International Journal of Biochemistry Research & Review

Volume 33, Issue 1, Page 21-30, 2024; Article no.IJBCRR.110398
ISSN: 2231-086X, NLM ID: 101654445

Plasma Lipid Profile and Apolipoproteins in Patients with Coronary Artery Diseases and Healthy Persons, in Sana'a City, Yemen

Khater Gh. H. AL-Hamoodi [a], Fairouz K. AL-Showafi [b], Saeed M. AL-Shaibani [b], Mohammad M. AL-Kebsi [b], Mubarak Ghaleb H. Al-hamodi [c], Tibyan Abd Almajed Altaher [d], Ghanem Mohammed Mahjaf [e], Waha Ismail Yahia Abdelmula [f] and Babbiker Mohammed Taher Gorish [f,g*]

[a] Biomedical Engineering, School of Life Science and Technololgy, Xi'an Jiaotong University, Xi'an, Shaanxi 710049.P.R., China.
[b] Department of Biochemistry and Molecular Biology, Faculty of medicine and Health Science, Sana'a University, Yemen.
[c] Department of Medicine and Surgery, Faculty of Medicine, Shendi University, Shendi, Sudan.
[d] Department of Clinical Chemistry, Faculty of Medical Laboratory Sciences, Shendi University, Sudan.
[e] Department of Medical Microbiology, Faculty of Medical Laboratory Sciences, Shendi University, Sudan.
[f] Biofuels Institute, School of Emergency Management, School of Environmental and Safety Engineering, Jiangsu University, Zhenjiang, Jiangsu 212013, China.
[g] Department of Medical Microbiology, Faculty of Medical Laboratory Sciences, Omdurman Islamic University, Sudan.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IJBCRR/2024/v33i1849

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/110398

**Corresponding author: E-mail: qorish456@gmail.com;*

Original Research Article

Received: 15/10/2023
Accepted: 21/12/2023
Published: 22/01/2024

ABSTRACT

Background: Cardiovascular disease (CVD) is the primary cause of all disease loads and accounts for almost half of all fatalities. The early detection of coronary artery disease (CAD) has drawn a lot of attention because it is anticipated that the global burden of cardiovascular disease will rise by almost 75% by 2020. They are easily oxidized, which causes an increase in affinity for arterial proteoglycans and a decrease in affinity for LDL receptors. It demonstrates significant CAD importance.

Objective: The measurement of plasma lipid profiles and apolipoproteins (ApoA-1, ApoB, and Apo B/A-1 ratio) in healthy individuals and patients suffering from coronary artery disease.

Methods: In this cross-sectional comparative study, 90 Yemeni subjects aged 45 to 70 years were divided into three groups: Group I: 30 cases as CAD positive (CAD+). Group II: 30 cases CAD negative (CAD -). Group III: 30 healthy person was as control. A standardized questionnaire was administered to collect demographic and clinical data from participants. Venous blood (10 ml) was collected from each individual and divided into two portions. The first portion was 5 ml in plain tubes, ApoA-1 and Apo B vacuum tubes for freezing at -20°C until analysis. 5ml in a plain tube for measuring fasting blood glucose, and lipid profile.

Results: ApoB and Apo B/A ratios were significantly higher in CAD+ and CAD− subjects compared to controls. In addition, ApoB and Apo B/A ratios were significantly higher in CAD+ subjects compared to CAD- subjects. (P. value =0.002). In contrast, Apo A-1 was significant in CAD+ compared to CAD- and controls, and not significantly different between CAD and controls P. value = (0.001, 0.032). Furthermore, FBS and LDL-c were significantly higher in CAD+ compared to CAD subjects (P value = 0.05). In contrast, HDL-c was significantly lower in CAD+ compared to CAD and controls, with no significant difference between CAD and controls (P value=0.038, 0.004, 0.70). However, TG was not substantially different between CAD- and controls, but it was significantly greater in CAD+ compared to controls (P. value =0.002, 0.09, 0.31). Nevertheless, there was no difference in TC between study groups (P. value = 0.08, 0.12, 0.98).

Conclusions: The degree of CAD is significantly positively correlated with WHR. More important than overall obesity, abdominal obesity is a risk factor for coronary heart disease (CHD). A significant positive connection has been observed between the Apo B/Apo A ratio and CAD. One thing to think about as a CAD risk factor is Apo B.

Keywords: Apolipoprotein; LDL; Cardiovascular disease; Yemen.

