**Association ofPlasma Lipid Profile and Apolipoprotein with Coronary Artery Diseases, inSana'a City, Yemen**


**Abstract:**

***Background*:**Cardiovasculardisease (CVD) accounts for nearly 50% of all deathsand is the leading cause of all disease burdens. With the global burden of cardiovascular disease predicted to increase by nearly 75% by 2020, much attention has been focused on the early prediction of coronary artery disease (CAD). They are readily oxidized, resulting in decreased affinity for LDL receptors and increased affinity for arterial proteoglycans. It shows important relevance to CAD. ***Objective:*** To determine plasma lipid profiles and apolipoproteins (ApoA-1, ApoB, and Apo B/A-1 ratio) in 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. : CAD positive (CAD+). Group II: 30 cases of CAD negative (CAD -). Group III: 30 healthy person is 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). Was significantly higher in CAD+ and CAD-subjects compared to controls. 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). On the other hand, TG was significantly higher in CAD+ compared to controls, and not significantly different between CAD- and 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:***Thereisasignificantpositivecorrelationbetween WHRand CAD severity.Abdominalobesityisariskfactorfor CHD andismorerelevantthan general obesity.ThereisasignificantpositivecorrelationbetweentheApo B/Apo AratioandCAD. ApoBis a factor to consider asariskfactorforCAD
***Keywords:***Apolipoprotein,LDL, Cardiovascular disease, Yemen.

**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.

**Materials and methods:**

**Study design:**

A hospital-related cross-sectional comparative study.

**Study area:**

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

**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. She works under the supervision of a cardiology center doctor. A selected patient is considered CAD(+) if she has ≥50 stenosis in at least one of her coronary arteries. Exclusion criteria were patients with the polycystic ovarian disease, taking liver, and taking oral statins.

**Data collection and processing:**

The questionnaire is filled out by filling out the following information (age, gender, weight, height, blood pressure, waist circumference) for each participant. Waist circumference is measured midway between the rib arch and the iliac crest and is measured at the waist. Measure the waist with a tape measure at the top of the man's hipbone. Systolic and diastolic blood pressure are measured with a mercury

sphygmomanometer after 10 minutes of rest. Diabetes (type I or type II), 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. Serum is divided into two parts. (1) The first part is frozen at -20 °C until analysis of ApoA-1 and Apo B. (2) the second part is used to measure fasting blood glucose and lipid profile.

**Statistical analysis:**

All statistical analyzes were performed by the Social Package of Statistical Science (SPSS) 20.0 (LEAD Technologies; Inc. USA). Missing data was 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) were analyzed by linear regression adjusting for age and weight as covariates for all subjects. Correlations of Apo A, Apo B, and Apo B/Apo A-1 ratios among all subjects were assessed by linear regression controlling for age and weight. Mean differences were considered significant if the *P.value* was less than 0.05.

**Ethical approval and consent**:

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).

## 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]$5.0\times10^{-14}$, [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]$1.0\times10^{-8}$, [b]$1.2\times10^{-16}$** |

**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]$9.5\times10^{-27}$, [b]$1.3\times10^{-23}$** |
| **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]$3.6\times10^{-31}$,[b]$1.5\times10^{-26}$** |

**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)** | **ApoBb(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**($6.4\times10^{-5}$)** | 5.38**($5.9\times10^{-7}$)** |
| **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**($2.4\times10^{-4}$)** | 8.07**($4.5\times10^{-7}$)** |
| **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(**$2.6\times10^{-8}$**) | 4.19 (**$2.4\times10^{-20}$**) |

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

| **Parameters** | **ApoA-1r(p.value)** | **ApoB-r(p.value)** | **ApoB/A-1 ratior(p.value)** |
|---|---|---|---|
| **ApoA-1 (mg/dl)** | | -0.100- (0.175) | -0.517(**$8.6\times10^{-8}$**) |
| **ApoB (mg/dl)** | -0.100- (0.175) | | 0.883(**$5.0\times10^{-31}$**) |

**Discussion:**

Ourstudyaimedtodetermine the lipidprofiles,ApoB,ApoA-1,andApoB/A-1 ratios of coronaryartery disease andhealthy subjects. Inthepresentstudy,waist,hip,andwaist-to-hipratio(WHR)weresignificantlyhigherinCAD+and CAD− subjects than in controls. Obesity or overweight isknown to promote or exacerbate allthermogenicriskfactors that predispose individuals ofallagestocoronaryevents.Abdominalfat accumulation asmeasuredbyWCor WHR isassociatedwithmetabolicand CHD risk,type2diabetesmellitus,hypertension,coronaryarterydisease,andstroke,and is more associated withabdominal obesity thanwith all-cause obesity is known, asmeasured using BMI.The current studyshowed that BMI was significantlyhigherinCAD+comparedto 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 nosignificant difference betweenBMIandCAD.This can beexplainedbythefactthatBMIquantifiesgeneral obesity. Overweight orobese people may haveexcessfat, but BMIdoesnotindicatehow that fatisdistributed throughout thebody.However,fatdistributionisanimportantdeterminantofCAD,independentofBMIand other classical riskfactorsforCAD**[36].**BMI isthemost studied predictorofrisk for obesity-

