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A Study of Visfatin Levels and Other Related Parameters in Sera of Iraqi Non Diabetic Male with Hyperlipidemia

Dr. Tariq M.Ali *
Dr. Zeinab M. M. Al-Rubaei *
Ban mahmood Shaker Al-joda **
*Chemistry Department, College of Education For Pure Science /Ibn Al-Haitham ,Baghdad University.
** Chemistry Department , College of medicine , Babylon University .

Abstract

The aim of the present study is to evaluate the levels of visfatin in sera of non-diabetic male with hyperlipidemia classified according to Frederickson classification .At the same time to find the correlation of visfatin with related parameters in the studied groups. The study included (22) healthy Iraqi male as control group (G1) and (62) non-diabetic male diagnosed with hyperlipidemia which divided into three groups as follows: group(2)consist of (20)male with hypercholesterolemia[Type I] ,group (3)consist of (20) male with hypertriglyceridemia [Type IIa] and group (4):consist of (22)male with hypercholesterolemia and hypertriglyceridemia [Type IIb] .The age of all studied groups ranged between (21-50) years and BMI with (19.8-24.3) Kg/m^2 .Serum was used for determination of FBG, lipid profile(HDL,LDL,VLDL,TG) ,insulin ,CRP and visfatin .Whole blood was used for determination of HbA1C. The results revealed no significant elevation in FBS and HbA1C levels were seen in patients groups when comparing to healthy control . The results indicates a significant elevation in TC and LDL in G2and G4 comparing to G1. Also, there are significant elevation in G3 comparing to G 2 and G4 comparing to G3,while no significant elevation was found in TC and VLDL in G3 with G1and G4 with G2.The results ,also, illustrated significant elevation in TG and VLDL in G3 and G4 when comparing to G1, and in G3,G4 comparing to G2 . No significant elevation was noticed in G2 comparing to G1and G4 comparing to G3 . HDL levels showed significant decrease in G2,G3 and G4 comparing to G1. No significant decrease seen in G3 and G4 comparing to G2 and in G4 comparing to G3.A high significant elevation in insulin and HOMA-IR levels in G2,G3 and G4 was found when comparing with G1 , while a significant elevation was noticed in G3 and G4 when comparing with G2 and in G4 when comparing with G3. The results, also ,showed significant increase in CRP and visfatin levels for G2,G3 ,G4 when comparing to G1 and in G4 when comparing to G2 and G3 . But no significant elevation was found in G3 when comparing to G2 .A significant positive correlation was found between visfatin levels and TC in G1,G2 and G4 while significant negative correlation was observed in G3 between visfatin and TC. A high significant positive correlation coefficients was found between visfatin and TG in four studied groups .The results , also, showed a significant negative correlation between visfatin levels and HDL for all studied groups .The results illustrated significant positive correlation in visfatin levels with LDL in G1 and G4 while there are significant negative correlation in visfatin levels with LDL in G2 and G3. A significant negative correlation between visfatin and insulin ,HOMA-IR levels was noticed in all studied groups.

The conclusion could be drown from this study that visfatin increased in G4 more than other groups comparing to G1 . Also, there are significant correlation between visfatin levels with insulin , HOMA-IR and CRP levels.

Keywords : visfatin , Insulin , CRP , Hyperlipoproteinemia

Introduction

Lipoproteins are substances made of cholesterol ,triglycerides ,and proteins . They move cholesterol , triglycerides , and other lipids to different parts of the body [(1)] .Hyperlipoproteinemia is a metabolic disorder characterized by abnormally elevated concentrations of specific lipoprotein particles in the plasma . Hyperlipidemia (i.e. elevated plasma cholesterol or triglyceride levels or both) is present in all hyperlipoproteinemia [(2)] .