1. INTRODUCTION

"Cardiovascular disease (CVD) is responsible for nearly 50% of all deaths and is the leading cause of all disease burdens in Europe" [1]. "Much attention has been focused on the early prediction of coronary artery disease (CAD), as it was well-predicted that the global burden of cardiovascular disease would increase by almost 75% by 2020" [2]. "CAD is also known as ischemic heart disease (IHD)" [3]. "A group of diseases includes stable angina, unstable angina, myocardial infarction, and sudden cardiac death" [4]. "It belongs to the group of cardiovascular diseases and is the most common type among them" [5]. "In 2015, CAD affected 110 million people and killed 8.9 million" [6]. "It is the leading cause of death worldwide, accounting for 15.9% of all deaths. Especially in developed countries, the risk of death from CAD decreased at specific ages between 1980 and 2010" [7]. "It is present in 7% of 45-64-year-olds and 1.3% of 18-45-year-olds, with a higher proportion in men than women at any given age" [8]. "Coronary artery disease has several well-defined risk factors. The most common risk factors include smoking, family history, hypertension, obesity, diabetes, physical inactivity, stress, and elevated blood lipid levels" [9]. **"**Smoking is associated with approximately 36% of cases and obesity

with 20%" [10]. "Although some people have a genetic predisposition to develop atherosclerosis, it appears that most people can develop the disease, dietary fats, especially cholesterol, that are carried in the blood. High levels of LDL cholesterol in the blood can cause and exacerbate atherosclerosis. Other factors that contribute to atherosclerosis include smoking, hypertension, type 2 diabetes, age, sex, sedentary lifestyle, and obesity" [11]. "High blood cholesterol levels (especially serum LDL levels). HDL (high-density lipoprotein) has a protective effect against the development of coronary artery disease" [12]. "Plasma lipids, particularly cholesterol, and triglycerides have long been implicated in the pathogenesis of coronary artery disease (CAD)" [13,14,15,16,17]. "In vivo, water-insoluble cholesterol and other lipids form complexes with proteins (apoproteins) to form lipoproteins for transport and metabolism" [18,19]. "Lipoproteins are classified into five main types according to their size and density. These are exogenous and endogenous triglyceride-transporting chylomicrons and very low-density lipoproteins (VLDL). VLDL remnant - intermediate density lipoprotein (ILDL). The major cholesterol-transporting low-density lipoprotein (LDL). Hypothesized tissue cholesterol-scavenging high-density lipoprotein (HDL). Most epidemiological, experimental, clinical, and genetic studies have emphasized the role of elevated levels of LDL, or cholesterol contained in this lipoprotein fraction (LDL-C), in atherogenesis. Although the relatively high amount of cholesterol in the LDL fraction is generally thought to be an atherogenic factor, cholesterol in LDL in familial hypercholesterolemia or "familial" type 2 disease is associated with early CAD" [17,20,21,22]. "Seems to have a causal relationship with From Fredrickson et al. Proposed diagnostic criteria" [23]. "Type 2 disease, includes (1) Elevated LDL. (2) Enter 2 for first-degree relatives. Or (3) supple xanthomas. Moreover, these patients show no significant reduction in LDL-C levels on a standardized low-cholesterol diet" [24]. "More recently, dysfunction of specific LDL cell receptors was reported by Brown and Goldstein" [25]. "Familial type 2 patients. Given accelerated atherosclerosis in patients with well-defined metabolic abnormalities, lowering plasma LDL-C levels using effective therapeutic regimens may slow progression. Most people would agree that familial type 2 patients with CHD can be used to determine whether it is possible to induce regression. CAD to ameliorate angina, prevent myocardial infarction and reduce death from CAD. Recently, there has been an increasing interest in HDL" [26]. "Early observations showed that plasma alpha-lipoprotein (HDL) was lower in post-myocardial infarction patients than in healthy individuals. Suggesting that high HDL is an independent negative risk factor for CHD; epidemiological studies"; [27,28,29]. "Clinical correlative studies show that CHD patients with normolipidemic often have low HDL"; [30]. "Families with higher HDL levels live longer" [31]. "Some experimental data suggest that HDL may facilitate the removal of cholesterol from tissues" [32,33]. These observations made on LDL and HDL demonstrate that intracellular cholesterol can be regulated by developing intervention programs that can achieve significant reductions in atherogenic LDL and VLDL fractions. A program that increases the anti-atherosclerotic HDL fraction while reducing LDL and VLDL to normal levels would be highly desirable.

2. MATERIALS AND METHODS

2.1 Study Design

A hospital-related cross-sectional comparative study.

2.2 Study Area

Cardiac Center, Al Thawra and General Military Hospital (Referral Hospital), Sana'a City, Yemen.

2.3 Sample Size and Subjects

Sample size was 90, which calculated according to Pradeep, *et, al*. 2015. Using Open Epi program with 95% confidence level and mean ± SD of Apo B of 95.2 ± 74.7 cases and mean ± SD of controls of 25.3 ± 23.0 and a 2:1 case: control ratio using the Open Epi program. The validity is 80%. This study is a cross-sectional comparative study conducted between March 2018 and January 2019. Subjects were divided into three groups: CAD negative (CAD-) if no occlusion is detected by coronary angiography and healthy controls. Group I: 30 examples: CAD positive (CAD+). Group II: 30 cases of CAD negative (CAD -). Group III: 30 A healthy person is a control. Inclusion and exclusion criteria: Subjects were selected for coronary angiography based on one or more of the following criteria: chest pain, shortness of breath, and hypertension. A selected patient is considered CAD (+) if she has ≥50 stenosis in at least one of her coronary arteries.

