Study of Lipid Profile and it's Risk Factor AIP with BMI Division for CVD Patients Compared to Healthy Control

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Abstract

This study was conducted in Ibn- Albetar specialized Hospital for Cardiovascular Disease / Baghdad from December 2011 to April 2012.
Sera of 71 person of both sex aged (33-67) years, include 51 patients diagnosed with Cardiovascular disease (CVD), and 20 healthy volunteers, were used to estimate some biochemical parameters which include lipid profile (TCh,TG,HDL,LDL,VLDL) and CRP. The risk factor Atherogenic Index of Plasma (AIP) was calculated.

None of the control were atherosclerotic vascular disease, diabetic, renal disease, history of allergy, alcoholic consumption and heavy smoker.
Sera of the samples were divided into:

1-Control group: includes (20) healthy (both sex), ages range (25-63) years, with no previous diseases which may interfere with the parameters analyzed in this study.
2-Patients with ages ranged (33-67) years were divided into three groups according to Body Mass Index (BMI):
a- Group (1): includes (11) patients (both sex) with BMI (20-24.9) Kg/m².
b- Group (2): includes (28) patients (both sex) with BMI (2-29.9) Kg/m².
c- Group (3): includes (12) patients (both sex) with BMI (30 to over) Kg/m².

The data of this study reveled a significant increase in the levels of TCh, TG, LDL, VLDL and AIP value, while a significant decrease in HDL level in sera of CVD patients compared to control. Also CRP showed significant increase in CVD patients groups as compared to control group. For BMI the results showed a significant increase in CVD for G2 and G3 while G1 showed a significant decrease as compared to control group.

These results showed a significant correlation between the risk factor AIP and the parameters studied in CVD.

Key words : CVD ,AIB ,CRP and BMI

INTRODUCTION

Cardiovascular disease (CVD) is a class of disease that involves the heart or blood vessels (arteries, capillaries and veins) [(1)]. The causes of cardiovascular disease are diverse but atherosclerosis and /or hypertension are the most common. The term Acute Coronary Syndrome (ACS) is a general term used to describe the following continuum of events: angina, reversible tissue injury, unstable angina and Myocardial Infarction (MI) [(2)].

It is well established that atherosclerosis is an inflammatory disorder, not a cholesterol issue, as there are many mechanisms that lead to the cellular injury in atherosclerosis, such as bacterial infection, hyperlipidemia, glycosylated products seen in diabetes mellitus ,and proinflammatory cytokines, among others[(3, 4)].

Lipids and lipoproteins, which are central to the energy metabolism of the body, have become increasingly important in clinical practice primarily because of their association with Coronary Heart Disease (CHD). [(5)]

The Atherogenic Index of Plasma (AIP) defined as log (TG / HDL) has been proposed as a marker of plasma atherogenicity, and calculated as log(TG/HDL) (6). Value of AIP corresponds closely to those of esterification rate in apo-B-lipoprotein – depleted plasma and to lipoprotein particle size. AIP reflects the delicate metabolic interactions within the whole lipoprotein complex[(7)].

The present study was conducted with an aim to study the atherogenic index and various lipid levels of plasma in Acute Myocardial Infarction (AMI) and to compare lipid Profile and Atherogenic Index of plasma in the patients of Myocardial Infarction with healthy controls according to body mass index (BMI).

C-Reactive Protein (CRP) is protein found in the blood, the levels of which rise in response to inflammation (i.e. C-reactive protein is an acute-phase protein). [(8)]

The Body Mass Index (BMI), or Quetelet index, is defined as the individual's body mass divided by the square of his or her height. It is a heuristic proxy for human body fat based on an individual's weight and height. BMI does not actually measure the percentage of body fat.[(9)]

Laboratory work

1- Collection of Blood

Ten ml of venous blood was placed in a plain tube (no anticoagulant), which was taken from the groups attending, left for (15 min) at room temperature , then centrifuged (at 2500 rpm for 10 min) to get the serum , which was stored about (-20°C) unless used immediately.

2- Sampling (Subjects)

The samples were collected from cardiovascular patients whom ages ranged between (33 – 67) years, at the diagnosis time at (Ibn Al-Betar Specialized Hospital/ Baghdad) during the period from December 2011 to April 2012.

None of the control were atherosclerotic vascular disease, diabetic, renal disease, history of allergy, alcoholic consumption and heavy smoker.

The samples have been classified into four groups as follow :

1-Control group: includes (20) healthy (both sex), ages range (25-63) years, with no previous diseases which may interfere with the parameters analyzed in this study.

2- Patients group were divided into three groups according to BMI :

a- Group (1): includes (11) patients (both sex) with BMI (20-24.9) Kg/m^2.

b- Group (2): includes (28) patients (both sex) with BMI (25-29.9) Kg/m^2.

c- Group (3): includes (12) patients (both sex) with BMI (30 to over) Kg/m^2.

3- Procedures :-

The lipid fractions were assayed as follow:

- Serum total cholesterol was measured by enzymatic colorimetric method described by Allain et.al.(1974) [10], using kit supplied by BioLabo-France.

- Serum Triglycerides (TG) was measured by enzymatic method using kit supplied by BioMerieux-Franc. (Tietz,1995[11] , Fossati,1982[12] and Trinde,1969[13])

-Serum HDL-Cholesterol was measured enzymaticaly using kit supplied by BioMerieux- France (Tiezt,1999). [14]

- LDL- Cholesterol was calculated mathematically by the equation (Bostom AG, 1996) [15].

LDL mmol /L = Total cholesterol – (VLDL + HDL-cholesterol)

- VLDL-cholesterol was calculated by the following equation (Friedewald, 1982) [16].

VLDL in mmol/L = Triacylglycerols / 2.2

-The Atherogenic Index of Plasma (AIP), was calculated as log (TG/HDL-C), with TG and HDL-C expressed in molar concentrations[6]. (AIP was calculated by using the Czech online calculator of atherogenic risk.)

-C-Reactive Protein (CRP) was investigated qualitatively using Latex, which is a slide agglutination test .(Lars- OlofHanson et.al.,1997).[17]

Statistical analysis

Data presented were the means ± standard deviations, student's t-test was used to compare the significance of the difference in the mean values of any two groups. ($P \leq 0.05$) was considered statistically significant[18].
The overall predictive values for the results in all studied groups was performed according to program of office EXCEL 2007.

Results and Discussion

T-Cholesterol and TG

The results of T-Cholesterol and TG in sera of the studied groups are shown in the appendix. The level of TCh for control group was (4.01 ± 0.30) mmol/L and for CVD patients groups (G1, G2, G3) were (4.46 ± 0.61) mmol/L, (4.89 ±1.25) mmol/L and (4.5±1.65) mmol/L respectively. The levels of TG for control group was (1.14±0.29) mmol/L and for CVD patients groups were G1 (1.71 ±0.87) mmol/L, G2 (1.80 ± 0.62) mmol/L and G3 (1.33 ± 0.41) mmol/L. These results showed significant increase in TCh and TG for CVD patients groups (G1, G2, G3) compared to control group, yet the patients groups were within the normal range.

Studies have shown that an elevated concentration of total cholesterol in the blood is a powerful risk factor for coronary disease[19]. It is often found in hypertension. It therefore follows that a reduction in plasma total cholesterol level which will reduce the risk of cardiovascular disease. Authors goal was to assess the magnitude of association and the quality of supporting evidence linking hypertriglyceridemia with cardiovascular events[20]. High plasma concentrations of triglyceride is both an independent and synergistic risk factor for cardiovascular disease[21,22] and is often found in hypertension, abnormal lipoprotein metabolism, obesity, insulin resistance and diabetes mellitus[23]. High BMI and high TG levels correlate in metabolic syndrome, which may involve, not only low HDL, but more inflammatory HDL as well[24].
The increased risk of cardiovascular disease which is associated with metabolic syndrome has many causes, but dyslipidemia plays a prominent role in it[25].