Adipose tissue is considered as a hormonally active system with metabolic effects. Visfatin is one of the most abundant adipocytokines recently discovered with the capacity to modulate several functions [(3, 4)] . Visfatin that binds to insulin receptor but at a different binding site than insulin itself . The affinities of visfatin and insulin for insulin receptors are similar but circulating visfatin concentration is at least 10 times lower than that of insulin . The molecular mechanisms revealed that visfatin activates intracellular cascade for insulin signaling , including tyrosine phosphorylation of the insulin receptor and insulin receptor as well as downstream activation of protein kinase B [(5)] . The study demonstrated insulin-mimetic effects of visfatin such as: increased glucose uptake in adipocytes and myocytes, suppression of hepatic glucose release, stimulated triglyceride accumulation, and increase in its synthesis in pre-adipocytes [(6)] .

Competitive inhibition study showed that each of visfatin and insulin compete for the insulin receptor, raising the hypothesis that visfatin could have a binding site different from that of insulin. Recombinant receptors with structural changes decreasing affinity for insulin didn't change visfatin binding [(7)]. Increased

visfatin levels are associated to coronary artery disease (CAD) and acute coronary syndromes even after correction for classic cardiovascular risk factors such as cholesterol , smoking , hypertension , diabetes , and obesity [8-10] .

The present study aimed to :

Compare the levels of visfatin in non-diabetic male with hyperlipidemia and compare it with healthy control . Also to find the correlation of visfatin with TC,TG,HDL,LDL, insulin ,HOMA-IR and CRP .

Material & Methods

This study included eighty four male with aged ranged (21-50) years and BMI ranged between (19.8-24.3) Kg/m^2. Subjects were divided into four groups group(1) consist of (22) male as healthy control .Patient divided into (3) groups according to Fredrecsons classification :

Group (2) : consist of (20) non-diabetic male with hypercholesterolemia[Type I] .

Group(3):consist of (20) non-diabetic male with hypertriglyceridemia [Type IIa] .

Group(4): consist of (22) non-diabetic male with hypercholesterolemia and hypertriglyceridemia [Type IIb] .

The patients attended the Ibn –Al Naphes hospital in Baghdad and Al-Hilla General Teaching Hospital in Babylon for the period from January 2014 to August 2014 .Ten milliliters of blood were collected after an overnight fasting from all subjects by venipuncture . A liquate of (0.5 ml) of whole blood was used in determination of HbA1C . The other part was left in 37C˚ for (15 min) to clot then centrifuged at (3000 rpm) for(25 min) . The serum which obtained was freezed until analysis of lipid profile , insulin ,CRP and visfatin .

HbA1C was determined by a bromate affinity assay traceable to the IFCC reference method[11]. Total serum cholesterol determined by utilizing a kit based on the enzymatic hydrolysis [12] . The absorbance was recorded for the quinonimine (red complex) at 500 nm .The TG determination based on the enzymatic hydrolysis . The intensity of the color formed is proportional to the triglycerides concentration in the sample [13] .

The Chylomicrons and lipoproteins of VLDL, and LDL contained in the serum sample were precipitates. HDL levels were determined in the supernatant which obtained after centrifugation according to the based method [14] .

LDL-cholesterol and VLDL were estimated indirectly by using Friedewald formula [15] :

LDL-c =Total Cholesterol - (HDL-c + VLDL-c)

VLDL-c(mg⁄dl) =TG/5

Insulin ,CRP and visfatin were determined by using ELIZA teqniech based on the sandwich method [16] . HOMA-IR insulin resistance calculated by using the following formula [17] : [fasting glucose × fasting insulin] / 22.5 .

All parameters were expressed as(mean ± SD) . T-test was used for comparsion among the four studied groups . The P-vales < 0.05 and < 0.001 were considered statistically significant and high significant, respectively.

The Results :

Analytical parameters :

The mean ± SD and T-test of descriptive parameters for non –diabetic male in the studied groups with aged ranged (21-50) years and BMI ranged between(19.8- 24.3) Kg/m^2 are presented in table (1) .