2.4 Data collection and processing

Each participant must complete the questionnaire by providing their age, sex, weight, height, blood pressure, and waist circumference. Waist circumference is measured at the waist, halfway between the iliac crest and the rib arch. Using a tape measure, measure the man's waist at the top of his hipbone. After ten minutes of rest, the systolic and diastolic blood pressure is measured using a mercury sphygmomanometer. Type I or type II diabetes, metabolic syndrome (classified as high triglycerides, low HDL, small high-density LDL, or high non-HDL cholesterol), and smoking. Sample collection and processing: From a fasting patient, he draws 10ml of venous whole blood into a scheduled tube and separates the blood sample in the scheduled tube to obtain the serum. There are two components to the serum. (1) The first part is frozen at -20 °C until analysis of ApoA-1 and Apo B. (2) the second part measures fasting blood glucose and lipid profile.

2.5 Statistical analysis

All statistical analyzes were performed by the Social Package of Statistical Science (SPSS) 20.0 (LEAD Technologies; Inc. USA). Missing data were removed list by list. If any variable was missing, the entire observation was removed from the analysis. The significance of all parameters in the three groups was assessed by ANOVA (used to account for anthropometric and biochemical parameters). Except for Apo A, Apo B, and BAR values assessed by univariate analysis (general linear model) and adjusted for age. And BMI as a covariate. Association of Apo A, Apo B, and BAR with risk factors for CAD parameters. (BMI, waist, SBP, DBP, TG, T-C, HDL-c, LDL-c, and FBS (dependent variables) Correlations of Apo A, Apo B, and Apo B/Apo A-1 ratios across all participants were evaluated by linear regression controlling for age and weight. Were studied by linear regression adjusting for age and weight as covariates for all subjects. Mean differences were considered significant if the *P. value* was less than 0.05.

3. RESULTS

Anthropometric parameters by study group, during the analysis, waist, hip, and waist-to-hip ratio (WHR) were significantly higher in CAD+ and CAD− subjects compared with controls. Furthermore, waist and hip were significantly higher in CAD+ compared to CAD− subjects, and none were significantly higher in WHR. There were no significant differences between CAD and other groups. Nevertheless, there were no differences in age, weight, height, diastolic and systolic blood pressure between the study groups (Table 1). Biochemical parameters; fasting blood glucose, total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol between groups. Results showed that FBS and LDL-c were significantly higher in CAD+ and CAD− patients compared with controls. Furthermore, FBS and LDL-c were significantly higher in CAD+ subjects compared to CAD- subjects. In contrast, HDL-c was significantly lower in CAD+ compared to CAD and controls, with no significant difference between CAD and controls. On the other hand, TG was significantly higher in CAD+ compared to controls, not significantly higher compared to CAD−, and not significantly different between CAD and controls. However, there was no difference in TC between study groups (Table 2). Comparison of the ratios of apolipoprotein A-1, apolipoprotein B, and apolipoprotein B/A in all groups. The ratios of apolipoprotein A-1, apolipoprotein B, and apolipoprotein B/A between study groups. In general, ApoB and ApoB/A ratios were significantly higher in CAD+ and CAD− subjects compared to controls. In addition, ApoB and ApoB/A ratios were significantly higher in CAD+ compared to CAD- subjects. In contrast, Apo A-1 was significantly higher in CAD+ compared to CAD and control groups, with no significant difference between CAD and control groups (Table 3). Associations between ApoA-1, ApoB, and Apo B/A-1 ratios and coronary risk factor parameters. Apo B and Apo B/A-1 ratios were significantly positively correlated with WC, LDL-C, and FBS, and were independent of systolic and diastolic blood pressure. Furthermore, Apo B was significantly positively associated with BMI, HDL-C, TG, and total cholesterol. On the other hand, the Apo B/A-1 ratio was significantly negatively correlated with HDL-C, BMI, TG, and total cholesterol. However, there was no association between ApoA-1 and other parameters (Table 4).

The correlation between ApoA-1, ApoB, and Apo B/AI ratio in group studies. The ratios of apolipoprotein A-1, apolipoprotein B, and apolipoprotein B/A among the study groups. In general, the Apo B/A-1 ratio was significantly positively correlated between the Apo B/A-1 ratio and ApoB, and negatively significantly correlated with ApoA-1, whereas ApoB and ApoA-1 were significantly correlated (Table 5).

Table 1. Anthropometric parameters of control, negative and positive coronary artery disease

