



Estimation of ALP in Patients with Coronary Artery Disease

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ABSTRACT

Back ground: This study examined the associations between emerging lipid biomarkers, Alkaline phosphatase (ALP), and coronary artery disease (CAD). Globally, cardiovascular disease (CVD) continues to be the leading cause of death, with CAD playing a major role. Elevated ALP levels are one of the major risk factors for CAD.

Objective: This study set out to determine whether there is a link between serum ALP levels and the likelihood and severity of CAD, to assess the relationship presentation with CAD.

Materials and Methods: A case-control study involving 100 subjects; 50CAD patients, and 50 healthy individuals as a control who aged 40-75 years, was carried out. Serum ALP levels were made use of a Spectro photometer.

Results: The study revealed a significant increase in serum ALP levels when comparing patients to healthy control groups (73.208 ± 22.704 U/I compared to 65.223 ± 20.408 U/I with $P = 0.049$) respectively.

Conclusion: Patients who suffer from coronary artery disease and coronary arteries had higher level of ALP.

KEY WORDS: ALP, CAD, Inflammation, Enzyme, plaque

INTRODUCTION

Coronary artery disease (CAD), commonly known as coronary heart disease, is a prevalent kind of heart ailment defined by the narrowing of coronary arteries due to plaque deposition, resulting to restricted blood supply to the heart muscle [1]. This disease, which is mainly brought on by atherosclerosis, can take years to show symptoms.

It frequently starts as dyspnea and chest pain, and if the blood supply is completely blocked, it may result in a heart attack. Surgery and medication are available as treatments for coronary artery disease (CAD), and preventing the condition with healthy eating, regular exercise, and quitting smoking can help reduce its risk factors [1]. methods for preventive and control to deal with this expanding health issue [2].

Alkaline phosphatase (ALP) is a crucial enzyme involved in various physiological processes, particularly in the liver, bones, kidneys, and intestines. Its levels in the bloodstream serve as important biomarkers for diagnosing liver and bone disorders. Elevated or decreased ALP levels can indicate specific health conditions, making its measurement vital in clinical settings[3].

A higher risk of coronary artery disease (CAD) has been linked to elevated levels of alkaline phosphatase (ALP). Multiple processes are thought to be involved in the link between ALP and CAD.

Calcium in the Vascular System ALP encourages the blood vessels to deposit calcium. This may result in vascular calcification, or the hardening of the arteries[4].

This is a major cause of atherosclerosis, a disorder that narrows and stiffens the arteries and causes coronary heart disease (CAD). Inflammation: High ALP readings could be a symptom of underlying chronic inflammation, which is a recognized risk factor for heart conditions like CAD [5].

The lining of the coronary arteries may be harmed by inflammation, which encourages the accumulation of plaque. Metabolic Status of Bone and Cardiovascular Health Given that ALP contributes to abnormalities in bone metabolism increases the chance of calcified plaques in coronary arteries, which can have a cascading effect on vascular health. Although further research is needed to completely explain the association, a number of studies have demonstrated a correlation between higher ALP levels and a greater incidence of CAD, suggesting that ALP could be a helpful marker for predicting cardiovascular risk[6].

MATERIALS AND METHODS

The study used a case control research approach, data was collected from 100 subjects obtained from AL-Najef Heart Center (AL-Sadder Hospital) in Najef , Iraq. Directorates between April, 2023 and Nov., 2023. The subjects, aged ranged between 40 to 75 years, were divided into two groups: 50 patients with CAD and 50 apparently healthy as a control group. a SMART-120 chemistry analyzer was used to measure the levels of lipid profiles and other compounds in human serum. (colorimetric enzymatic method).

STATISTICAL ANALYSIS

Using (SPSS software, version 17.0), (SPSS Inc., Chicago, IL, USA), statistical analysis was carried out. Continuous variables were provided as mean \pm SD or as median and interquartile range in order to assess if the distribution was normal. The variables with a normal distribution that were examined included. Spearman's correlations were used to examine the relationship between the variables. at $p = 0.49$, the significance level was established.

RESULT

Clinical characteristics and metabolic parameters of the study participants are summarized in Table 1-1. The mean alkaline phosphatase (ALP) levels in patients (73.208 ± 22.704 U/I) were substantially higher than in controls (65.223 ± 20.408 U/I), with a P-value of 0.049, in a study comparing the patient and control groups. With a highly significant P-value of 0.000, total cholesterol (TC) levels were also higher in patients (221.447 ± 76.715 mg/ml) compared to controls (159.89 ± 68.597 mg/ml). Triglycerides (TG) revealed a significant difference, with a P-value of 0.000 and greater levels in patients (158.890 ± 68.597 mg/ml) than in controls (50.527 ± 36.492 mg/ml). With a P-value of 0.000, the patients' high-density lipoprotein cholesterol (HDL-C) was considerably lower than the controls' (49.140 ± 7.623 mg/ml) at 23.07 ± 7.774 mg/ml. With a P-value of 0.001, low-density lipoprotein cholesterol (LDL-C) was likewise greater in patients (173.647 ± 75.088 mg/ml) than in controls (134.860 ± 68.175 mg/ml). Last but not least, VLDL-C, or very-low-density lipoprotein cholesterol, was marginally elevated in patients (10.437 ± 7.2385 mg/ml) compared to controls (8.104 ± 4.541 mg/ml), with a P-value of 0.025.

Table1.1. Comparison of ALP level in patients and healthy groups with $p = 0.049$.