Table(1) : Descriptive parameters for all studied groups

***(S) significant differences , (NS) no significant differences .**

Groups / Parameters	G1 n=22	G2 n=20	G3 n=20	G4 n=22	T-test G1 vs G2	T-test G1 vs G3	T-test G1 vs G4	T-test G2 vs G3	T-test G2 vs G4	T-test G3 vs G4
HbA1C %	5.04 ± 0.43	4.79 ± 0.37	4.74 ± 0.45	5.0 ± 0.42	S	NS	S	S	NS	S
TC (mg/dl)	135.36± 29.88	223 ± 10.53	175 ± 25.73	231 ± 26.75	NS	NS	NS	NS	NS	NS
TG (mg/dl)	109 ± 17.8	130 ± 14.7	240 ± 41.7	302 ± 87.2	NS	S	S	S	S	NS
HDL (mg/dl)	51.59 ± 8.8	43.7 ± 10.5	33.5 ± 5.32	40.45 ± 8.36	S	S	S	NS	NS	NS
LDL (mg/dl)	92.2 ± 25.4	157.1 ± 20	94.32 ± 31.7	130.0 ± 20.2	S	NS	S	S	NS	S
VLDL (mg/dl)	21.8 ± 7.13	26.01 ± 5.84	48.1 ± 10.71	60.5 ± 14.9	NS	S	S	S	S	NS

The results in table (1) revealed no significant elevation in FBS and HbA1C levels was found in patients groups when comparing to healthy control . The results indicate significant elevation in TC and LDL in

G2 and G4 comparing to G1. Also , there are significant elevation in G3 comparing to G 2 and G4 comparing to G3,while no significant elevation was found in TC and VLDL in G3 comparing to G1and G4 comparing to G2 .The results , also, illustrated significant elevation in TG and VLDL in G3 and G4 when comparing to G1, and in G3,G4 comparing to G2 . No significant elevation was noticed in G2 comparing to G1and G4 comparing to G3 for TG and HDL . HDL levels showed significant decrease in G2,G4 comparing to G1,while there are significant decrease in G3 comparing to G1.

Elevated TC primarily reflects elevated LDL cholesterol , which constitutes 70% of plasma cholesterol . Disorders characterized by elevation of TC alone are classified as Fredrickson type IIa hyperlipoproteinemia [18 ,19] .

Familial type IIa hypercholesterolemia is a genetic disorder caused by a defect on chromosome 19 .The defect makes the body unable to remove low density lipoprotein cholesterol from the blood .This results in high levels of LDL in the blood [20, 21] . Type I hyperlipoproteinemia is rare disorder characterized by severe elevations in chylomicrons and extremely elevated triglycerides , It is caused by mutations of either the lipoprotein lipase gene which is critical for the metabolism of chylomicrons and VLDL [22] , This condition is characterized by inflammation of the pancreas (pancreatitis) [23].An elevated in TC and decreased in HDL levels is observed among hyperinsulinemic and hypertriglyceridemic individuals [24 ,25] . It is proposed that the ability of this parameters to predict risk is explained by the fact that it is a relevant cumulative marker of the metabolic abnormalities found in individuals with high TG-low , HDL-C dyslipidemia which is a consequence of insulin resistance [26] .

Table (2) showed (mean ± SD) and (T-Test) of insulin , HOMA-IR , visfatin , for all studied groups . The results in table (2) showed a highly significant elevation in insulin and HOMA –IR levels for G2, G3 and G4 when comparing to G1 , while significant elevation was noticed in G3 and G4 when comparing to G2 and in G4 with G3 .

Table (2) : levels of Insulin , HOMA- IR , visfatin in all studied groups

Groups / Parameters	G1 n=22	G2 n=20	G3 n=20	G4 n=22	T-test G1 vs G2	T-test G1 vs G3	T-test G1 vs G4	T-test G2 vs G3	T-test G2 vs G4	T-test G4 vs G3
Insulin (Mu /ml)	4.88 ±0.4	8.83 ±1.34	11.97 ±1.91	15.98 ±2.91	HS	HS	HS	S	S	S
HOMA-IR	18.9 ±3.22	38.26 ±8.9	50.11 ±14.4	65.3 ±15.34	HS	HS	HS	S	S	S
CRP (mg /dl)	2.49 ±0.6	3.54 ±0.4	3.28 ±0.4	6.1 ±0.96	S	S	S	NS	S	S
Visfatin (ng /dl)	14.85 ±2.3	21.77 ±2.73	23.06 ±3.29	29.76 ±5.27	S	S	NS	NS	S	S