Parameters	Control	CAD^{-}	CAD^{+}
Age (years)	56.90 ± 6.283	56.87 ± 7.171	55.87 ± 6.699
P. value		[a]**1.0**	[a]**0.82,** [b]**0.83**
Weight (kg)	60.90 ±4.318	64.63±7.604	64.90±1 1.678
P. value		[a]**0.20**	**0.16 , 0.99**
Height (cm)	161.30 ± 6.396	162.33±5.909	158.57± 6.377
P. value		[a]**0.79**	[a]**0.21,** [b] **0.55**
Body Mass Index (kg/m^2)	23.52±2.511	24.62±3.393	25.73±3.796
P. value		[a]**0.40**	[a]**0.028,** [b]**0.39**
Waist Circumferences (cm)	79.47±5.015	90.23±7.486	95.03± 8.257
P. value		[a]**2.0×10^{-7}**	[a]**5.1×10^{-9}**, [b]**0.027**
Hip	78.23±5.263	88.20±7.170	92.50± 7.427
P. value		[a]**3.6×10^{-7}**	[a]**5.1×10^{-9}, [b]0.039**
Waist-to-Hip ratio	1.0134±.00694	1.020±.00643	1.0254±.01312
P. value		[a]**0.021**	[a]**1.0×10^{-5}, [b]0.069**
Diastolic Blood pressure (mmHg)	78.00±6.103	81.33 ±9.371	79.00± 8.449
P. value		[a]**0.25**	[a]**0.88,** [b]**0.50**
Systolic Blood Pressure (mmHg)	118.00±8.052	122.00±12.704	121.33± 12.521
P. value		[a]**0.36**	[a]**0.49 ,** [b]**0.97**

Table 2. Comparison of fasting blood glucose and lipid profiles of all groups

Parameters	Control	CAD-	CAD+
Fasting blood glucose (mg/dl)	85.67 ± 8.97	94.97 ± 12.66	104.13 ± 18.45
P. value		[a]**0.03**	[a]**5.0×10^{-14}**, [b]**0,033**
Triglyceride (mg/dl)	96.57 ±14.46	101.33±11.94	108.23 ± 11.22
P. value		[a]**0.31**	[a]**0.002,** [b]**0.09**
Total-Cholesterol (mg/dl)	112.53± 17.260	113.17±16.735	121.47± 13.68
P. value		[a]0.987	[a]0.08, [b]0.12
High Density Lipoprotein (mg/dl)	41.07±6.64	39.40±8.67	34.17±8.81
P. value		[a]**0.70,**	[a]**0.004,** [b]**0.038**
Low Density Lipoprotein (mg/dl)	59.07±8.00	64.67±9.73	74.80± 9.41
P. value		[a]**0.05**	[a]**1.0×10^{-8}**, [b]**1.2×10^{-16}**

Table 3. Comparison of apolipoprotein A-1, apolipoprotein B and apolipoprotein B/A ratio among in all groups

Parameters	control	CAD-	CAD+
ApoA-1 (mg/dl)	101 (96-106)	96 (96-101)	87 (82-92)
P. value		[a]**0.146,**	[a]**0.001,** [b]**0.028**
ApoB (mg/dl)	76 (70-82)	86 (80-91)	144 (138-150)
P. value		[a]**0.032**	[a]**9.5×10^{-27}**, [b]**1.3×10^{-23}**
ApoB/A-1 ratio	0.72 (0.65-0.70)	0.88 (0.81-0.94)	1.63 (1.56-1.69)
P. value		[a]**0.002**	[a]**3.6×10^{-31}, [b]1.5×10^{-26}**

Table 4. Association of ApoA, ApoB, and ApoB/A-1 ratios with risk factors for coronary parameters in study groups

Risk factors of CAD	ApoA-1 b(P-value)	ApoB b(P-value)	ApoB /A-1 ratio b(P-value)
Body Mass Index (kg/m^2)	-0.035 (0.70)	0.07**(0.002)**	-4.05 **(0.02)**
Waist circumference (cm)	0.024 (0.25)	0.84**(6.4×10^{-5})**	5.38**(5.9×10^{-7})**
Diastolic Blood Pressure (mmHg)	-0.37 (0.24)	0.26(0.54)	-23.4 (0.51)
Systolic Blood Pressure (mmHg)	-0.13 (0.95)	0.18(0.08)	-11.0 (0.17)
Triglyceride (mg/dl)	-0.18 (0.17)	0.17**(0.006)**	-5.85**(0.005)**
HDL- cholesterol (mg/dl)	-0.08 (0.23)	0.04**(0.002)**	-10.9**(0.001)**
LDL-cholesterol (mg/dl)	-0.03 (0.04)	0.06**(2.4×10^{-4})**	8.07**(4.5×10^{-7})**
Total – cholesterol(mg/dl)	-0.37 (0.16)	0.29 (**0.02**)	-17.6 (**0.01**)
Fasting Blood Sugar (mg/dl)	0.17 (0.61)	0.20(**2.6×10^{-8}**)	4.19 (**2.4×10^{-20}**)

Table 5. Correlation of ApoA-1, ApoB, and Apo B/AI ratio in group studies

Parameters	ApoA-1 r(p.value)	ApoB-r(p.value)	ApoB/A-1 ratior(p.value)
ApoA-1 (mg/dl)		-0.100- (0.175)	-0.517(**8.6×10^{-8}**)
ApoB (mg/dl)	-0.100- (0.175)		0.883(**5.0×10^{-31}**)

