail. 2009;11(5):444-52.
15. Lim J-Y, Park SJ, Hwang H-Y, Park EJ, Nam JH, Kim J, et al. TGF-β1 induces cardiac hypertrophic responses via PKC-dependent ATF-2 activation. J Mol Cell Cardiol. 2005;39(4):627-36.
16. Yang Y-K, Wang L-P, Chen L, Yao X-P, Yang K-Q, Gao L-G, et al. Coenzyme Q10 treatment of

cardiovascular disorders of ageing including heart failure, hypertension and endothelial dysfunction. Clinica Chimica Acta. 2015;450:83-9.

17. Swarnakar NK, Jain AK, Singh RP, Godugu C, Das M, Jain S. Oral bioavailability, therapeutic efficacy and reactive oxygen species scavenging properties of coenzyme Q10-loaded polymeric nanoparticles. Biomaterials. 2011;32(28):6860-74.
18. Mirhashemi SM, Najafi V, Raygan F, Asemi Z. The effects of coenzyme Q10 supplementation on cardiometabolic markers in overweight type 2 diabetic patients with stable myocardial infarction: A randomized, double-blind, placebo-controlled trial. ARYA Atheroscler. 2016;12(4):158.
19. Muthukumaran K, Leahy S, Harrison K, Sikorska M, Sandhu JK, Cohen J, et al. Orally delivered water soluble Coenzyme Q10 (Ubisol-Q10) blocks on-going neurodegeneration in rats exposed to paraquat: potential for therapeutic application in Parkinson's disease. BMC Neurosci. 2014;15(1):1-11.
20. Fumagalli S, Fattirolli F, Guarducci L, Cellai T, Baldasseroni S, Tarantini F, et al. Coenzyme Q10 terclatrate and creatine in chronic heart failure: a randomized, placebo-controlled, double-blind study. Clin Cardiol. 2011;34(4):211-7.
21. Biradar MS. Lipid Profile Study in Patients Diagnosed with Acute Myocardial Infarction for First Time and Admitted in Tertiary Care Hospital Mysuru. J Assoc Physicians India. 2022;70(4):11-2.
22. Thabet NI, Hassanin HA, Kamal YM. Study of High Density Lipoprotein Cholesterol among Patients with Acute Coronary Syndrome in Sohag University Hospital. J Cardiol Curr Res. 2015;2(5):00073.