Article

Cardiovascular Disease and its Correlation with Myeloperoxidase Clinical Study for Babylon Patients Governorate

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Abstract

Background: Cardiovascular disease (CVD) is the general term used to describe diseases that affect the heart and circulatory system. Furthermore, it is believed that myeloperoxidase plays a part in the onset and progression of several heart conditions, the most prominent of which being atherosclerotic heart disease. It has been closely linked to several aspects of cardiovascular disease. Heart troponins are among the most crucial markers for a number of cardiovascular diseases, including acute myocardial infarction. Troponin's availability.

Objective: Evaluate the role of Myeloperoxidase in Cardiovascular Disease development.

Materials and methods: This research conducted a case-control study involving a total of 100 participants, with 50 individuals diagnosed with cardiovascular disease (CVD) and 50 apparently healthy individuals serving as the control group. Blood samples were collected to measure Myeloperoxidase, Cholesterol, Triglycerides, LDH, HDL and VLDL. In addition, other factors including age and BMI were measured. ROC-curve

analysis was one of the statistical techniques used to assess Myeloperoxidase's diagnostic accuracy.

Results: According to the results of the Myeloperoxidase ROC, the percent of area under the curve was 77%, and the current investigation discovered a significant rise in Myeloperoxidase concentration with the control group at p values significant ($p<0.05$).

Conclusion: The results emphasize that Myeloperoxidase play a crucial role in cardiovascular disease.

Keywords: Myeloperoxidase, Triglycerides, Cardiovascular disease (CVD).

Introduction

Any condition that affects the heart and circulatory system of the body is referred to as cardiovascular disease (CVD) or literally "disease of the heart and blood vessels." This includes peripheral vascular disease (PVD), which is any illness or disorder of the circulatory system that affects areas other than the heart and brain [1], hypertension (high blood pressure), arteriosclerosis, and stroke. Angina attacks (chest discomfort) or a more major heart attack can be brought on by cardiovascular disease's impact on the heart. A stroke might occur from CVD if it affects the brain; it could be a large stroke or a smaller one (also known as a TIA or transient ischemic attack). Many significant cardiovascular risk factors may be changed by changing one's lifestyle, adjusting one's social environment, or changing one's medication regimen. However, other risk factors, including age, sex, or genetic susceptibility or family history, cannot be changed (for example prevention of hypertension, hyperlipidemia, and diabetes). Atherosclerosis of the coronary arteries is more common in obese people [2].

The world's biggest cause of death is thought to be chronic cardiovascular illnesses, or CVDs. Myocardial infarction (MI), rheumatic heart disease, coronary heart disease (CHD), and stroke are the leading causes of CVD-related mortality [3]. Members of the hem peroxidase superfamily, which also includes lactoperoxidase (LPO) and eosinophil peroxidase (EPO), include the tetrameric, highly glycosylated peptidic enzyme

myeloperoxidase MPO (EC 1.11.1.7) [4]. Promyelocytes and promyelomonocytes in the bone marrow produce MPO during myeloid development, while fully developed myeloid cells stop producing MPO [5]. Serum MPO levels can be affected by age, gender, smoking status in males, and usage of birth control pills in women [6].

MPO is thought to have a role in the development and spread of several cardiac diseases, the most significant of which being atherosclerotic heart disease. It has been extensively associated with numerous facets of cardiovascular illness [7]. Myeloperoxidase produces a large number of diffusible radical species and reactive oxidants that can cause lipid peroxidation and a variety of post-translational changes to target proteins, such as oxidative cross-linking, nitration, and halogenation. LDL is changed into a highly absorbable state via lipid peroxidation and protein nitration, which is then readily absorbed by the macrophage scavenger receptor CD 367 [8].

Additionally, myeloperoxidase is known to catalyze the carbamylation of LDL, which turns it into a ligand for the SRA-18 scavenger receptor. Consequently, MPO produces several high-uptake variants of LDL that contribute to the development of atherosclerotic plaque. Additionally, it has been proposed that MPO alters apolipoprotein A-1, resulting in defective HDL (high-density lipoprotein) [9].