HDL, LDL and VLDL

The results of HDL, LDL and VLDL in sera of the studied groups are shown in the appendix. The level of HDL for control group was (1.26 ± 0.14) mmol/L and for CVD patients groups were G1 (0.9 ± 0.14) mmol/L, G2 (1.0 ± 0.20) mmol/L and G3 (1.1 ± 0.14) mmol/L. The level of LDL for control group was (2.1±0.33) mmol/L and for CVD patients groups were G1 (2.7 ± 0.28) mmol/L, G2 (3.7 ± 0.90)mmol/L and G3 (4.23 ± 2.02)

mmol/L. The level of VLDL for control group was (0.55±0.17) mmol/L and for CVD patients groups were G1 (0.76 ± 0.37) mmol/L, G2 (0.81 ± 0.28) mmol/L and G3 (0.60± 0.18) mmol/L. These results showed a significant increase in LDL and VLDL for CVD for G1, G2 and G3 compared to control group. While a significant decrease in HDL for CVD for G1, G2 and G3 compared to control group. Yet all patients groups were within the normal range.

Interest in high-density lipoproteins (HDL) is growing due to findings linking diabetes and atherosclerosis to the formation of dysfunctional HDL. Inflammatory index of HDL measures one aspect of HDL's many purported atheroprotective properties. HDL is a complex molecule that changes its structure and composition with small changes in the environment, even slightly altered diets can produce different HDL molecules[26]. From an epidemiologic viewpoint, normal or high HDL cholesterol levels have been noted to fail to protect against vascular disease by an absent inverse relation between HDL cholesterol and coronary heart disease (CHD) incidence in subjects at high risk[27], and a systematic review disclosed that no association existed between treatment-induced change in HDL-cholesterol and risk ratios for cardiovascular disease morbidity and mortality, when changes in low-density lipoprotein (LDL)-cholesterol were adjusted for [28]. In dyslipidemic patients, normal levels of HDL-cholesterol were not associated inversely, but rather positively (albeit insignificantly) with elevated gamma-glutamyl transferase (GGT) activity[29], recognized to reflect generation of reactive oxygen species. HDL-cholesterol levels did not significantly predict newly developing cardiovascular disease in diabetic Iranian men and women, nor in non-diabetic females[30]. Cardiovascular disease is one of the world's leading causes of death. One of the major risk factors for the development of cardiovascular disease is dyslipidemia, which may be primary or associated with hypertension, diabetes mellitus and obesity[23]. Dyslipidemia usually involve elevated plasma levels of triglyceride, cholesterol, LDL and VLDL cholesterol and a low level of HDL cholesterol. Dyslipidaemia is a known as a major risk factor for CVD in DM[31]. Lipid abnormalities were found in a large number of our patients. Total cholesterol, TG levels and TG/HDL ratio were significantly higher and HDL cholesterol levels significantly lower in patients with CVD compared with the non-CVD group. This result is similar to other study reported a dyslipidaemia prevalence of 77.7% in type2 DM[32] .

High serum LDL cholesterol values are well-known risk factors for atherosclerotic cardiovascular disease[33]. Decrease in plasma LDL

cholesterol have been considered to reduce risk of coronary heart disease[23].

Among patients with diabetes mellitus, a high prevalence of coronary heart disease is observed at a relatively young age[34]. Thus, risk factors for atherosclerosis must be defined and avoided in patients with diabetes mellitus. The low density lipoprotein (LDL) cholesterol level, which is a major risk factor for atherosclerosis, can be lowered by lifestyle modifications and by the use of statins, which reduces the risk of cardiovascular events among diabetic patients. Concomitantly with LDL cholesterol-lowering therapy, the effect of other cardiovascular risk factors was enhanced[35]. Researchers and numerous confirmatory studies have demonstrated a strong inverse correlation between high-density lipoprotein (HDL) level and cardiovascular disease risk. Despite aggressive lowering of low-density lipoprotein (LDL), low HDL remains a significant cardiovascular risk factor in high-risk patients[36]. Furthermore, the most recent trial of cholesteryl ester transfers protein inhibition raised HDL levels by more than 70% but resulted in significant harm[37]. Additionally, mutations in HDL metabolism or structure result in varying levels of HDL that do not correlate with atherosclerotic risk as predicted by these researchers , and modifications to HDL that occur in various disease statues seem to attenuate its atheroprotective effect[38,39]. Lipid profile consists of a group of biochemical tests often used in predicting, diagnosing and treating lipid- related disorders including atherosclerosis. Generally, the hyperlipidemias are of interest to the physician in the context of risk factors for ischemic heart disease (IHD) and peripheral vascular disease. The first step in diagnosis of hyper- and hypolipoproteinaemias is to define the lipoprotein pattern by chemical analysis of the plasma lipids and lipoproteins. Abundant evidence has accumulated relating of the concentrations of lipids (total cholesterol and triglycerides) and their associated blood transporting lipoproteins (HDL, LDL, VLDL) with the occurrence of atherosclerosis in general and coronary artery disease CAD in particular. The strong association between the risk of coronary artery disease CAD, high levels of LDL and low levels of HDL has been well established[40]. However the enormous contributions of triglycerides TG to cardiovascular risk have been underestimated especially in our environment. Indeed high levels have been associated with an increased incidence of CAD and an increased population of small dense population of small dense LDL particles has lot of work which has been done on the relationship between TG and HDL-C, and it has been shown that the ratio of TG to HDL was a strong predictor of myocardial infarction[41].

Abnormality of lipids such as high triglyceride levels[42] and low HDL levels[43] emerged as residual cardiovascular risks for diabetic patients.

The TG/HDL ratio was calculated for assessment of the level of dense LDL, a relatively novel lipoprotein index that could serve as a good predictor of CHD[44,45].

Increased in plasma HDL have been considered to reduce risk in coronary heart disease[46]. High HDL exerts a protective effect by enhancing reverse cholesterol transport by scavenging excess cholesterol from peripheral tissues, which it esterifies with the aid of lecithin cholesterol acyltransferase, and delivers to the liver and steroidogenic organs for subsequent synthesis of bile acids, lipoproteins, and eventual elimination from the body[19], inhibiting the oxidation of LDL as well as the atherogenic effects of oxidized LDL by virtue of its antioxidant[47] and anti-inflammatory property[48].

Data are emerging that atherogenic dyslipidemia can reduce endogenous ant inflammatory pathways mediated by HDL and amplify proinflammatory actions of VLDL[49].

High plasma concentrations of LDL and VLDL is a risk factor for cardiovascular disease[19] and is often found in diabetes mellitus[23] hypertension and obesity[50]. Decreases in plasma LDL have been considered to reduce risk of coronary heart disease[46]. Clinical data show that there is an increase in plasma HDL concentration decrease cardiovascular risk[23].

AIP

The results of AIP in sera of the groups studied are shown in the appendix.

The value of AIP for control group was (0.04 ± 0.046) and for CVD patients groups were G1 (0.37 ± 0.11), G2 (0.23 ± 0.09) and G3 (0.16 ± 0.07). These results showed a significant increase in AIP for CVD for G1, G2 and G3 as compared to control group. The risk values of AIP are :

AIP< 0.11 low risk

AIP (0.11-0.21) intermediate risk

AIP > 0.21 increased risk

A number of lipid related parameters have been used to predict the risk of coronary artery disease (CAD). According to Grover[51], either the ratio of LDL/HDL or TG/HDL is the best related predictor of future cardiovascular events. Later, TG/HDL was shown to be a more accurate predictor of coronary heart disease. The logarithamatically transformed ratio of plasma TGL to HDL correlated closely with the LDL particle size and could serve as an indicator of the atherogenic lipoprotein phenotype[51].

The appendix depicts the cardiovascular risk in all studied groups. A significant increase was seen in the TG/HDL ratio and in the AIP when compared to the LDL/HDL ratio in the type 2 diabetic patients with metabolic syndrome when compared with the type 2 diabetic patients without metabolic syndrome[52].

Rather than the lipid profile, the TC/HDL ratio and AIP showed higher efficiency in indicating the atherogenic lipid abnormalities.

The TC/HDL ratio has the best discriminatory power to indicate the atherogenic lipid abnormalities.

Coronary artery disease CAD is one of the major health problems which are responsible for the increasing mortality and morbidity. The total cholesterol TC/HDL and the LDL/HDL molar ratios have a good predictive value for future cardiovascular events. Dobiasova and Frohlich proposed a term Atherogenic Index of Plasma(AIP), which was defined as log (TG/HDL), an indication that plasma atherogenicity was also a significant independent predictor of CHD[53].