*** (S)significant differences , (HS) high significant differences ,(NS)No significant differences**

The HOMA-IR is a condition in which the body's cells become resistance to the effects of insulin . That is the normal response to a given amount of insulin is reduced . As a results, higher levels of insulin are needed in order for insulin to have its proper effects [27] . The results in table (2) , also , showed significant increase in CRP and visfatin levels for G2 ,G3 and G4 when comparing to G1 and in G4 comparing to G2 and G3, but non-significant elevation was found in G3 when comparing to G2 .Levels of visfatin are linked to the CRP which is one of the powerful predictors of atherosclerosis and vascular death [27,28] which in agreement with ours study. A results of the previous investigation indicate that autocrine effects of visfatin may play an important role in regulating insulin sensitivity in the liver. It can be hypothesized that insulin-sensitizing effects of visfatin are related to its essential role in the NAD biosynthetic pathway [29] . It was proven that oxidized LDL increases the visfatin expression in the monocytes cell cultures . More than that, visfatin determines the expression increase of some extracellular degrading molecules , causing the instability of the atheroma plaque [30 , 31] .

The conclusion could be drown from this study that visfatin increased in G4 more than other groups comparing to G1 . Also, there are significant correlation between visfatin levels with insulin , HOMA-IR and CRP levels.

As far as to our knowledge this is the first study determined the levels of visfatin in young male with normal BMI and find its relation with lipid profile in patients with hyperlipoproteinemia which divided according to Fredrecsons classification .

Relationships and correlation coefficients :

Relationship between visfatin viruses, TC,TG,HDL, LDL, insulin , HOMA-IR and CRP were studied for all studied groups which shown in table (3) .

Table (3) : Correlation coefficient and p-value between visfatin levels and other parameters for all studied groups

Groups / Parameters	G1		G2		G3		G4	
	r	p-value	r	p-value	r	p-value	r	p-value
(Visfatin) vs (TC)	0.0967	S	0.3947	S	-0.2317	S	0.343	S
(Visfatin) vs (TG)	0.0831	HS	0.5051	HS	0.3273	HS	0.146	HS
(Visfatin) vs (HDL)	-0.2824	S	-0.0329	S	-0.4425	S	-0.256	S
(Visfatin) vs (LDL)	0.0874	S	-0.1067	S	-0.3132	S	0.096	S
(Visfatin) vs (insulin)	-0.057	S	-0.152	S	-0.1066	S	-0.349	S
(Visfatin) vs (HOMA-IR)	- 0.3146	S	-0.0387	S	-0.0455	S	-0.014	S
(Visfatin) vs (CRP)	-0.181	HS	0.2477	HS	-0.4212	HS	-0.082	HS

***(S) Significant differences,(HS) highly significant doifferences,(NS)No significant differences**

A significant positive correlation was found between visfatin levels and (TC) in G1,G2 and G4 (r_1 =0.0967 , r_2 = 0.3947 , r_4 = 0.3435) respectively , as shown in figure (1 A,B, and D) while significant negative correlation was observed in G3 (r_3 =-0.2317) between visfatin and (TC) , as shown in figure (1 C) .

