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## Research Article

# The Relationship Between Serum Levels Of Vitamin C, Cholesterol, Triglyceride, HDL, LDL, VLDL, And C-Reactive Protein With Coronary Heart Disease

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## ABSTRACT

Background: coronary heart disease is the major cause of death and disability in women and in men. Lipid abnormalities, high blood pressure, and smoking are major risk factors for CHD. Objective: This study aimed to evaluated the level of Cholesterol, Triglyceride, HDL, LDL, VLDL, Vitamin C and C-Reactive Protein in patients with Coronary artery disease. Materials and methods: This study investigated 90 samples, consisting of 60 patients with CHD and 30 controls, with ages ranging (30- >70) years. Samples were obtained from patients who were referred to Tikrit Hospital during the period from September 2023 to March 2024. Results: This study showed that increase prevalence of coronary heart disease in male that was 39(65%), while in female was 21(35%)in the female at P. Value(0.03). whereas no differences peak age of CHD patients was between > 70 years and its percentage was 48.3%, while the least age group 30 - 49 years and its percentage which was found to be 20%. This study show increase the level of Cholesterol, Triglyceride, LDL VLDL, and C-Reactive Protein in CHD that were (213.2±27.0, 235.3±66.9, 128 ± 16.2, 47.6±8.3, 4.63±2.9 ) mg/dl respectively, as compared with control that were (123.4±23.5, 103.9 ± 22.5, 102.6 ± 12.2, 20.8 ± 4.5, 2.04 ± 1.05 ) mg/dl respectively, at p-value <0.05 While no differences in the HDL level between CHD and control (37.6 ±6.3, 35.9±5.4)mg/dl respectively, at p-value >0.05. While decrease the level of Vitamin C in the CHD patients that was(5.65±1.7) mg/dl as compared with control that was (11.17±2.7) mg/dl, at p-value <0.05. Conclusion: The results showed a positive correlation between level of Cholesterol, Triglyceride, LDL VLDL, and C-Reactive Protein in patients with coronary heart disease. While there is a there is a decrease in the level of vitamin C and no difference in the level of HDL in

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patients with coronary heart disease the level of vitamin C and no difference in the level of HDL in patients with coronary heart disease

## INTRODUCTION

Coronary heart disease (CHD) was the most prevalent cause of mortality around the world, constituting around 28% of the global fatalities in the year 1983 [1]. The recent study from the United States Predictive Service Working Group highlights an annual incidence of around 1.6million occurrences of cardiac infarction, resulting in approximately 520,000 fatalities. The appearance of CHD is influenced by a range of factors, which can be categorized between modifiable and non-modifiable causes. The non-modifiable factors contain advanced age and a familial predisposition to early CHD, while the modifiable factors encompass elevated blood cholesterol levels and hypertension, diabetes, smoking, lack of physical exercise, and being male, have been thoroughly documented and widely researched [2]. Nevertheless, when considering all the identified factors contributing to CHD, some individuals believe that these factors can only explain around 50-60% of the occurrence of the condition. There is a relationship among level of vitamin C and CHD, The use of Vitamin C reduced the increase in troponin and CKMB levels [3]. Furthermore, the administration of vitamin C alone in animal experiments resulted in a reduction in infarct size. The findings from preclinical studies indicate that vitamin C may possess preventive properties for the myocardium in humans during cardiac stress scenarios. By the presence of Vitamin C the continual enhancement of the endothelial system is significantly influenced [4]. The therapeutic significance of these biochemical changes is supported by the observation Vitamin C has been found to enhances endothelium metabolism in individuals who have been diagnosed as having atherosclerotic and cardiac disease [1]. Moreover,

vitamin C demonstrated enhanced myocardial perfusion in those having percutaneous coronary intervention (PCI) and those diagnosed with CHD[5]. Vitamin C has been shown to have a big role in many different processes in biology, such as removing radicals that are harmful, converting cholesterol into bile acid, protecting membranes of lipids, and producing the protein collagen, and specific neurotransmitters [6]. Numerous research have been undertaken to examine the potential of vitamin C intake in mitigating or decelerating the progression of (CVDs) additional ages related with persistent diseases, such as Alzheimer's disease (AD), which are impacted by oxidative stress [7]. There is a hypothesis suggesting that Vitamin C could potentially serve as a preventative agent in the advancement of atherosclerotic heart disease through its ability to hinder the oxidation process of low-density lipoproteins[8].