HDL facilitates the efflux of cholesterol from cells such as macrophages, promoting reverse cholesterol transport. A recent study showed that cholesterol efflux capacity, or the ability of HDL to promote reverse cholesterol transport from macrophages, correlated with atherosclerosis, independent of HDL mass[54]. Numerous additional functions have been ascribed to HDL, including roles in countering inflammation, oxidation, platelet activation, and promoting endothelial health. Many cell-based assays have been developed to measure HDL function, but the high-density lipoprotein inflammatory index (HII) is a measure of how well HDL can prevent the oxidation of LDL in a cell-free environment. A few, mostly small, studies have suggested that patients with underlying inflammatory conditions tend to have HDL that has less anti-inflammatory capacity as measured by the HII[55,56].

Indeed, HDL /LDL ratio has been of great value in the assessment of cardiovascular risk especially when the absolute values of the individual lipoproteins seem normal. Thus, the use of other indexes which has been minimally applied should be encouraged. Isolated elevation in triglycerides increases CHD risk more in women than men, but its effect Isolated elevation in triglycerides increases CHD risk more in women than men, but its effect can be counteracted by the levels of HDL. The atherogenic index of plasma which is a mathematical relationship between TG and HDL has been successfully used as an additional index when assessing cardiovascular CV risk factors[57]. It has been demonstrated that the development of CAD is a function of the particle size of LDL and HDL,

with the small particle size of both HDL and LDL exhibiting great atherogenic potential (particle size exhibiting great atherogenic potential). Indeed, cholesterol esterification rate in HDL plasma (FERHDL) has a strong relationship between lipoprotein particle sizes and thus can be considered as a functional risk marker for CAD. More recently, researchers have shown that the log arithmetically transformed ratio TG/HDL is the best determinant for FERHDL and thus a better predictor of cardiovascular risk than other previously used lipid parameters[58] Furthermore, in situations where other atherogenic risk parameters appear normal, AIP may be the diagnostic alternative[57].

Low atherogenic indices are protective against coronary heart disease[59]. In conclusion, results of the study suggest a possible protective role of the extract against the development of atherosclerosis and coronary heart disease, as well as the dyslipidemic conditions that characterize diabetes mellitus, hypertension, metabolic syndrome and obesity. It also suggests that some lower in lipid extract can help manage the dyslipidemic conditions[60] that accompany the administration of thiazide diuretics. Atherogenic index of plasma indicates a balance between the actual concentration of plasma TG and HDL, which predetermine the direction of the cholesterol transport in an intravascular pool i.e. the flux of newly produced cholesteryl esters by lecithin cholesterol acyltransferase (LCAT) towards atherogenic LDLs or beneficial HDLs. Clinical studies have shown that AIP predicts cardiovascular risk and that it is an easily available cardiovascular risk marker and a useful measure of the response to treatment. The atherogenic index of plasma AIP (log TG/HDL) is a good marker for cardiovascular risk. Atherogenic Index of Plasma [log (TG/HDL-C)] as markers of lipoprotein particle size, was examined in relation to biomarkers and conventional risk factors[52]. In multivariate analysis BMI was the strongest correlate of AIP levels[61].

It have been proposed the term AIP, on the basis that people with high AIP have a higher risk for CHD than those with low AIP, that AIP is positively correlated with the fractional esterification rate of HDL (FERHDL), and that AIP is inversely correlated with LDL particle size which in turn predicts CHD risk, the simultaneous use of TGs and HDL (both readily available in a plasma lipoprotein profile) as AIP may be useful in predicting plasma atherogenicity.

Log (TG/HDL-C) ratio was taken as an index of LDL particle size. However, with subsequent research, it was revealed that LDL particles are heterogeneous in nature. LDL particles containing less cholesterol and increasing triglyceride content are small and dense in nature. Small dense

LDL particles are highly atherogenic. These particles possess an increased readiness for oxidation due to their lower antioxidant content, stimulate the endothelial dysfunction and are easily recognized by scavenger receptors leading to foam cell formation[62]. The significance of small dense LDL particles in CAD patients with normal lipid and lipoprotein cholesterol levels is not well covered in the literature even though many individuals suffering from coronary artery disease do have a normal lipid profile. It have been evaluated the nature of LDL particles in CAD patients with normal LDL cholesterol levels. Normolipidemic CAD patients had higher positive values of log (TG/HDL) as compared to healthy subjects. This ratio is taken as an index of LDL particle size. Log(TG/HDL) ratio has been reported to range from negative to positive with a zero value corresponding to LDL particle diameter of 25.5nm. Small dense LDL particles can penetrate the arterial wall easily and are more prone to oxidation than normal LDL particles. Increased predominance of small dense LDL particles in CAD patients has also been reported by other researchers[63].

The cascade of atherosclerosis commences with the formation of oxidized LDL, leading to coronary artery disease and indicates the importance of estimating LDL oxidation status in patients with normal lipid and lipoprotein cholesterol levels. Log (TG/HDL) ratio is a simple index which could efficiently predict the nature of LDL particles even in normolipidemics[64].

Atherogenic indices are powerful indicators of the risk of heart disease the higher the value, the higher the risk of developing cardiovascular disease and vice versa[21]. The Atherogenic Index and various lipid levels of plasma are in Acute Myocardial Infarction (AMI), and to compare lipid profile and Atherogenic Index of plasma in the patients of Myocardial Infarction with healthy control.

Serum cholesterol, TG, VLDL, LDL and AIP were significantly higher in AMI patients as compared to controls. Atherogenic Index of plasma is a very useful research tool to assess the effect of risk factors pertaining cardiovascular diseases. While HDL was significantly lower in AMI patients as compared to controls[65].AIP may be an important tool for analyzing the results of clinical trials. The association of TGs and HDL in this simple ratio theoretically reflects the balance between risk and protective lipoprotein forces, and both TGs and HDL are widely measured and available.

Since Gaziano et al (1997). reported that "the ratio of triglycerides to HDL was a strong predictor of myocardial infarction" additional findings have been made regarding the relationship between HDL

It is difficult to fully appreciate the complexity of the lipoprotein phenotype or changes in the lipoprotein profile induced by therapeutic interventions. Perhaps changes in the rate of enzyme reactions such as cholesterol esterification may help us better to understand this complexity. The principle of this test is the measurement of the rate of cholesterol esterification (FER_{HDL}) by lecithin cholesterol acyltransferase (LCAT) in plasma containing only HDLs.

Total cholesterol (TC), and the various subfractions; high density lipoprotein cholesterol (HDL-C), very low density lipoprotein cholesterol (VLDL-C), low density lipoprotein cholesterol (LDL-C) and triglycerides (TG) were determined, Atherogenic index of plasma (AIP); log (log (TG/HDL-C) was calculated. AIP which can easily be calculated from standard lipid profile can act as an adjunct that significantly adds predictive value beyond that of the individual lipids, and/or TC/HDL, LDL/HDL ratios[57].

C-Reactive Protein

The results of C-RP in sera of the groups studied are shown in the appendix.

Average risk: 1.0-3.0 mg/L

C-reactive protein (CRP), an inflammatory marker, is well known to be increased in inflammatory conditions. CRP was recommended to measure as the risk for atherosclerosis. It was the strongest determinant of AIP among women ,independent of body mass index. High AIP, a surrogate of small LDL particle size, reflects obesity and hyperinsulinemia in men and high CRP status in women. It predicts CHD independently, type 2 diabetes mediated by obesity in men and in women[66].

A high TG/HDL ratio alone was predictive of CHD risk only in men, whereas combined presence of high TG/HDL ratio and elevated CRP conferred CHD risk in women. The interplay across gender, hypertriglyceridemia, elevated CRP and HDL dysfunction in the development of cardio metabolic risk sex the joint role of the pro-inflammatory state, represented by elevated circulating TG, CRP and HDL dysfunction[67].

Acute myocardial infarction (AMI) triggers an inflammatory reaction, which plays an important role in myocardial injury . Inflammatory markers such as C-reactive protein (CRP) reflect the extent of myocardial necrosis and correlate with cardiac outcomes following AMI[68].

High C-reactive protein (CRP) levels have been associated with higher mortality rate in patients with acute myocardial infarction (AMI). However, it is not known whether inflammation plays a role in the time-course of heart failure (HF) in this clinical setting[69].

Recent research suggests that patients with elevated basal levels of CRP are at an increased risk of diabetes [70] hypertension and cardiovascular disease. Although one group of researchers indicated that CRP may be only a moderate risk factor for cardiovascular disease. Others have shown that CRP can exacerbate ischemic necrosis in a complement-dependent fashion and that CRP inhibition can be a safe and effective therapy for myocardial and cerebral infarcts; so far, this has been demonstrated in animal models only[71].