The most significant regulatory proteins in the troponin-tropomyosin complex, which is found on the actin (thin) myofilaments of cardiac myocytes, are cardiac troponin isoforms (cTnI, cTnT, and cTnC). The protein molecule cTnI functions as an inhibitory subunit, preventing the hydrolysis of adenosine triphosphate and actin-myosin interaction during the diastolic phase when calcium ions are not present [10]. The fact that slight variations in the amino acid sequence of the protein molecules cTnI, cTnT, and cTnC are linked to serious and potentially fatal impairments of the contractile function of the heart's muscular layer—known as hereditary cardiomyopathies—demonstrates the significance of cardiac troponins in the regulation of myocardial contractile function [11].

Materials and Methods

There were 100 participants in this case-control study , 50 of them had cardiovascular disease while the remaining 50 seemed to be in good condition. All of the samples were gathered during August and October of 2023. Samples were taken from the care unit in Babylon City, Iraq's Marjan and Imam Al-Sadiq Teaching Hospitals. The patients' ages varied from 40 to 60 year. Numerous details were recorded, including the patient's history, weight, height, sex, and age. Specialist physicians and cardiologists diagnosed

the patient groups based on selection and exclusion criteria, which included subjects with diabetes, hypertension, kidney and liver illness, obesity, thyroid disease, and smoking.

Every participant had a blood sample drawn from their vein. The patient's sample was then gently pushed into a gel tube, allowed to clot at room temperature for ten to fifteen minutes, then centrifuged at 3000 xg for ten minutes. The serum was extracted, placed in Eppendorf tubes, and the levels of MPO, cholesterol, triglycerides, LDH, HDL, and VLDL were measured.

Weight and height in kilos were measured, and the BMI was computed. Six milliliters of venous blood were extracted for biochemical examination. Among the biochemical assays were the MPO enzyme immunoassay, triglyceride, and lipid profile serum total cholesterol. While LDL was computed using the Friedwald formula, serum total cholesterol, HDL, and triglycerides were measured using a fully automated chemistry autoanalyzer, the Dimension-RXL. As directed by the makers (Bioassay Technology Laboratory), myeloperoxidase was measured using Enzyme Immunoassay (EIA), a "sandwich" ELISA kit.

Ethical approval:

All research participants were informed and given the opportunity to give verbal permission prior to sample collection. The study protocol, subject details, and consent form were reviewed and authorized by a local college and hospital ethics committee under the document number [IRB: 5-25,8/8/2023].

Statistical analysis:

For the statistical analysis, SPSS version 25 was employed. Categorical variables were represented by percentages and frequencies. For continuous variables, the format was (Means ± SD). The student t-test was used to compare the means of the two groups. The paired t-test was used to compare the means of the two paired readings. A p-value of less than 0.05 was deemed significant. Using a receiver operating characteristic (ROC) curve, the diagnostic utility of CKD was evaluated.

Results

*** The study subject's demographic characteristics:**

The association between the various characteristics of every patient and the mean difference between the control and cardiovascular disease groups were computed statistically using the t-test, based on the findings of the present comparative research between the patient and control groups. One hundred adult participants were split into two groups for the research (50).

Based on demographic data, Table 1 displays the results for the (50) patient group, whose ages varied from 40 to 60 years.

Table-1: Demographic characteristics of the study groups

Variable	Patients (Mean ± SD)	Control (Mean ± SD)	P-value
Age (years)	50.98 ± 6.26	49.33 ± 6.75	0.070
BMI (kg/m^2)	26.677 ± 1.86	26.418 ± 1.95	0.084
Number	50	50	
NS: Non-Significant, * (P≤0.05).			

Age

Results showed that the difference in p value was not statistically significant (0.07). Table 1 shows the age distribution of the rate of illness, with the mean of patients (50.98 ± 6.26) and the healthy control group (49.33 ± 6.75) differing accordingly. outcome that may be brought about by the wide age range. [12]

BMI

showed that BMI was not significantly difference in Control ($p \leq 0.05$) the mean and SD was (26.418 ± 1.95) compared to Patients of CVD the mean and SD was (26.677 ± 1.86) , as illustrated in table-1.