Therefore, Further research is required to elucidate the efficacy of vitamin C levels in the prevention, reduction, or treatment of CHD. Hence, the objective of this investigation was to assess the vitamin C levels by analyzing blood samples collected from individuals who had not eaten.

## MATERIALS AND METHODS

### Subjects

The present study included 90 samples, with 30 individuals presenting coronary heart disease (CHD), and 30 healthy individuals as control. The age range of the participants was between 30 and over 70 years, including both genders. Specimens were obtained from hospital between September 2023 and March 2024. The participants involved in this research were classified into two distinct cohorts: First cohort comprised 60 individuals diagnosed with heart disease, whereas the subsequent cohort comprised 30 peoples without any diseases. Data on the clinical history, including age, weight, height, family history of



heart disease, and treatment progression, were gathered by a concise questionnaire.

### **Ethics approval**

This study was conducted based on the ethical standards stipulated in the Declaration of Helsinki. Before taking the sample, The patient's informed written and verbal agreement was obtained, after the review and approval of the study protocol and subject's information by the local ethics committee

### **Sample Collection**

Samples For biochemical analysis, venous blood samples were collected from both patients and controls after a minimum 12-hour fasting period. Using a typical method of drawing blood from the inner elbow, two tubes were filled, one containing an anticoagulant called EDTA. The samples transferred to an EDTA-free tube and allowed to coagulate at room temperature. Subsequently, The specimens of blood were acquired using the process of centrifugation at a speed of 2500 rpm for 9-10m to duration, leading to the separation of serum. Next, the serum was partitioned into two separate tubes and subsequently stored at a temperature of -20°C until it was required for analysis. Prior to testing physiological parameters, such as TC, HDL, T.G, LDL, VLDL, CRP, and Vitamin C, the samples were let to thaw at room temperature. Measurements were taken for serum levels of TC, HDL, T.G, LDL, VLDL, CRP, and Vitamin C.

#### **a. The quantification of serum amounts of vitamin C (ascorbic acid):**

The method described by Subash-Babu et al[9] was employed for determining the content of vitamin C. A volume of 100 µL of the samples was subjected to precipitation using 250 µL of a 5% solution of ice-cold tricarboxylic acid. The precipitating sample underwent centrifugation at 2,500 revolutions per minute for a duration of 20 minutes using a tabletop centrifuge. The supernatant was combined with 0.2 mL of 2, 4 dinitrophenylhydrazine: thiourea: copper

sulphate, resulting in a combination of one-tenth of 1.0 mL. It was incubated at a temperature of 37°C for a duration of 3 hours. Ultimately, 1.5 mL of chilled 65% H<sub>2</sub>SO<sub>4</sub> was introduced and meticulously blended. The resulting solution was maintained at 36 -37°C for an additional duration of 30 minutes, during which the absorbance of the solution was measured at a wavelength of 530 nm using an UV spectrophotometer. The vitamin C values were quantified as <sup>1</sup>g/mg protein.