4. DISCUSSION

Our study aimed to determine the lipid profiles, ApoB, ApoA-1, and ApoB/A-1 ratios of coronary artery disease and healthy subjects. In the present study, waist, hip, and waist-to-hip ratio (WHR) were significantly higher in CAD+ and CAD− subjects than in controls. Obesity or overweight is known to promote or exacerbate all thermogenic risk factors that predispose individuals of all ages to coronary events. Abdominal fat accumulation as measured by WC or WHR is associated with metabolic and CHD risk, type 2 diabetes mellitus, hypertension, coronary artery disease, and stroke, and is more associated with abdominal obesity than with all-cause obesity is known, as measured using BMI. The current study showed that BMI was significantly higher in CAD+ compared to controls. This is consistent with previous results by Anand Sharma and workmates at 2014 [34]. However, our results are inconsistent with the study reported by Gregory and his colleagues at 2017 [35]. “There was no significant difference between BMI and CAD. This can be explained by the fact that BMI quantifies general obesity. Overweight or obese people may have excess fat, but BMI does not indicate how that fat is distributed throughout the body. However, fat distribution is an important determinant of CAD, independent of BMI and other classical risk factors for CAD” [36]. BMI is the most studied predictor of risk for obesity-related complications. Of note, some people within the normal BMI range may exhibit excessive central fat accumulation and increased metabolic risk, suggesting that central (visceral or intraperitoneal) obesity is more common than peripheral fat distribution is associated with the subsequent development of cardiovascular disease. [34]. “Since the central fat distribution is thought to be more atherogenic than peripheral fat, much attention has been focused on methods that can assess central fat depots” [34]. “In the current study, FBS was significantly higher in CAD+ and CAD− subjects compared to controls, a result consistent with the study reported by Nariman Moradi and her colleagues at 2018” [37]. In this study, significantly higher LDL-c levels were observed in CAD+ and CAD− patients compared to controls. Our funding yielded the same results as previously described [38]. LDL causes endothelial dysfunction through local inflammation and oxidative stress in the vessel wall, which leads to the attraction of monocytes from the blood and macrophages, and LDL infiltrates the intima-retained subendothelial space. Atherosclerosis occurs when oxidative LDL phagocytes by macrophages form a foamy matrix [39,40]. The present study showed that HDL-c was significantly lower in CAD+ compared to CAD- and normal subjects. The role of HDL in reverse cholesterol transport is probably most important in reducing plaque development [41]. In the present study, we observed that TG was significantly higher in CAD+ compared with normal subjects. Our funding mentioned the same result done by Pechlaner at 2017 with his colleagues [42]. TG can represent residual cholesterol levels [43]. And the smaller chylomicrons directly increase cholesterol accumulation as they penetrate the arterial wall [44]. These residual TRL particles directly contribute to plaque formation [45]. In this study, no differences in TC were found between study groups. Our funding yielded the same results mentioned Mashayekhi at 2014 [46]. In the current study, ApoB values and ApoB/A ratios were generally significantly higher in CAD+ and CAD− subjects compared with controls. Our funding is the same results mentioned by Hem 2014 [47]. ApoB is the major apolipoprotein of chylomicrons, VLDL, IDL, and LDL particles [48]. Furthermore, high apo B levels indicate increased risk, even though LDL-C or non-HDL-C levels typically remain low in severe atherogenic conditions such as metabolic syndrome and type 2 diabetes [49]. The Apo B/Apo A-I ratio represents the balance between Apo B-rich and Apo A-I-rich anti-atherogenic particles and is more predictive of cardiovascular risk than lipid, lipoprotein, and lipid ratios [50]. Apolipoproteins may be more informative risk markers than lipoproteins (such as LDL and HDL) [49]. In particular, the ratio of apolipoprotein B to apolipoprotein A-I (apoB/apoA-I) [51,52,53]. In the current study, apo A-1 was observed to be significantly higher in CAD+ compared to CAD and control studies. Apolipoproteins A-I and B/A-I are significantly higher in CAD+ than in CAD-. Apo B and the Apo B/A-1 ratio were significantly positively associated with WC, LDL-C, and FBS in a series of studies. Higher values of WC, LDL-C, and

FBS were associated with a higher risk in CAD. We demonstrate the importance of the ratio of Apo B to ApoA-1 as a predictive marker. The same results are mentioned by Sheriff at 2013 and his colleagues [38]. Another finding was a significant positive association between Apo B and BMI, HDL-C, TG, and total cholesterol. The same results are mentioned by (Anand, 2014, Hem, 2014, and Pechlaner, 2017). [34,47,42]. Concentrations of lipid parameters can vary with diet. However, apolipoprotein levels are not affected by diet. Therefore, fasting blood samples are not required for apolipoprotein measurements. HDL cholesterol can lead to misleading results because HDL cholesterol composition can vary in response to different physiological and pathological conditions. Therefore, measuring the protein fraction of HDL, Apo A1, is a better predictor of CAD [54,55].

5. CONCLUSIONS

The degree of CAD is significantly positively correlated with WHR. More important than overall obesity, abdominal obesity is a risk factor for coronary heart disease (CHD). A significant positive connection has been observed between the Apo B/Apo A ratio and CAD. One thing to think about as a CAD risk factor is Apo B.