To clarify whether CRP is a bystander or active participant in atherogenesis, a 2008 study compared people with various genetic CRP variants. Those with a high CRP due to genetic variation had no increased risk of cardiovascular disease compared to those with a normal or low CRP[72]. A study published in 2011 shows that CRP is associated with lipid responses to low-fat and high-polyunsaturated fat diets[73].

Arterial damage results from white blood cell invasion and inflammation within the wall. CRP is a general marker for inflammation and infection, so it can be used as a very rough proxy for heart disease risk. Since many things can cause elevated CRP, this is not a very specific prognostic indicator[74].

BMI

The results of BMI in sera of the groups studied are shown in the appendix. The BMI for control group was (25.1 ± 0.30) kg/m² and for CVD patients groups were G1 (23.02 ± 1.51) kg/m², G2 (27.34±1.51) kg/m² and G3 (32.99 ± 3.17) kg/m². This study a showed significant increase in BMI for CVD for G2 and G3 compared to control group while a significant decrease was in G1.

While a BMI classification system has been developed and used extensively to place adults in weight categories based on health risk, a recent report by the World Health Organization concluded that, where possible, abdominal obesity should also be measured and used in conjunction with BMI to assess and predict CVD risk[75]. This may have affected the degree to which measures of abdominal obesity are associated with CVD risk factors within BMI categories, particularly, normal-weight and obese. Using logistic regression models, CVD risk factors were examined in relation to the health risk categories for the adiposity variables. Regardless of the variable, people in the increased/high health

risk categories generally had higher odds of having CVD risk factors. Based on nationally representative data, the results of their study provide evidence that measures of BMI and abdominal obesity are associated with increased prevalence of CVD risk factors, moreover, within certain BMI categories.

In the earlier Canadian study, sample size was insufficient to consider waist circumference (WC) among normal-weight women. However, by 2007-2009, close to one quarter of normal-weight women were classified at increased/high health risk based on their WC, [76] and results from the current study show that WC is now associated with increases in CVD risk factors for normal-weight women.

The heart case-control study, which had the benefit of a very large sample size (12,000 myocardial infarction cases and 15,000 controls), found that the risk of myocardial infarction was more strongly associated with waist-to-hip ratio (WHR) than with WC [77].

The high correlation between BMI and WC (more than 0.9) in this study suggests that BMI is an excellent proxy in assessing obesity-related CVD risk when it is not feasible to measure abdominal obesity. The current analysis indicates that BMI is associated with increases in CVD risk factors, and therefore, can be used to measure obesity-related health risk in the context of population-based surveys. The study also supports the Canadian clinical practice guideline that, in addition to BMI[78] obesity, which is reported to play a key role in the pathogenesis of metabolic syndrome, promotes inflammation, hypertension and dyslipidaemia, thus leading to the development of type 2 diabetes mellitus and atherosclerosis[79]. Moreover, a higher blood pressure (BP) is a strong risk factor for cardiovascular disease (CVD). Abdominal fat which is associated with central obesity, a characteristic feature of the metabolic syndrome, is a major source of the excessive flux of free fatty acids which are known to have pro-arrhythmic parameters.

Metabolic syndrome is a combination of medical disorders that increase the risk of developing cardiovascular disease and diabetes.

Central obesity is the key feature of this syndrome, reflecting the fact that the syndrome's prevalence is driven by the strong relationship between waist circumference and increasing adiposity[80].

Research indicates morbidly obese individuals suffer from serious co-morbidities with nearly half (48%) diagnosed with hypertension, 29% diagnosed with diabetes, and 25% diagnosed with heart failure[81].

Treating many of these conditions is difficult because morbidly obese individuals are often physically unable to avail themselves of modern

diagnostic medicine because of their size, especially when attempting to be treated for cardiovascular diseases[82]. A noteworthy finding in this study is the effect of moderate physical activity (PA) had on BMI among those in the "obese class III" BMI classification, also known as the "morbidly obese". Part of the challenge in addressing PA for the morbidly obese is overcoming barriers endemic to physical conditions of obese individuals. One such barrier is the perception of breathlessness associated with exercise[83].This barrier can prevent obese individuals from beginning an exercise regimen. However, there is an encouraging evidence that training programs incorporating respiratory muscle development can be successful in helping obese individuals reach their PA goals, improve their metabolic health, and sustain their level of exercise. In addition, mobility limitations of obese individuals caused by diseases such as osteoarthritis and joint pain, must be taken into account when planning appropriate PA programs[84]. Another barrier to PA is motivation, particularly because obese individuals often report a lack of energy or feeling too tired to exercise[85].

These are all unique barriers to PA that programs must recognize in order to be successful.

Future studies would benefit from incorporating individual disease variables as well as individual values of lipoprotein lipase (LPL), low-density lipoprotein cholesterol (LDL-C), blood sugar, and other biometric markers where appropriate.

Appendix showing all the results of this study for patients groups (G1,G2,G3)and control

Subject	No.	TCh (mmol/L)	P	TG (mmol/L)	P	HDL (mmol/L)	P	LDL (mmol/L)	P	VLDL (mmol/L)	P	AIP	P	CRP	BMI (Kg/m^2)	P
Control	20	4.01± 0.30	—	1.14±0.29	—	1.26± 0.14	—	2.1±0.33	—	0.55±0.17	—	0.04± 0.046	—	-ve	25.1± 0.30	—
G1	11	4.46 ± 0.61	≤0.05	1.71±0.87	≤0.05	0.9 ± 0.14	≤ 0.05	2.7±0.28	≤0.05	0.76±0.37	≤0.05	0.37 ± 0.11	≤0.05	+ve	23.02 ± 1.51	≤0.05
G2	28	4.89±1.25	≤0.05	1.80±0.62	≤0.05	1.0±0.20	≤0.05	3.7±0.90	≤0.05	0.81±0.28	≤0.05	0.23±0.09	≤0.05	+ve	27.34±1.51	≤0.05
G3	12	4.5±1.65	≤0.05	1.33±0.41	≤0.05	1.1±0.14	≤0.05	4.23±2.02	≤0.05	0.60±0.18	≤0.05	0.16±0.07	≤0.05	+ve	32.99±3.17	≤0.05

Relationship and correlation coefficient

Relationship between AIP and TCh

Figures (1a), (1b), (1c) and (1d) showed a significant positive correlation relation between AIP and TCh with $p \leq 0.05$ for control group and G3 with correlation coefficient value r(0.19), (0.47) respectively. While a significant negative correlation relation was between AIP and TCh with $p \leq 0.05$ for G1, G2 with r value (-0.27),(-0.20) respectively.