related complications. Of note, somepeoplewithinthenormalBMIrangemay exhibit excessivecentralfataccumulationand increased metabolic risk, suggesting thatcentral(visceralor intraperitoneal) obesity is more common thanperipheralfat distribution is associated with the subsequentdevelopmentofcardiovascular disease. **[34].**Since the centralfatdistributionis thought to be moreatherogenicthanperipheral fat, muchattentionhasbeenfocusedonmethodsthatcan assess central fat depots **[34].**Inthe current study,FBSwassignificantlyhigherinCAD+and CAD− subjectscomparedto controls, a result consistent with the study reportedbyNariman Moradi and her colleagues at 2018**[37].**In this study,significantlyhigher LDL-c levels were observed inCAD+and CAD− patients comparedto controls.Ourfunding yielded thesameresults as previously described (Sheriff, et.al. 2013)**[38].**LDLcausesendothelialdysfunction through localinflammationandoxidative stress in the vessel wall, which leads to the attraction of monocytes from the bloodandmacrophages,andLDLinfiltratesthe intima-retained subendothelial space.Atherosclerosis occurs when oxidative LDLphagocytesbymacrophagesform a foamy matrix**[39,40].**Thepresent study showed that HDL-cwassignificantlylowerinCAD+comparedtoCAD-andnormal subjects. The role of HDLinreversecholesteroltransport is probably mostimportantin reducing plaquedevelopment**[41].**Inthepresentstudy,weobserved that TGwassignificantlyhigherinCAD+compared with normalsubjects. Our funding mentioned thesameresult done byPechlaner at 2017 with his colleagues**[42].**TG can represent residual cholesterol levels**[43].**And the smallerchylomicrons directly increase cholesterol accumulation as they penetrate thearterialwall**[44].**These residual TRLparticles directly contributetoplaqueformation **[45].**In this study,no differences inTC were found between study groups. Ourfunding yielded thesameresultsmentionedMashayekhi at2014**[46].**Inthe current study, ApoB values and ApoB/A ratios were generally significantlyhigherinCAD+and CAD− subjectscompared with controls. Our funding is the same results mentioned by Hem2014**[47].**ApoBisthe major

apolipoproteinofchylomicrons,VLDL,IDL,andLDLparticles**[48].**Furthermore,highapoB levels indicate increased risk, even though LDL-Cornon-HDL-Clevels typically remain lowin severe atherogenic conditions suchasmetabolicsyndromeandtype2diabetes**[49].**The

Apo B/Apo A-Iratiorepresentsthebalancebetween Apo B-rich and Apo A-I-rich anti-atherogenic particles andis more predictive ofcardiovascularriskthan lipid, lipoprotein, andlipidratios**[50].**Apolipoproteinsmaybemoreinformativeriskmarkersthanlipoproteins (such as LDLandHDL)**[49].** In particular, theratio of apolipoproteinB to apolipoproteinA-I(apoB/apoA-I)**[51,52,53].** Inthe current study, apo A-1was observed to be significantlyhigherinCAD+comparedto CAD and control studies. ApolipoproteinsA-IandB/A-I are significantlyhigherinCAD+than in CAD-.ApoBand the ApoB/A-1 ratio weresignificantlypositivelyassociatedwithWC, LDL-C, andFBS in a series ofstudies. Higher values of WC,LDL-C,andFBSwere associated with a higher risk inCAD. We demonstrate theimportanceof the ratio of ApoB to ApoA-1asapredictivemarker. The sameresults are mentionedbySheriffat2013 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, andPechlaner,2017).**[34,47,42].**Concentrations oflipidparameterscan vary with diet. However, apolipoprotein levels arenotaffectedby diet. Therefore, fastingblood samples are not required for apolipoprotein measurements. HDLcholesterol can lead to misleading results because HDL cholesterolcompositioncanvaryinresponseto different physiologicalandpathologicalconditions. Therefore, measuring the protein fraction of HDL, ApoA1,isabetterpredictorofCAD**[54].**

**Conclusions:**

ThereisasignificantpositivecorrelationbetweenWHRand CAD severity.Abdominalobesityisariskfactorfor CHD andismorerelevantthan general obesity.ThereisasignificantpositivecorrelationbetweentheApo B/Apo AratioandCAD. ApoBis a factor to consider asariskfactorforCAD.

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