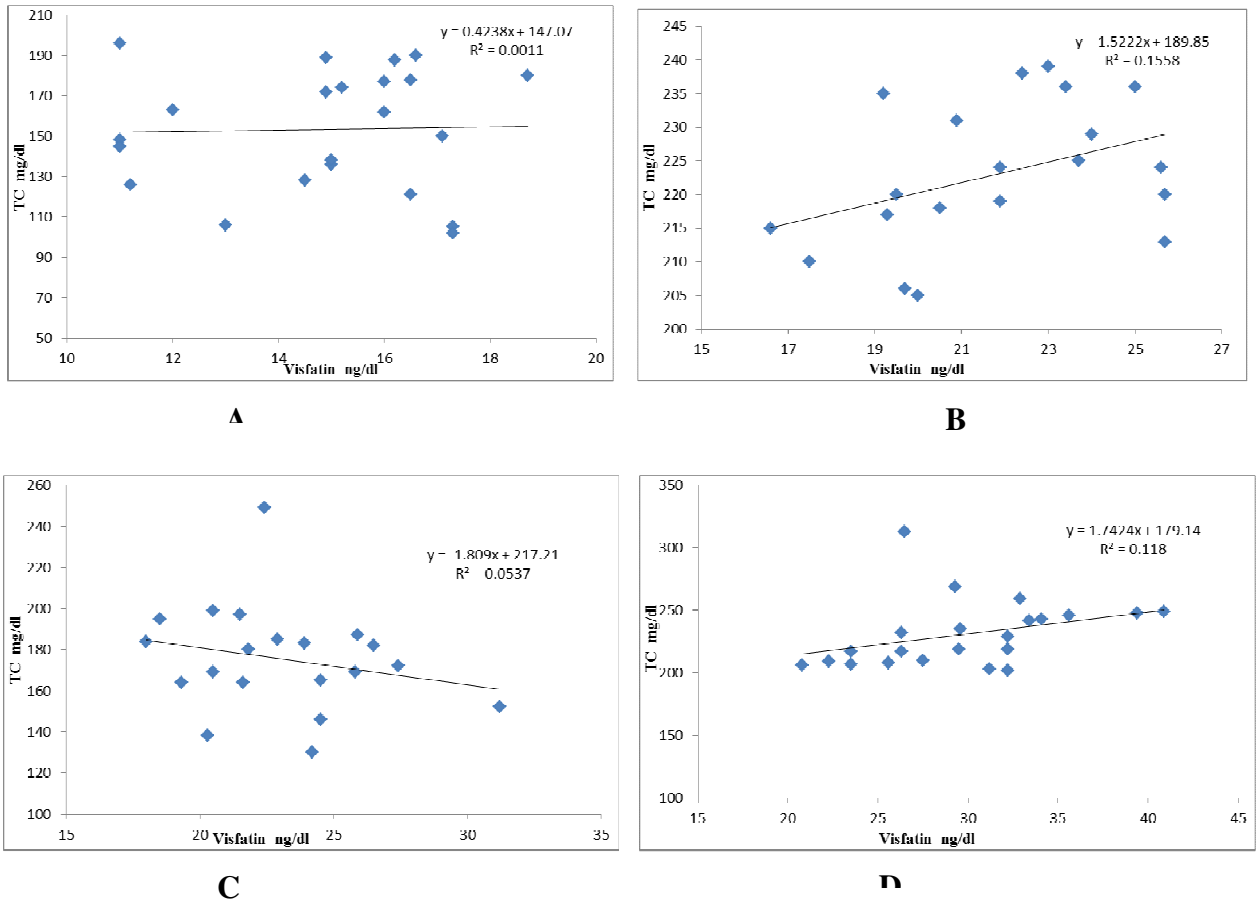


Figure (1) Correlation between visfatin and TC in G1(A) , G2 (B) , G3 (C) , G4 (D)

A high significant positive correlation coefficients was found between visfatin and TG in four studied groups (r_1= 0.08312 , r_2 =0.505 , r_3 = 0.3278 , r_4 =0.1462) respectively , as shown in figure (2 A,B,C,D) .