- a. The Enzyme-Linked Immunosorbent Assay (ELISA) was employed to measure serum C-reactive protein (CRP) level of hormone testosterone. The plasma levels of CRP concentration were quantified using a sandwich ELISA, specifically the CRP ELISA Kit from Sunlong, China.
- b. level of cholesterol were assessed utilizing the BIOLABO kits reagent for in vitro detection of cholesterol CHOD PAP. The principle AL-OMARI et al [10] provide a suggested value of less than 200mg/dl.
- c. The blood triglyceride level was measured using the BIOLABO reagents kit for triglycerides GPO Method, which is an in vitro diagnostic test. According to AL-OMARI et al.,[10]. The acceptable range is 35-160 mg/dl.
- d. The elevated levels of the cholesterol in high density lipoprotein in the blood were assessed by employing the BIOLABO kits for HDL-cholesterol (PTA) precipitant through in vitro diagnostic. Expected result: A risk factor of low magnitude is delineated, as being below 40 mg/dl, while a high level of protective factor is defined as being equal to or more than 60 mg/dl.
- e. Serum low density lipoprotein cholesterol was calculated using an indirect approach. The levels of cholesterol, triglycerides (T.G), and HDL cholesterol were assessed, while LDL cholesterol was derived from the initial data



using the Friedewald equation (AL-OMARI et al., [10]. Recommended value <130 mg/dl. For the calculation of LDL – Cholesterol Use formula:-

$$\text{LDL (mg /dl)} = \frac{\text{Total cholesterol} - (\text{HDL} + \frac{\text{Conc. of triglyceride}}{5})}{5}$$

g. To estimate VLDL, divide the triglyceride value by 5 according to this formula [12] :

$$\text{VLDL} = \frac{\text{Conc. of triglyceride}}{5}$$

### Statistical Analysis

The analysis of variation (F-test) Fisher test was employed to analyze the results, while Duncun's multiple range test was utilized to assess the statistical variations among the means of arithmetic at a significance level of P>0.05.

**Table (2): The division of the study's sample based on age cohorts.**

Age groups	n:	%
( 30 - 49 years)	12	20%
(50 – 69 years)	19	31.7%
( > 70 years )	29	48.3%
<b>Total</b>	100	100%
<b>P. Value</b>	0.06	

Table (3): This study show increase the level of Cholesterol, Triglyceride, LDL VLDL, and C-Reactive Protein in CHD that were (213.2±27.0, 235.3±66.9, 128 ± 16.2, 47.6±8.3, 4.63±2.9 ) mg/dl respectively, as compared with control that were (123.4±23.5, 103.9 ± 22.5, 102.6 ± 12.2, 20.8 ± 4.5, 2.04 ± 1.05 ) mg/dl respectively, at p-value

## RESULTS

The results in Table 1, show the total number of participants in research consisted of 90 individuals, comprising 60 patients diagnosed with heart disease and 30 healthy persons. The study revealed a higher prevalence of CHD in males, the rate of 39 (65%), compared to 21 (35%) in females, with a P-value of 0.03. This information is presented in Table (1).

**Table (1): Distribution the coronary heart disease patients According to gender**

CHD patients	%
<b>Male</b>	39(65%)
<b>Female</b>	21(35%)
<b>P. Value</b>	0.03

Table(2): This study showed that the no differences peak age of CHD patients was between > 70 years and its percentage was 48.3%, while the least age group 30 - 49 years and its percentage which was found to be 20%.

<0.05 While no differences in the HDL level between CHD and control (37.6 ±6.3, 35.9±5.4) mg/dl corresponding , at p-value >0.05. While an increase the levels of Vitamin C in the CHD patients that was(5.65±1.7) mg/dl as compared with control that was (11.17±2.7) mg/dl, at p-value <0.05

**Table (3): Comparison of cholesterol and triglyceride HDL, LDL and vit C, C-RP hs VLDL for total study groups**

Parameters	CHD	Control	p-value
<b>Cholesterol (mg/dl)</b>	213.2±27.0	123.4±23.5	<0.05
<b>Triglyceride (mg/dl)</b>	235.3±66.9	103.9 ± 22.5	<0.05
<b>HDL (mg/dl)</b>	37.6 ±6.3	35.9±5.4	>0.05
<b>LDL (mg/dl)</b>	128 ± 16.2	102.6 ± 12.2	<0.05
<b>VLDL (mg/dl)</b>	47.6±8.3	20.8 ± 4.5	<0.05
<b>Vitamin C</b>	5.65±1.7	11.17±2.7	<0.05
<b>C-Reactive Protein</b>	4.63±2.9	2.04 ± 1.05	<0.05