Table 2 compares several factors between study groups

Variable	N	Patients (Mean ± SD)	Control (Mean ± SD)	P-value
Myeloperoxidase	50	96.594 ± 48.14	55.938 ± 8.87	0.000
HDL (mg /dl)	50	40.60 ± 8.17	48.62 ± 6.95	0.000
LDL (mg /dl)	50	112.510 ± 48.51	114.882 ± 18.35	0.000
Vldl	50	41.883 ± 18.44	23.178 ± 8.71	0.000
Cholesterol	50	200.450 ± 61.21	186.680 ± 17.23	0.000
Triglycerides	50	236.700 ± 164.09	115.890 ±43.57	0.000
Troponin I	50	0.351 ± 0.20	0.316 ± 0.12	0.327
	*** (P≤0.05)**			

Table3: Correlation between variables

Study variables	Myeloperoxidase		Cardiac troponin1	
	R	**p-value**	**r**	**p-value**
Age	0.132	0.238	0.082	0.462
BMI	0.052	0.644	-0.129	0.249
Myeloperoxidase	-------	-------	0.091	0.433
Cholesterol	-0.254	**0.021**	-0.020	0.856
TG	-0.239	**0.030**	-0.016	0.890
HDL	0.185	0.097	0.129	0.246
LDL	-0.222	**0.045**	-0.031	0.785
vLDL	-0.295	**0.008**	-0.103	0.365

Table 4: ROC-curve analyses of Myeloperoxidase to predict patients with CVD.

AUC	Std. Error	Sig.	Specificity	Sensitivity	95% Confidence Interval	
					Lower Bound	Upper Bound
.7700	.0570	.0000	94%	73%	.6570	.8830
AUC= area under the curve Cut off point= 66.1						

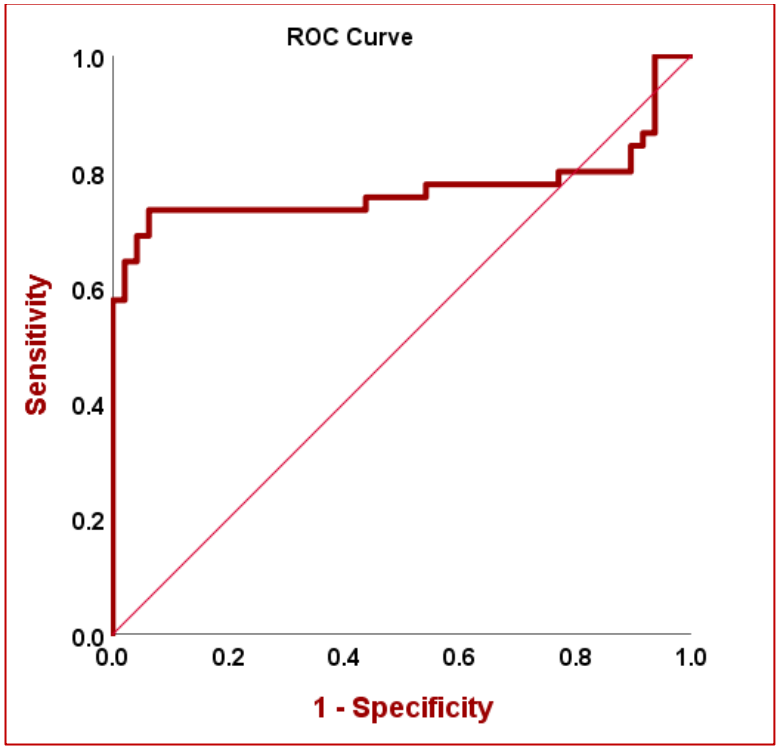

Figure-1: The curve of receiver operating characteristic (ROC) The group of patients' myeloperoxidases

Discussion

This study looked at using lipid profile to estimate myeloperoxidase in patients with CVD in Babylon Governorate. The main conclusions of this investigation were that the patients' myeloperoxidase and lipid profile levels were greater.