CONSENT AND ETHICAL APPROVAL

Ethical approval for the study was obtained by the Ethics Committee of the Sana'a University School of Medicine and Health Sciences. The written informed consent form was obtained from each guardian of the participant as well as from the subject himself before recruitment into the study. All protocols in this study were done according to the Declaration of Helsinki (1964).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. European cardiovascular disease statistics. cardiovascular Disease Statistics. 2010; (2):7683. Available:http://www.heartstats.org/datapage.asp?id=7683, 2 November.
2. Desai CS, Blumenthal RS, Greenland P. Screening low-risk individuals for coronary artery disease. Curr Atheroscler Rep. 2014;16(4):402. DOI: 10.1007/s11883-014-0402-8. PMID: 24522859.
3. Bhatia SK. Biomaterials for clinical applications. springer; 2014.
4. Wong ND. Epidemiological studies of chd and the evolution of preventive cardiology. Nat Rev Cardiol. 2014;11(5):276-89. DOI: 10.1038/nrcardio.2014.26. Epub 2014 Mar 25. PMID: 24663092.
5. GBD Mortality and Causes of Death Collaborators. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2015;10;385(9963): 117-71. DOI: 10.1016/S0140-6736(14)61682-2. Epub 2014 Dec 18. PMID: 25530442; PMCID: PMC4340604.
6. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, et al. Gbd-nhlbi-jacc global burden of cardiovascular diseases writing group. global burden of cardiovascular diseases and risk factors, 1990-2019: Update From the GBD 2019 Study. J Am Coll Cardiol. 2020;22;76(25):2982-3021. DOI: 10.1016/j.jacc.2020.11.010. Erratum in: J Am Coll Cardiol. 2021 Apr 20;77(15):1958-1959. PMID: 33309175; PMCID: PMC7755038.
7. Moran AE, Forouzanfar MH, Roth GA, Mensah GA, Ezzati M, Flaxman A, Murray CJ, Naghavi M. The global burden of ischemic heart disease in 1990 and 2010: the global burden of disease 2010 study. Circulation. 2014;8;129(14): 1493-501. DOI:10.1161/CIRCULATIONAHA.113.004046. Epub 2014 Feb 26. PMID: 24573351; PMCID: PMC4181601.
8. Centers for Disease Control and Prevention (CDC). Prevalence of coronary heart disease--united states, 2006-2010. MMWR Morb Mortal Wkly Rep. 2011;14;60(40):1377-81. PMID: 21993341.
9. Dai X, Wiernek S, Evans JP, Runge MS. Genetics of coronary artery disease and myocardial infarction. World J Cardiol. 2016;26;8(1):1-23. DOI: 10.4330/wjc.v8.i1.1. PMID: 26839654; PMCID: PMC4728103.
10. Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT; Lancet Physical Activity Series Working Group. Effect of physical inactivity on major non-

communicable diseases worldwide: an analysis of burden of disease and life expectancy. Lancet. 2012;21;380(9838): 219-29.
DOI: 10.1016/S0140-6736(12)61031-9. PMID: 22818936; PMCID: PMC3645500.