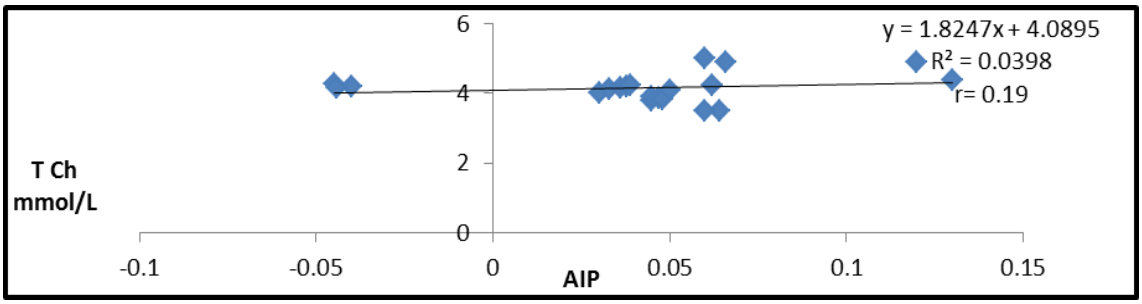


Figure (1a): Correlation relation between AIP and TCh for control group

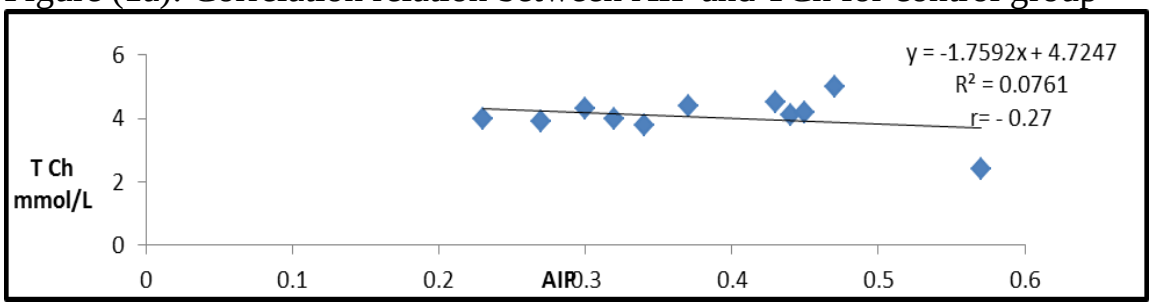


Figure (1b): Correlation relation between AIP and TCh for G1

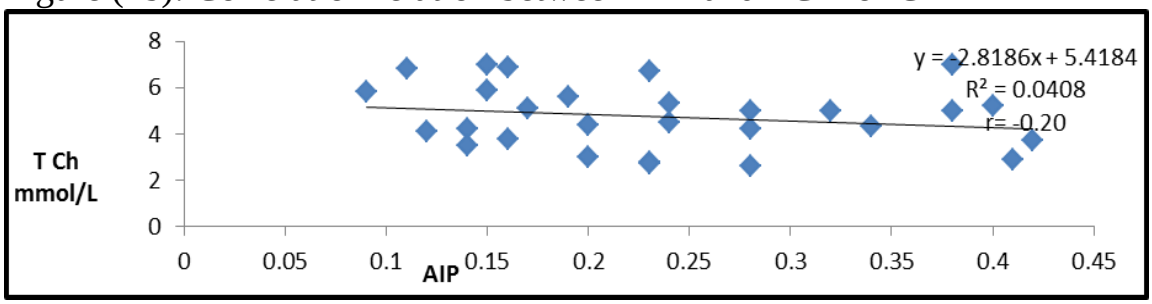


Figure (1c): Correlation relation between AIP and TCh for G2

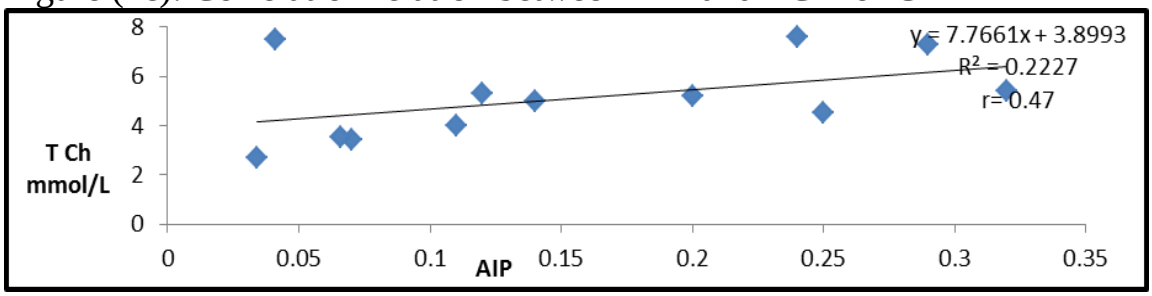
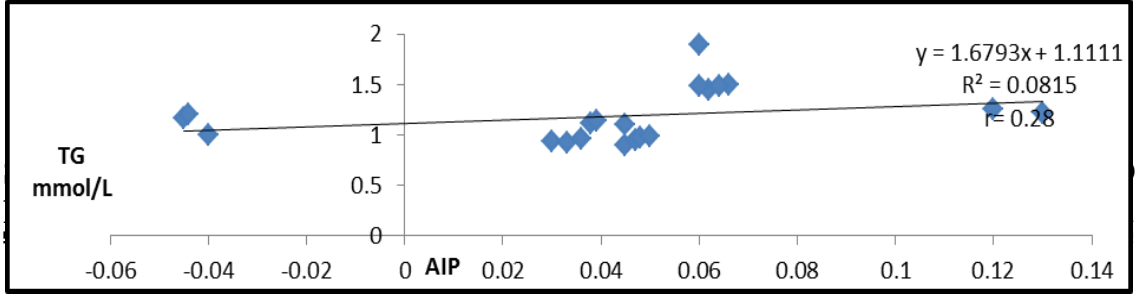


Figure (1d): Correlation relation between AIP and TCh for G3

Relationship between AIP and TG



Figures (2a), (2b), (2c) and (2d) showed a significant positive correlation relation between AIP and TG with p≤ 0.05 for control group and G1,G3 with correlation coefficient value r(0.28), (0.33),(0.10) respectively. While a significant negative correlation relation was between AIP and TG with p≤ 0.05 for CVD group, for G2 with r- value (-0.009) .

Figure (2a): Correlation relation between AIP and TG for control group

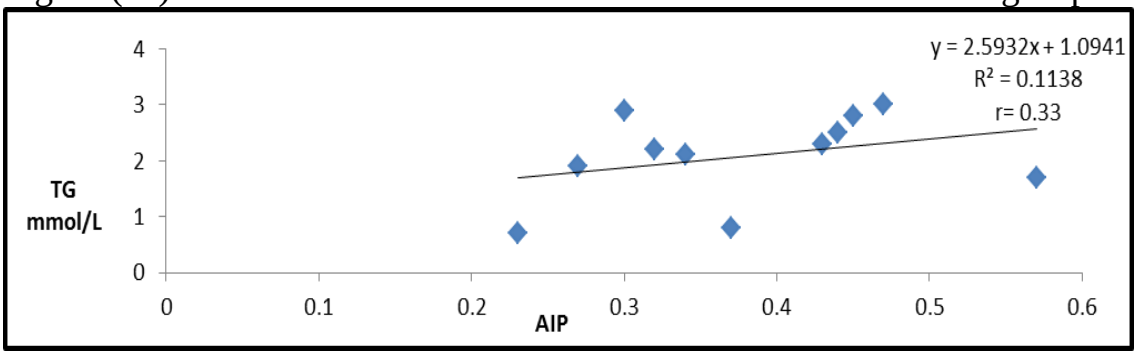


Figure (2b): Correlation relation between AIP and TG for G1

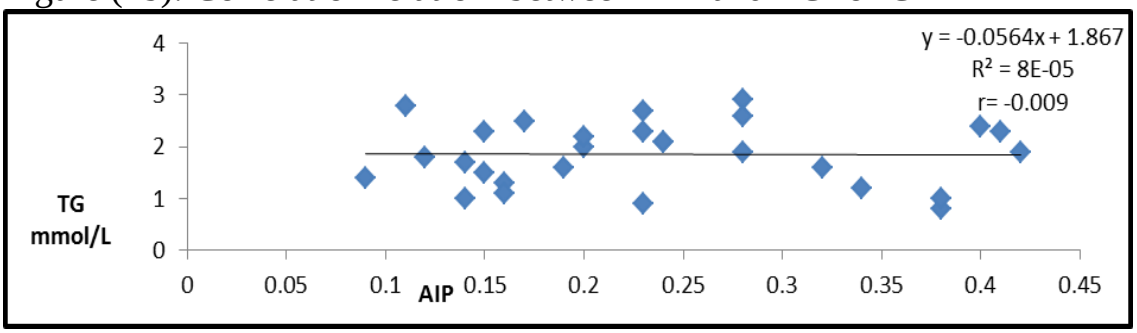


Figure (2c): Correlation relation between AIP and TG for G2

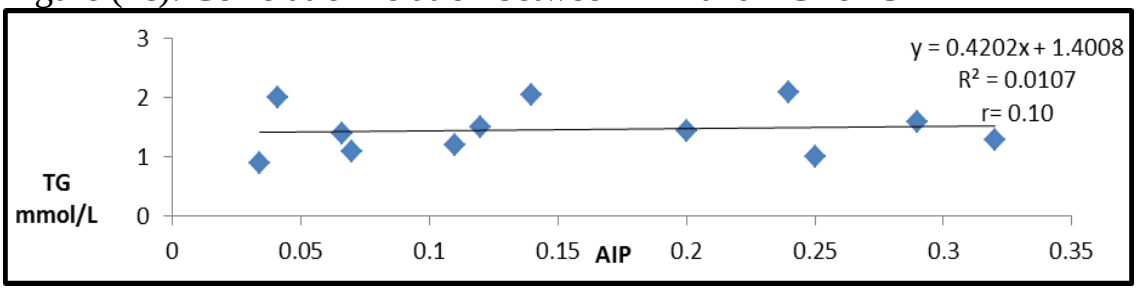


Figure (2d): Correlation relation between AIP and TG for G3

Relationship between AIP and HDL

Figures (3a), (3b),(3c)and(3d) showed a significant negative correlation relation between AIP and HDL with p≤ 0.05 for control group and G1,G3 with correlation coefficient value r(-0.02), (-0.32),(-0.52) respectively. While a significant positive correlation relation was between AIP and HDL with p≤ 0.05 for CVD group, for G2 with r- value (0.009).