The current investigation found that plasma myeloperoxidase (MPO), an enzyme mostly generated and released by certain kinds of white blood cells, notably neutrophils and monocytes, was considerably greater in CVD patients than in healthy controls. Due to its involvement in the development of oxidative stress and inflammation, MPO is important in the pathophysiology of cardiovascular diseases (CVD) [13]. MPO increases the generation of highly reactive oxidants and reactive oxygen species (ROS), which worsen oxidative stress in the arteries and encourage lipid oxidation [14].
Atherosclerotic plaque development, inflammatory reactions, and endothelial dysfunction can all result from this oxidative stress [15]. Moreover, MPO has the ability to change lipoproteins into a more atherogenic state, particularly low-density lipoprotein (LDL). It is more likely for modified LDL to build up in the artery walls, which will cause an inflammatory reaction and accelerate the onset of atherosclerosis. In individuals with CVD, elevated MPO levels have been linked to a higher risk of cardiovascular events as well as unfavorable outcomes. [16]
The development of cardiovascular diseases (CVD), such as atherosclerosis, and the regulation of lipid metabolism have both been linked to myeloperoxidase (MPO). It has interactions with low-density lipoprotein (LDL), triglycerides (TG), cholesterol, and high-density lipoprotein (HDL) MPO and cholesterol are related in that MPO has the ability to alter LDL cholesterol, increasing its vulnerability to oxidative damage. Atherosclerosis is typified by foam cells, which are formed when macrophages absorb oxidized low-density lipoprotein (LDL) [17]. Triglycerides (TG) and MPO-generated oxidants are related. TG-generated oxidants aid in the oxidative modification of cholesterol and the formation of atherosclerotic plaques. Higher TG levels have been linked to elevated MPO levels. MPO-generated oxidants have the ability to cause TG-rich lipoproteins to oxidize. This results in the production of pro-inflammatory oxidized lipoproteins, which worsen endothelial dysfunction. Low-density lipoprotein (LDL) and MPO are connected in that MPO has the ability to oxidize LDL particles and transform them into pro-inflammatory particles. The accumulation of oxidized low-density lipoprotein (LDL) in artery walls increases the risk of atherosclerosis and inflammation. Moreover, his relationship to high-density lipoprotein (HDL) is that MPO can alter HDL particles, reducing their ability to protect [18]. HDL's capacity to stimulate reverse cholesterol transport—the mechanism by which extra cholesterol is moved from

peripheral tissues back to the liver for excretion—can be diminished by MPO-mediated oxidation of HDL [19, 20].

MPO affects lipoprotein function and lipid metabolism in a major way overall. Its capacity to alter HDL and oxidize LDL may have a role in the development of CVD and the advancement of atherosclerosis. Assessing the risk of CVD and directing treatment strategies can be aided by monitoring MPO levels and their correlation with lipid markers.

Troponin I did not change substantially between the control group and the CVD patient group ($p \leq 0.05$) in this research. Acute coronary syndrome (ACS) and myocardial infarction are two heart-related disorders that are largely diagnosed and assessed with the troponin I test (heart attack). When there is injury to the heart muscle, the blood's concentration of a protein called troponin I, which is present in cardiac muscle cells, can rise [21]. Troponin I level may or may not increase in cardiovascular disease (CVD), a general term that encompasses a number of disorders affecting the heart and blood vessels [22]. This variance can be attributed to the fact that CVD is a broad category of illnesses, each with unique processes and consequences on the heart muscle. For instance, because the heart muscle is still getting enough blood flow in stable coronary artery disease, even if there may be partial blockages in the heart's blood arteries, troponin I levels may stay within the normal range [23]. Troponin I level, however, are more likely to increase during bouts of unstable angina or a heart attack, where there is an abrupt and severe obstruction in the blood vessels [24].

The findings of the ROC analysis show a reasonable discriminative value; nevertheless, because of the small number of research participants, it cannot be regarded as a biomarker for the diagnosis of cardiovascular disease in patients.

Conclusion

This study showed that the lipid profile and myeloperoxidase levels were higher in patients with cardiovascular disease. Myeloperoxidase may also serve as a guidance for the markers used in follow-up and diagnosis.

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