11. Kannel WB. Overview of atherosclerosis. Clin Ther. 1998;20. Suppl B:B2-17.
DOI: 10.1016/s0149-2918(98)80027-1. PMID: 9589828.
12. Ressee J, Britton R. General and Systematic Pathology; 2004.
13. Kannel WB, Castelli WP, Gordon T, McNamara PM. Serum cholesterol, lipoproteins, and the risk of coronary heart disease. The Framingham study. Ann Intern Med. 1971;74(1):1-12.
DOI: 10.7326/0003-4819-74-1-1. PMID: 5539274.
14. Carlson LA, Böttiger LE. Ischaemic heart-disease in relation to fasting values of plasma triglycerides and cholesterol. Stockholm prospective study. Lancet. 1972;22;1(7756):865-8.
DOI: 10.1016/s0140-6736(72)90738-6. PMID: 4111826.
15. Albrink MJ, Meigs JW, Man EB. Serum lipids, hypertension and coronary artery disease. Am J Med. 1961;31:4-23.
DOI:10.1016/0002-9343(61)90220-0. PMID: 13682175.
16. Brown DF, Kingh SH, Doyle JT. Serum triglycerides in health and in ischemic heart disease. N Engl J Med. 1965; 273:947.
17. Goldstein JL, Hazzard WR, Schrott HG et al. Hyperlipidemia in coronary heart disease: I. Lipid levels in 500 survivors of myocardial infarction; II. Genetic analysis of lipid levels in 176 families and delineation of a new inherited disorder, combined hyperlipidemia; and III. Evaluation of lipoprotein phenotypes of 156 genetically defined survivors of myocardial infarction. J Clin Invest. 1973;52:1533-1577
18. Gofman JW, Young W, Tandy R. Ischemic heart disease, atherosclerosis and longevity. Circulation. 1966;34:679
19. Fredrickson DS, Levy RI, Lees RS. Fat transport in lipoproteins–an integrated approach to mechanisms and disorders.n engl j med. 1967;276(148, 215, 273): 34-94.
20. Slack J. Risks of ischemic heart-disease in familial hyperlipoproteinemic states. lancet. 1969;2:1380.
21. Jensen J, Blankenhorn D. The inheritance of familial hypercholesterolemia. Am J Med. 1972;52:499.
22. Stone NJ, Levy RI, Fredrickson DS, Verter J. Coronary artery disease in 116 kindred with familial type ihyperlipoproteinemia. circulation. 1974;49:476.
23. Fredrickson DS, Goldstein JL, Brown MS. The familial hyperlipoproteinemias. in: Stanbury JB Wyngaarden JB Fredrickson DS The metabolic basis of inherited diseases. 4. McGraw Hill, New York. 1978:604-655.
24. American Heart Association, Planning fat-controlled meals for approximately 2000-2600 calories. American Heart Association Inc, New York;1967.
25. Brown MS, Goldstein JL. Familial hypercholesterolemia: A genetic defect in the low-density lipoprotein receptor. N Engl J Med. 1976;294:1386.
26. Barr DP, Russ EM, Eder HA. Protein lipid relationships in human plasma: ii. in atherosclerosis and related conditions.am j med. 1951;11:480
27. Rhoads G, Gulbrandsen CL, Kagan A. Serum lipoproteins and coronary heart disease in a population study of Hawaii Japanese men.N Engl J Med. 1976;294:293.
28. Castelli W, Doyle JT, Gordon T.et al. HDL cholesterol and other lipids in coronary heart disease, the cooperative lipoprotein phenotyping study. circulation. 1977;55:767.
29. Miller NE, Thelle DS, Forde OH, Mjos OD. The Tromsö heart-study, high-density lipoprotein and coronary heart disease:a prospective case-control study. Lancet. 1977;1:965
30. Miller GJ, Miller NE. Plasma-high-density-lipoprotein concentration and development of ischemic heart-disease. Lancet. 1975;1:16.
31. Glueck CJ, Fallat RW, Millett F, et al. Familial hyperalpha-lipoproteinemia: studies in 18 kindreds. Metabolism. 1975;24:1243.
32. Carew TE, Hayes SB, Koschinsky T, Steinberg D. A mechanism by which high-density lipoproteins may slow the atherogenic process.lancet. 1976;1: 1315.
33. Glomset JA. The plasma lecithin: cholesterol acyltransferase reaction.J Lipid Res. 1968;9:155.
34. Kaur S, Sharma A, Singh H. Waist-related anthropometric measures: simple and

useful predictors of coronary heart disease in women. National Journal of Physiology, Pharmacy and Pharmacology. 2015; 5(1):60.

35. Gregory AB, Lester KK, Gregory DM, Twells LK, Midodzi WK, Pearce NJ. The Relationship between Body Mass Index and the Severity of Coronary Artery Disease in Patients Referred for Coronary Angiography. Cardiol Res Pract. 2017; 2017:5481671
DOI: 10.1155/2017/5481671. Epub 2017 Apr 23. PMID: 28512592; PMCID: PMC5420422.
36. Després JP. CVD risk assessment: do we need the metabolic syndrome or better global cardiometabolic risk calculators? Int J Obes (Lond). 2008;32 Suppl 2:S1-4.
DOI: 10.1038/ijo.2008.27. PMID: 18469833.
37. Moradi N, Fadaei R, Emamgholipour S, Kazemian E, Panahi G, Vahedi S, Saed L, Fallah S. Association of circulating CTRP9 with soluble adhesion molecules and inflammatory markers in patients with type 2 diabetes mellitus and coronary artery disease. PLoS One. 2018;30;13(1): e0192159.
DOI:10.1371/journal.pone.0192159. PMID: 29381773; PMCID: PMC5790264.
38. De Asmundis, Riccardo (ed.). Modeling, Programming and Simulations Using LabVIEWTM Software. BoD–Books on Demand; 2011.
39. Libby P, Ridker PM, Hansson GK. Progress and challenges in translating the biology of atherosclerosis. Nature. 2011 May 19;473(7347):317-25.
DOI:10.1038/nature10146. PMID: 21593864.
40. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation. 2002;5;105(9):1135-43.
DOI: 10.1161/hc0902.104353. PMID: 11877368.
41. Fisher EA, Feig JE, Hewing B, Hazen SL, Smith JD. High-density lipoprotein function, dysfunction, and reverse cholesterol transport. Arterioscler Thromb Vasc Biol. 2012;32(12):2813-20.
DOI:10.1161/ATVBAHA.112.300133.
PMID: 23152494; PMCID: PMC3501261.
42. Pechlaner R, Tsimikas S, Yin X, Willeit P, Baig F, Santer P, Oberhollenzer F, Egger G, Witztum JL, Alexander VJ, Willeit J, Kiechl S, Mayr M. Very-Low-Density Lipoprotein-Associated Apolipoproteins Predict Cardiovascular Events and Are Lowered by Inhibition of APOC-III. J Am Coll Cardiol. 2017;21;69(7):789-800.
DOI: 10.1016/j.jacc.2016.11.065. PMID: 28209220; PMCID: PMC5314136.
43. Varbo A, Benn M, Tybjærg-Hansen A, Jørgensen AB, Frikke-Schmidt R, Nordestgaard BG. Remnant cholesterol as a causal risk factor for ischemic heart disease. J Am Coll Cardiol. 2013;29;61(4): 427-436.
DOI: 10.1016/j.jacc.2012.08.1026. Epub 2012 Dec 19. Erratum in: J Am Coll Cardiol. 2019 Mar 5;73(8):987-988. PMID: 23265341.
44. Rapp JH, Lespine A, Hamilton RL, Colyvas N, Chaumeton AH, Tweedie-Hardman J, Kotite L, Kunitake ST, Havel RJ, Kane JP. Triglyceride-rich lipoproteins isolated by selected-affinity anti-apolipoprotein B immunosorption from human atherosclerotic plaque. Arterioscler Thromb. 1994;14(11):1767-74.
DOI: 10.1161/01.atv.14.11.1767. PMID: 7947602.
45. Alaupovic P, Mack WJ, Knight-Gibson C, Hodis HN. The role of triglyceride-rich lipoprotein families in the progression of atherosclerotic lesions as determined by sequential coronary angiography from a controlled clinical trial. Arterioscler Thromb Vasc Biol. 1997;17(4):715-22.
DOI: 10.1161/01.atv.17.4.715. PMID: 9108785.
46. Mashayekhi NR, Sadrnia S, Chehrei A, Javaheri J. The correlation between serum apoa1 and b and coronary artery disease as well as its severity. Int Cardiovasc Res J. 2014;8(1):1-5. Epub 2014 Jan 1. PMID: 24757643; PMCID: PMC3987460.
47. Tamang HK, Timilsina U, Singh KP, Shrestha S, Raman RK, Panta P, Karna P, Khadka L, Dahal C. Apo B/Apo A-I Ratio is Statistically A Better Predictor of Cardiovascular Disease (CVD) than Conventional Lipid Profile: A Study from Kathmandu Valley, Nepal. J Clin Diagn Res. 2014;8(2):34-6.
DOI: 10.7860/JCDR/2014/7588.4000. Epub 2014 Feb 3. PMID: 24701475; PMCID: PMC3972591.
48. Davidson MH. Apolipoprotein measurements: is more widespread use clinically indicated? Clin Cardiol. 2009; 32(9):482-6.
DOI: 10.1002/clc.20559. PMID: 19743499; PMCID: PMC6653425.
49. Sniderman AD, Jungner I, Holme I, Aastveit A, Walldius G. Errors that result