Parameter	Patients Mean \pm SD	Control Mean \pm SD	P-value
ALP U/I	73.208 \pm 22.704	65.223 \pm 20.408	0.049
TC mg/ml	221.447 \pm 76.715	159.89 \pm 68.597	0.000
TG mg/ml	158.890 \pm 68.597	50.527 \pm 36.492	0.000
HDL-C mg/ml	23.07 \pm 7.774	49.140 \pm 7.6233	0.000
LDL-C mg/ml	173.647 \pm 75.088	134.860 \pm 68.175	0.001
VLDL-C mg/ml	10.437 \pm 7.2385	8.104 \pm 4.541	0.025

DISCUSSION

Coronary heart disease (CHD) remains a leading cause of morbidity and mortality worldwide. It is driven by atherosclerosis, a process characterized by the buildup of fatty deposits in the coronary arteries, leading to reduced blood flow and oxygen supply to the heart. Numerous biomarkers have been studied to better understand the risk factors and mechanisms involved in CHD[7]. One such marker is alkaline phosphatase (ALP), an enzyme primarily associated with bone and liver function, but which also has significant implications for cardiovascular health. Recent studies have shown a potential relationship between elevated ALP levels and an increased risk of CHD, sparking interest in understanding the underlying mechanisms and clinical implications of this association[8]. Alkaline phosphatase is an enzyme that catalyzes the hydrolysis of phosphate esters in an alkaline environment. It is found in various tissues throughout the body, including the liver, bones, kidneys, and intestines. Clinically, ALP is often measured to evaluate liver function and bone disorders, as elevated levels may indicate cholestasis (blockage in bile flow) or increased bone turnover. However, recent evidence suggests that ALP may also play a role in vascular health, particularly in the process of vascular calcification[9],[10]. One of the key mechanisms linking elevated ALP levels with coronary heart disease is its role in promoting vascular calcification. Vascular calcification is a pathological process where calcium deposits form in the arterial walls, leading to the stiffening and narrowing of blood vessels. This process is a hallmark of atherosclerosis, which is the underlying cause of most CHD cases[11]. ALP facilitates calcification by hydrolyzing inorganic pyrophosphate, a potent inhibitor of calcium phosphate deposition, into phosphate. This increases the local concentration of phosphate, promoting the formation of hydroxyapatite crystals that deposit in the arterial walls. This mechanism is particularly relevant in patients with chronic kidney disease (CKD), where disturbed calcium-phosphate metabolism accelerates vascular calcification. However, elevated ALP levels have also been observed in individuals without CKD, suggesting that this enzyme plays a broader role in cardiovascular pathology[12]. Another potential link between ALP and CHD is inflammation. Chronic inflammation is a well-established risk factor for atherosclerosis and cardiovascular disease. Elevated ALP levels have been associated with markers of systemic inflammation, such as C-reactive protein (CRP). Inflammation contributes to endothelial dysfunction, a precursor to atherosclerosis, by promoting the expression of adhesion molecules that attract inflammatory cells to the arterial wall. These cells, particularly macrophages, contribute to the development of atherosclerotic plaques[13],[14]. Elevated ALP may be both a marker and a contributor to this inflammatory process. Studies have demonstrated that higher ALP levels are correlated with increased cardiovascular risk, even after adjusting for traditional risk factors such as cholesterol levels, hypertension, and smoking. This suggests that ALP may serve as an independent marker of inflammation and cardiovascular risk[15]. In addition to its role in vascular calcification and inflammation, ALP may also be linked to coronary heart disease

through its interaction with lipid metabolism. Dyslipidemia, characterized by elevated levels of low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG), is a major risk factor for CHD[16]. Elevated ALP levels have been associated with unfavorable lipid profiles, including higher LDL-C and TG levels and lower high-density lipoprotein cholesterol (HDL-C) levels. This suggests that ALP may influence lipid metabolism, contributing to the development of atherosclerosis. One study demonstrated that patients with coronary artery disease (CAD) had significantly higher ALP levels compared to healthy controls, along with elevated total cholesterol (TC), LDL-C, and TG levels, and decreased HDL-C levels. These findings support the hypothesis that elevated ALP is associated with both metabolic disturbances and increased cardiovascular risk[17],[18].

The association between elevated ALP levels and coronary heart disease has important implications for clinical practice [19]. First, measuring ALP levels could provide additional information for cardiovascular risk assessment, particularly in individuals with traditional risk factors such as dyslipidemia, hypertension, or a family history of heart disease. Patients with elevated ALP levels may benefit from more aggressive cardiovascular risk management, including lifestyle modifications and pharmacological interventions to reduce the risk of atherosclerosis and coronary events [20].

Second, future research may explore the potential of ALP as a therapeutic target [21]. For example, strategies aimed at reducing vascular calcification, such as inhibitors of ALP or modulators of phosphate metabolism, could be investigated as novel approaches to preventing or treating coronary heart disease.

CONCLUSION

In summary, elevated alkaline phosphatase (ALP) levels have been increasingly recognized as a potential marker of coronary heart disease risk. Through its role in promoting vascular calcification, contributing to inflammation, and influencing lipid metabolism, ALP may play a multifaceted role in the pathogenesis of atherosclerosis and CHD. While more research is needed to fully understand the mechanisms and clinical applications, ALP represents a promising avenue for improving cardiovascular risk assessment and developing new therapeutic strategies to combat coronary heart disease.

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CONFLICT OF INTEREST

All parties involved certify that there is none. The donors had no input with the creation of the lesson, the gathering, analysing, or interpreting of data, the drafting of the manuscript, or the making of decisions. Release the findings.

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