Figure (3a): Correlation relation between AIP and HDL for control group

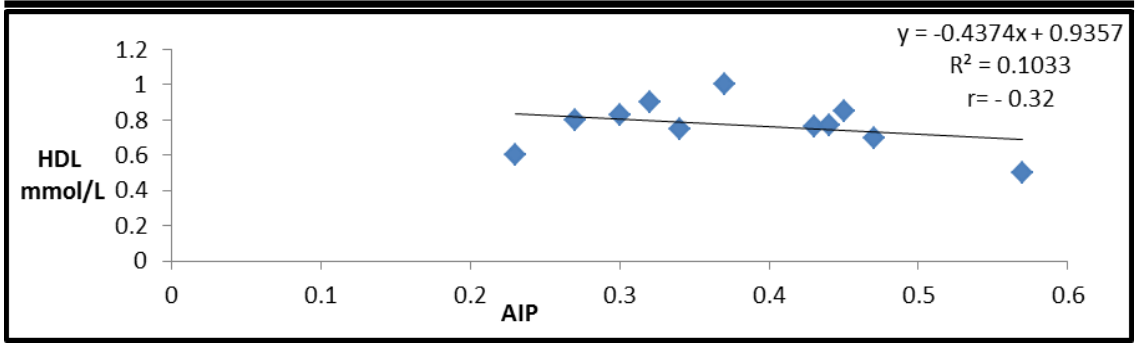


Figure (3b): Correlation relation between AIP and HDL for G1

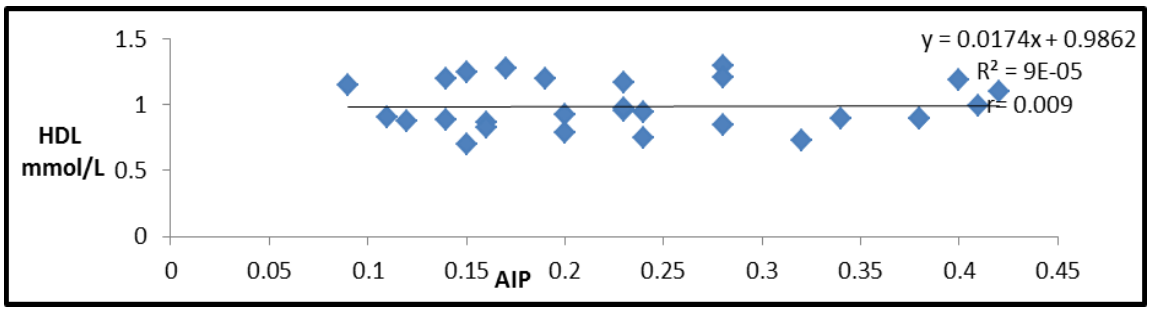


Figure (3c): Correlation relation between AIP and HDL for G2

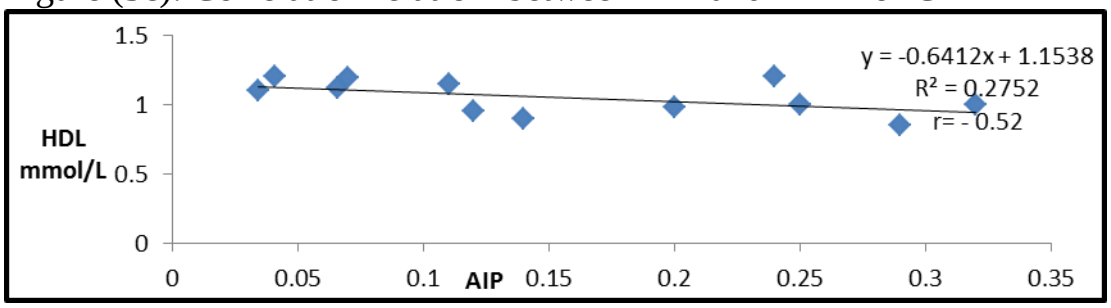


Figure (3d): Correlation relation between AIP and HDL for G3

Relationship between AIP and LDL

Figures (4-19a), (4-19b), (4-19c) and (4-19d) showed a significant positive correlation relation between AIP and LDL with $p \leq 0.05$ for control group and G3 with correlation coefficient value r (0.41), (0.06) respectively. While a significant negative correlation relation was between AIP and LDL with $p \leq 0.05$ for G1, G2 with r- value (-0.05), (-0.15) respectively.