from using the TC/HDL C ratio rather than the apoB/apoA-I ratio to identify the lipoprotein-related risk of vascular disease. J Intern Med. 2006;259(5):455-61.
DOI: 10.1111/j.1365-2796.2006.01649.x. Erratum in: J Intern Med. 2006 Aug;260(2):186. Junger, I [corrected to Jungner, I]. PMID: 16629851.

50. Lima LM, Carvalho Md, Sabino Ade P, Mota AP, Fernandes AP, Sousa MO. Apo b/apo a-i ratio in central and peripheral arterial diseases. Arq Bras Endocrinol Metabol. 2007;51(7):1160-5.
DOI: 10.1590/s0004-27302007000700020. PMID: 18157393.
51. Walldius G, Jungner I. The apob/apoa-i ratio: A strong, new risk factor for cardiovascular disease and a target for lipid-lowering therapy--a review of the evidence. J Intern Med. 2006;259(5): 493-519.
DOI: 10.1111/j.1365-2796.2006.01643.x. PMID: 16629855.
52. McQueen MJ, Hawken S, Wang X, Ounpuu S, Sniderman A, Probstfield J, Steyn K, Sanderson JE, Hasani M, Volkova E, Kazmi K, Yusuf S; INTERHEART study investigators. Lipids, lipoproteins, and apolipoproteins as risk markers of myocardial infarction in 52 countries (the INTERHEART study): A case-control study. Lancet. 2008; 372(9634):224-33.
DOI: 10.1016/S0140-6736(08)61076-4. PMID: 18640459.
53. Kastelein JJ, van der Steeg WA, Holme I, Gaffney M, Cater NB, Barter P, Deedwania P, Olsson AG, Boekholdt SM, Demicco DA, Szarek M, LaRosa JC, Pedersen TR, Grundy SM; TNT Study Group; IDEAL Study Group. Lipids, apolipoproteins, and their ratios in relation to cardiovascular events with statin treatment. Circulation. 2008;117(23):3002-9.
DOI: 10.1161/CIRCULATIONAHA.107.713438. Epub 2008 Jun 2. PMID: 18519851.
54. Ashmaig, Mohmed, et al. Levels of apolipoproteins as risk factors for coronary artery disease. Jornal Vascular Brasileiro, 2011;10:293-297.
55. As S, Sahukar S, Murthy J, Kumar K. A study of serum apolipoprotein A1, apolipoprotein B and lipid profile in stroke. J Clin Diagn Res. 2013;7(7):1303-6.
DOI: 10.7860/JCDR/2013/5269.3123. Epub 2013 Jul 1. PMID: 23998051; PMCID: PMC3749621.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/110398