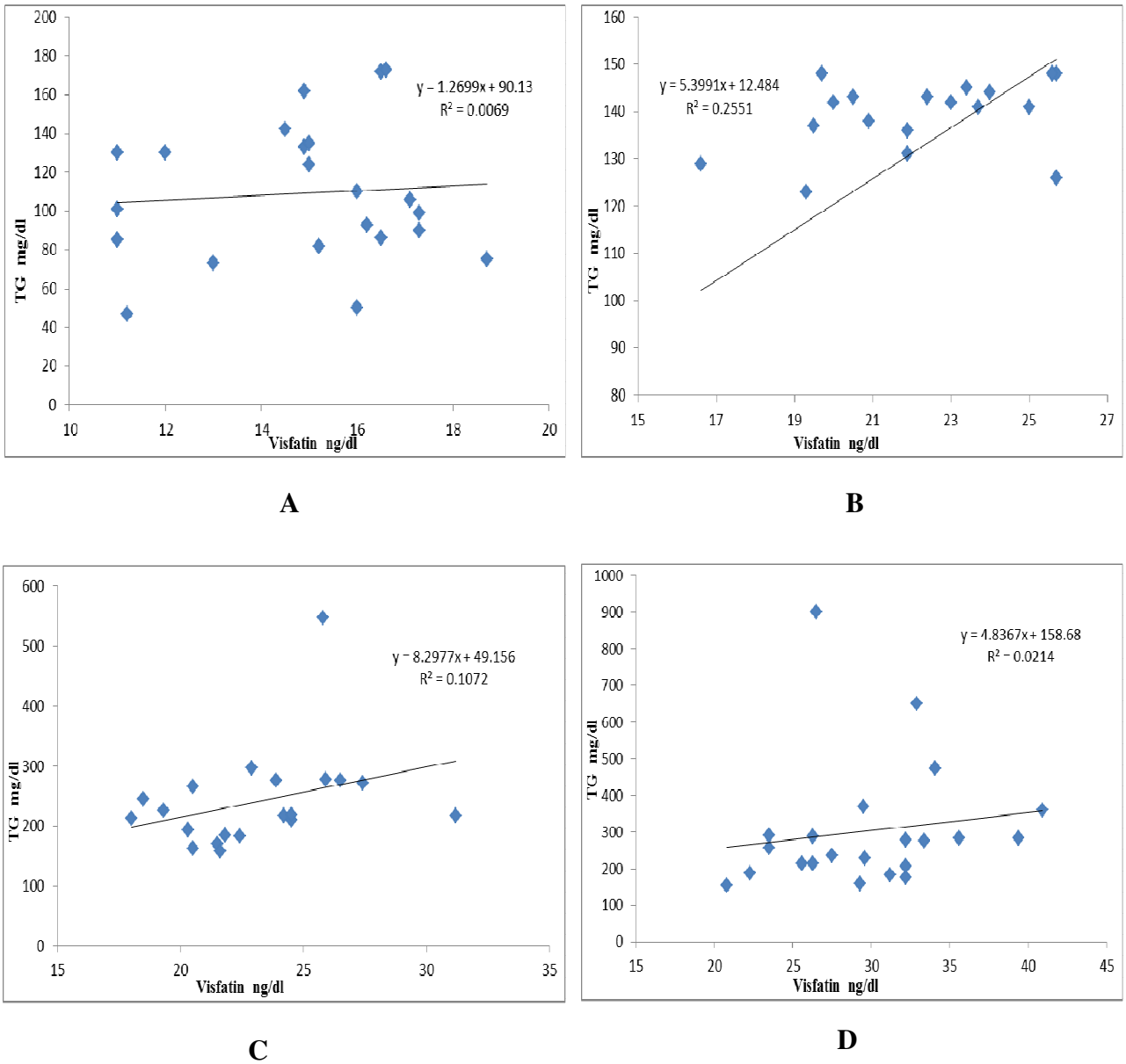
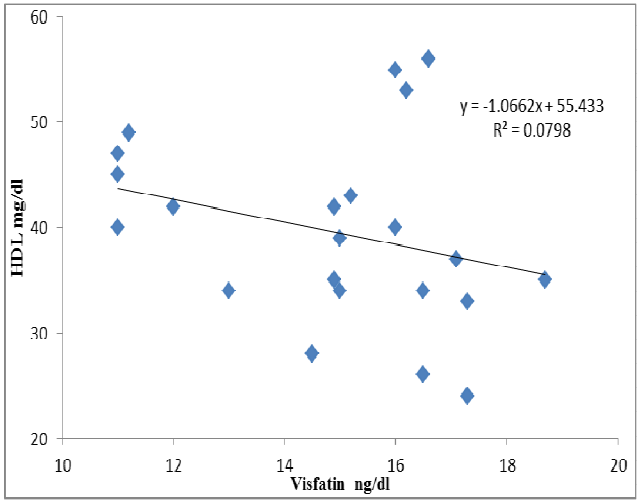
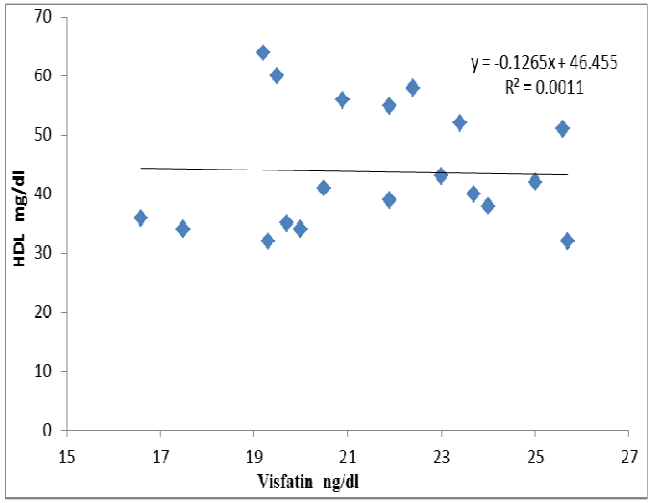