Table (4): This study show no variations at level. 50.7± 31.7, 5.6± 1.2, 2.6±1.9 ) mg/dl respectively of Cholesterol, Triglyceride, HDL, LDL, VLDL, in male, as compared with female that were Vitamin C and C-Reactive Protein in CHD (211.6± 27.9, 224.4±67.7, 39.6±2.5, 127.12, patients according to gender that were 44.9±19.0, 5.6± 2.0, 5.8±2.5 ) mg/dl respectively, (216.0±27.9, 253.5±67.3, 34.3±9.4, 131±29.7, at p-value >0.05.

**Table (4): Comparison of cholesterol and triglyceride for total study groups according to gender**

Parameter	Male	Female	P. Value
	Mean ±SD		
Cholesterol (mg/dl)	216.0± 27.9	211.6± 27.9	0.7
Triglyceride (mg/dl)	253.5±67.3	224.4±67.7	0.4
HDL (mg/dl)	34.3±9.4	39.6±2.5	0.1
LDL(mg/dl)	131±29.7	127.12	0.5
VLDL (mg/dl)	50.7± 31.7	44.9±19.0	0.2
Vitamin C	5.6± 1.2	5.6± 2.0	0.9
C- Reactive Protein	2.6±1.9	5.8±2.5	0.4

## DISCUSSION

This study aligns with the findings of [11], which indicate a higher incidence of (CHD) in males compared to females. Another study conducted by Adak et al. revealed that the total number of 599 patients, 317 were males while 282 were female [12]. The frequency of CHD made Men had a threefold greater incidence of CHD and a fivefold higher rate of mortality compared to women. Nearly half of reported sex difference in CHD can be attributed to the sex differences in the assessed cardiovascular risk variables[13]. Moreover, variations in smoking prevalence significantly contributed to the heightened risk of CHD among males. The influence of smoke on the gender discrepancy in (CHD) hazard may be more significant than what we have predicted in our analysis, as smoking can also reduce HDL cholesterol levels [14]. Larsson and colleagues conducted a study in the early 1990s to determine if variations in smoking amount. The disparity in the occurrence of (CHD) among 55-year-old Swedish men and women may be attributed to variations in cholesterol levels, elevated blood pressure and weight of body , between gender. The researchers reached the conclusion that variations in waist-to-hip ratio accounted for

nearly all of the disparity in CHD risk across sexes. Furthermore, the inclusion of other risk factors in the analysis had only a minimal impact on the outcomes[15]. That research disagree with [16] that show increase CHD incidence in female than male. incidence and mortality. According to this study, the risk of CHD increases noticeably with ageing. The research by Jousilahti, et al indicates a rise in CHD. Men begin to exhibit symptoms at the age between ( 45 to 50), while women see a fast increase in symptoms until they are 60 to 65 years old [17]. Similar to high cholesterol levels, blood pressure increases with age, with women experiencing this increase more pronouncedly than men [18]. Obesity is likely a contributing factor in the rise in blood pressure and its differing relationships with age in men and women [18, 19]. A different study reveals that of the 126 male patients analyzed, the largest number of patients, or 37 (18%), were between the ages of 40 and 50, 31 (15%), and 55- 60 or older, 31 (15%), and 25 (13%) were under 40 age . Of the 74 female patients in our study, the largest number of patients, or 25 (13%) were between the ages from 50 - 60, 20 (10%) were between the ages from 40 - 50, 20 (10%) were over 60, and 8 (4%) were under the age of 40 [1].