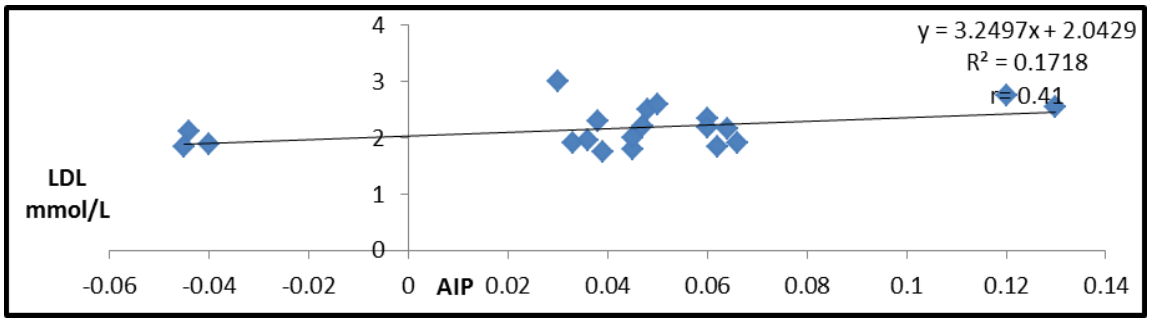


Figure (4a): Correlation relation between AIP and LDL for control group

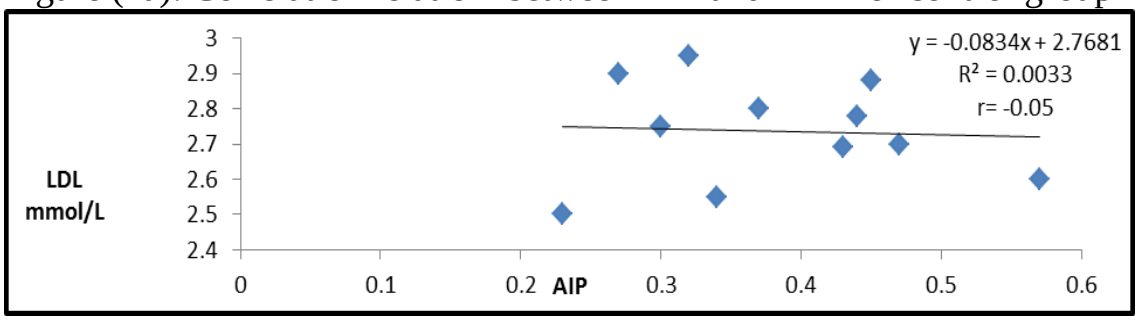


Figure (4b): Correlation relation between AIP and LDL for G1

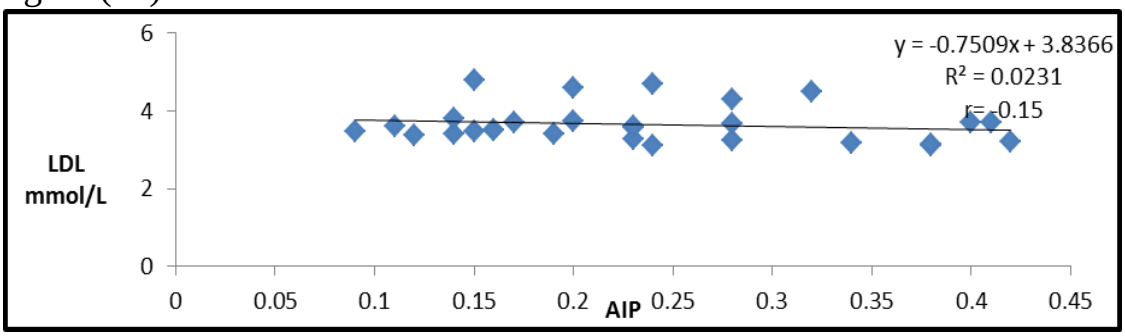


Figure (4c): Correlation relation between AIP and LDL for G2

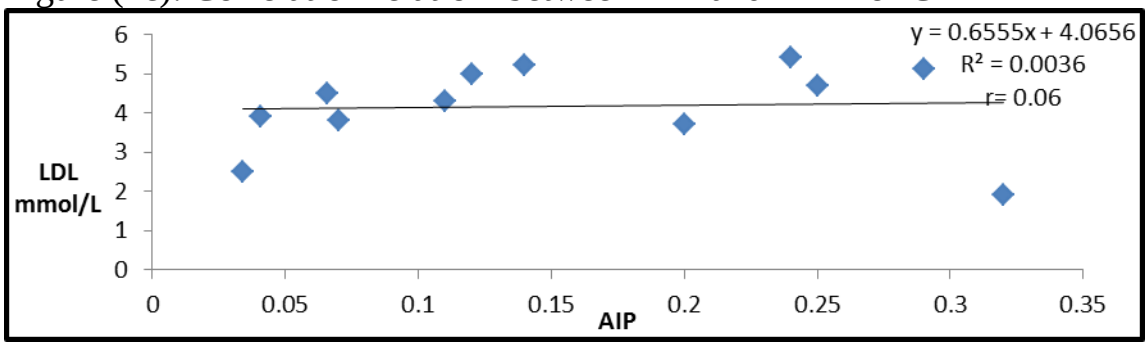


Figure (4d): Correlation relation between AIP and LDL for G3

Relationship between AIP and VLDL

Figures (5a), (5b), (5c) and (5d) showed a significant negative correlation relation between AIP and VLDL with $p \leq 0.05$ for control group and G2 with correlation coefficient value r (-0.10), (-0.29) respectively. While a significant positive correlation relation between was AIP and VLDL with $p \leq 0.05$ for G1, G3 with r value (0.23), (0.27) respectively.

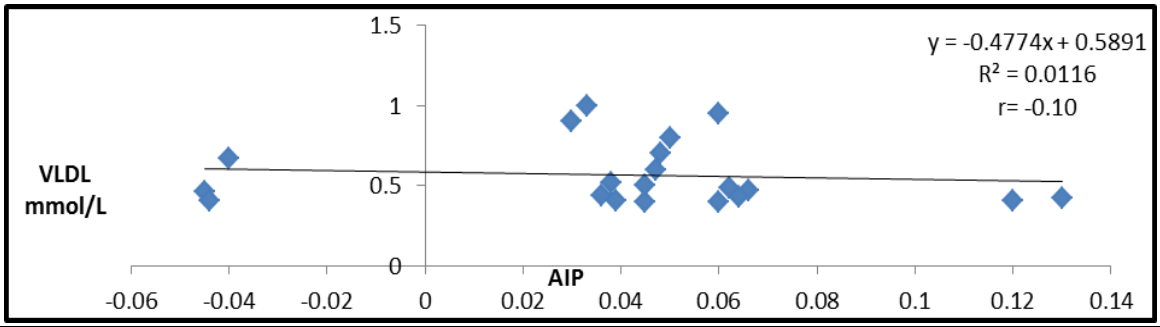
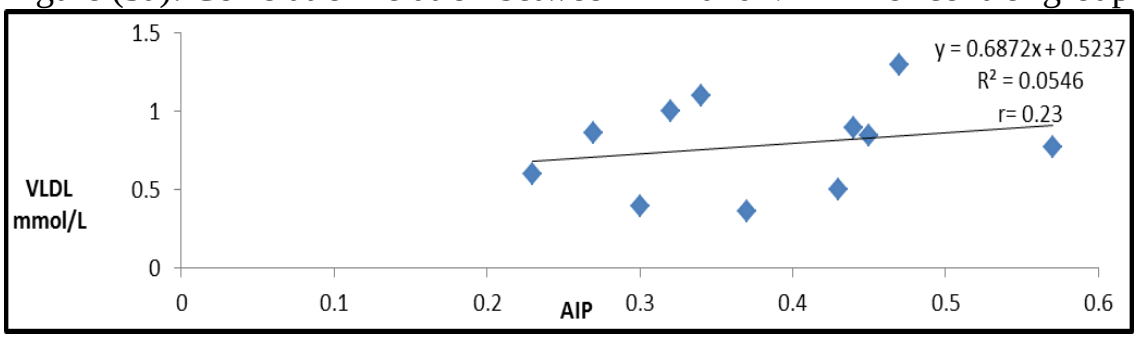


Figure (5a): Correlation relation between AIP and VLDL for control group



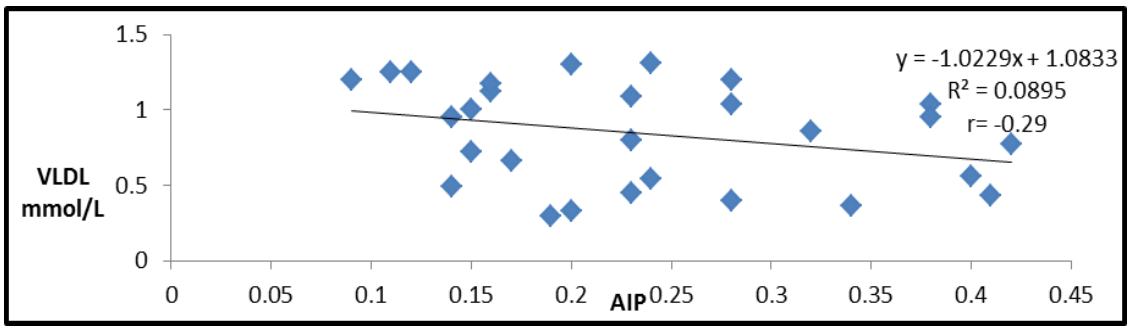


Figure (5b): Correlation relation between AIP and VLDL for G1

Figure (5c): Correlation relation between AIP and VLDL for G2

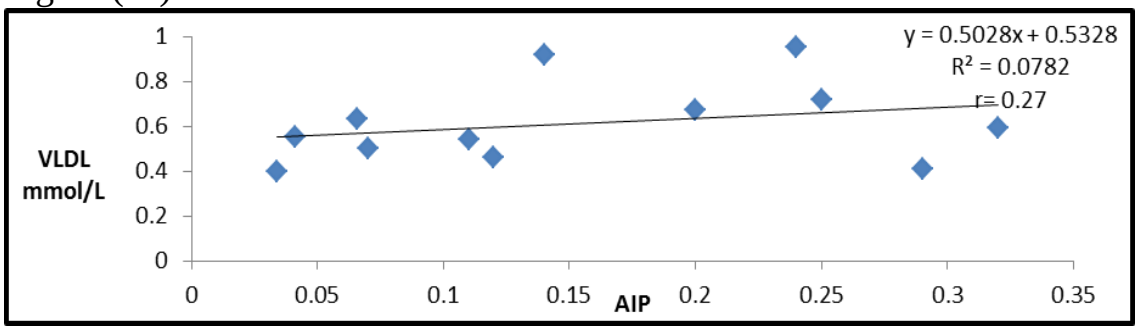


Figure (5d): Correlation relation between AIP and VLDL for G3

Relationship between AIP and BMI

Figures (6a),(6b),(6b), (6c) and (6d) showed a significant positive correlation relation between AIP and BMI with p≤ 0.05 for control group and G2, G3 with correlation coefficient value r (0.06), (0.05),(0.50) respectively. While a significant negative correlation was between AIP and BMI with p≤ 0.05 for G1 with r- value (-0.34).