The findings of this study indicate a reduction in high-density lipoprotein cholesterol (HDL-C) levels and an elevation in total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C). The present study provides support for the findings of [20], which suggest the presence of lipids in individuals diagnosed with heart disease. A primary aetiology of CAD also known as lipids. Classical danger signs for myocardial infarction patients include (TC), (TG), (LDL-C), and reduced (HDL-C)[10]. HDL is of paramount importance in the regulation of cholesterol equilibrium between the arterial vasculature and many organs, including liver [21]. When there is a malfunction in system of cholesterol transport reversal mediated by HDL, such as in individuals who are unable to move cholesterol from the macrophages in the artery intima to HDL, it leads to the development of early and severe atherosclerosis and CHD [22]. The results of this study confirm the significant importance of HDL cholesterol, since low levels of HDL cholesterol were strongly linked to a negative correlation with CHD, particularly (VLDL), are accountable for transportation of triglycerides (TG) in plasma. The VLDL concentration is a more reliable indicator of events compared to TGs[23]. VLDL is a heterogeneous collection of particles, with certain particles exhibiting a more pronounced correlation with CHD than others. Certain very low-density lipoproteins (VLDLs) are found to include an apolipoprotein known as apolipoprotein C-III. This apolipoprotein has been found to hinder the metabolism of VLDL as well as directly trigger atherogenic and inflammation activities in vascular tissue cells and monocyte[24]. Therefore, lipoprotein types that contain apolipoprotein C-III, such as very (VLDL[25]. and its metabolite apolipoprotein C-III-containing (LDL), are particularly associated with atherosclerosis. The

concentration of these lipoproteins in the bloodstream is a reliable Forecaster of CHD [26]. This study demonstrates a reduction in vitamin C levels in patients with CHD. The possible protective effect of plasma vitamin C on heart disease in both men and women has been suggested. Plasma vitamin C levels were linked to a notable decrease in the risk of CHD [27]. The research conducted by [28] demonstrated that the consumption of vitamin C supplements was linked to a notably reduced likelihood of CHD over a period of 16 years. Another study has determined that there is an inverse relationship between dietary vitamin C and the risk of CHD, but the consumption of vitamin C supplements does not show a meaningful correlation with CHD risks [29]. A separate investigation revealed a negative correlation between the consumption of vitamin C through a food frequency questionnaire and the mortality rate of CHD in Japanese women who did not have a previous history of cardiovascular disease or cancer . A study conducted in 2013 revealed that the use of vitamin C, in a dosage range of 120 to 1000 mg, along with vitamin E and beta-carotene, did not have a significant impact on CHD and major heart attacks [30]. Early atherosclerotic lesions have demonstrated the buildup of CRP. Moreover, C-reactive protein (CRP) exhibits chemotactic properties inside leukocytes possessing the receptor of CRP [31]. It is believed that the involvement monocytes in the process of inflammation leads to an increase in thermogenesis. Studies have demonstrated that CRP has an impact on cultured endothelial cells, leading to a reduction in nitric oxide levels and an increase in release of endothelin-1 [32]. Long-term follow-ups indicate that individuals with elevated CRP levels had a greater chance of developing CAD compared to the general healthy population [33]. Mani, et al. shown that plasma CRP is autonomously linked to the burden of atherosclerosis in the heart [34]. Furthermore,



there is a correlation between the rise in CRP levels during acute coronary syndrome and the occurrence of cardiovascular and all-cause mortality [35].

## CONCLUSION

This study concluded increase incidence of CHD in male than female and CHD risk increases with age. Also this study concluded increase the level of Cholesterol, Triglyceride, LDL VLDL, and C-Reactive Protein in CHD, While no differences in the HDL level between CHD and control. Furthermore Reduce the Vitamin C levels in people with CHD. Furthermore, there were no observed disparities in the levels of Cholesterol, Triglyceride, HDL, LDL, VLDL, Vitamin C and C-Reactive Protein in CHD patients according to gender.

## FUNDING

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## CONFLICTS OF INTEREST

There are no conflicts of interest.

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