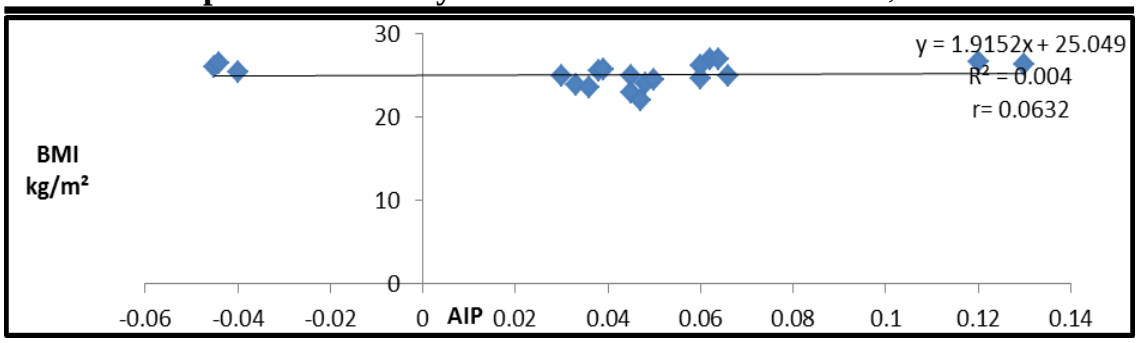


Figure (6a): Correlation relation between AIP and BMI for control group

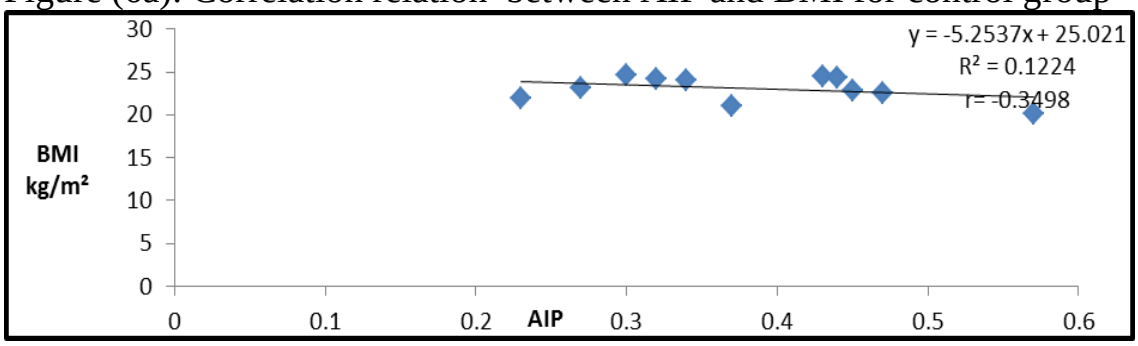


Figure (6b): Correlation relation between AIP and BMI for G1

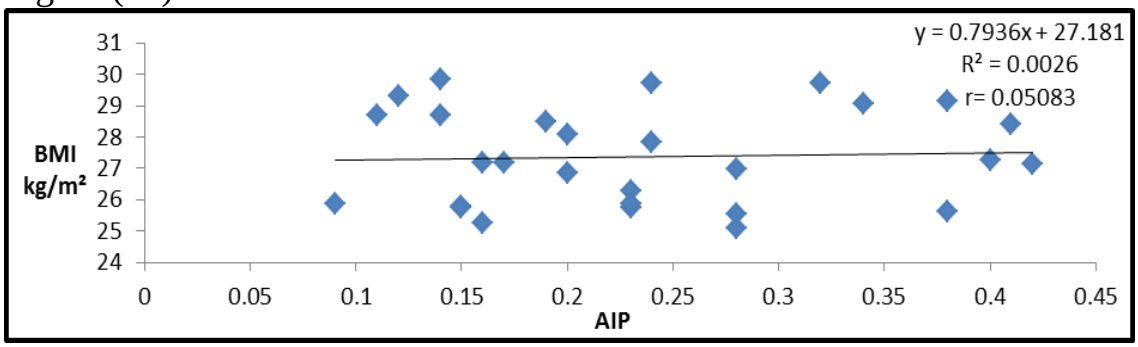


Figure (6c): Correlation relation between AIP and BMI for G2

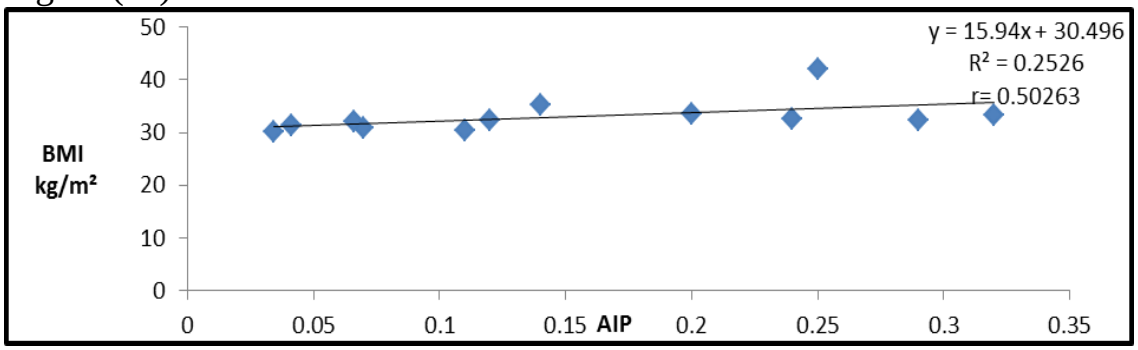


Figure (6d): Correlation relation between AIP and BMI for G3

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دراسة المحتوى الدهني Lipid Profile وعامل الخطورة AIP اعتمادا على معامل كتلة الجسم BMI في امصال دم المصابين بأمراض القلب ألوعائية مقارنة مع الاصحاء

الخلاصـــــة

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تم اجراء هذه الدراسة في مستشفى ابن البيطار التخصصي لأمراض القلب والأوعية الدموية / بغداد للفترة من كانون الاول ٢٠١١ ولغاية نيسان ٢٠١٢ .
لقد استخدم مصل دم (71) شخصا من كلا الجنسين تتراوح أعمارهم بين (٣٣-٦٧) سنه, بواقع(٥١) مريضا من المصابين بأمراض القلب والأوعية الدموية و(٢٠) شخصا من الأصحاء كمجموعة سيطرة لتقدير بعض الدوال الكيموحيوية والتي تضم الكولسترول(TCh) والكليسريد الثلاثي (TG) والبروتينات الدهنية [البروتينات الدهنية العالية الكثافة (HDL) والبروتينات الدهنية الواطئة الكثافة (LDL) والبروتينات الدهنية الضئيلة الكثافة (VLDL)] بالإضافة إلى قياس مستوى البروتين ألارتكازي – سي (CRP)
وقد تم حساب عامل الخطورة (AIP) Atherogenic Index of Plasma
وقد قسمت النماذج وكما يلي:
١- مجموعة السيطرة وتشمل(٢٠) شخصا من كلا الجنسين تتراوح أعمارهم بين (٢٥-٦٣) ولا يعانون من اي مرض او يتناولون علاجا قد يتداخل مع المتغيرات قيد الدراسة
٢ - أما العينات المرضية فقد قسمت إلى ثلاثة مجاميع اعتمادا على معامل كتلة الجسم Body Mass Index(BMI) :-
أ-المجموعة الأولى (G1) وتشمل (١١) مريضا بوزن طبيعي Normal weight (٢٠-٢٤,٩) كغم /م^2 .
ب-المجموعة الثانية (G2) وتشمل (٢٨) مريضا بوزن زائد Over weight (٢٥-٢٩,٩) كغم /م^2 .
ج- المجموعة الثالثة (G3) وتشمل (١٢) مريضا بزيادة مفرطة في الوزن Obese (٣٠ ←) كغم /م^2 .

أظهرت بيانات هذه الدراسة زيادة معنوية في تركيز كل من TCh و TG و LDL و VLDL في أمصال دم الأشخاص المصابين بأمراض القلب الوعائية مقارنة مع الأصحاء.
وكذلك اظهر مستوى CRP زيادة معنوية مقارنة مع الاصحاء.
أما HDL فقد أظهرت البيانات انخفاضا معنويا في تركيزه في أمصال دم الأشخاص المصابين بأمراض القلب الوعائية مقارنة مع الأصحاء.
لقد خلصت الدراسة الحالية الى وجود علاقات معنوية بين عامل الخطورة AIP والمتغيرات الكيموحيوية المدروسة آنفا في مجاميع الاشخاص المصابين بأمراض القلب الوعائية.