Figure (2) Correlation between visfatin and TG in G1(A),G2(B),G3(C),G4(D)

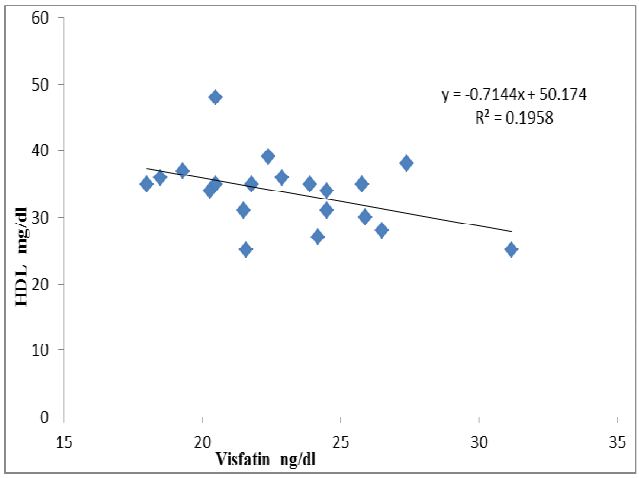
The result ,also , showed a significant negative correlation between visfatin levels and HDL for all studied groups (r_1= -0.2824, r_2 = -0.0329 ,r_3= -0.4425 , r_4 =-0.2567) respectively, as shown in figures (3 A,B,C and D) .



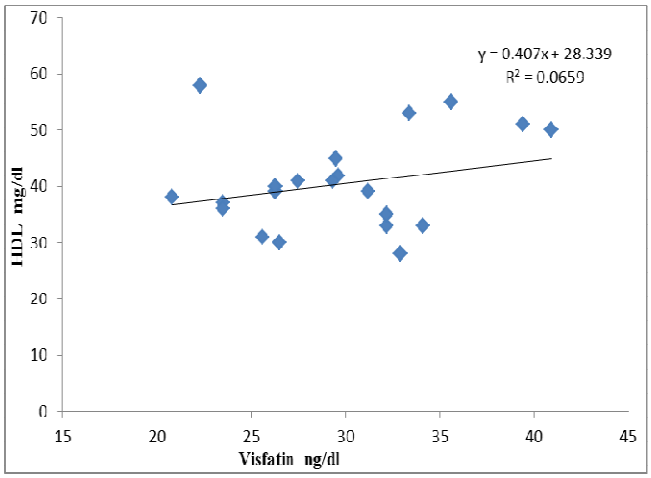
A



B



C



D

Figure (3) Correlation between visfatin and HDL in G1(A),G2(B),G3(C),G4(D)

The results in table (3) illustrated significant positive correlation in visfatin levels with LDL in G1 and G4 (r_1=0.0874 , r_4= 0.0967) respectively, as shown in figures (4 A,D),while there are significant negative correlation in visfatin levels with LDL in G2 and G3 (r_2=-0.1067, r_3= -0.313)respectively , as shown in figures (4 B, C) .

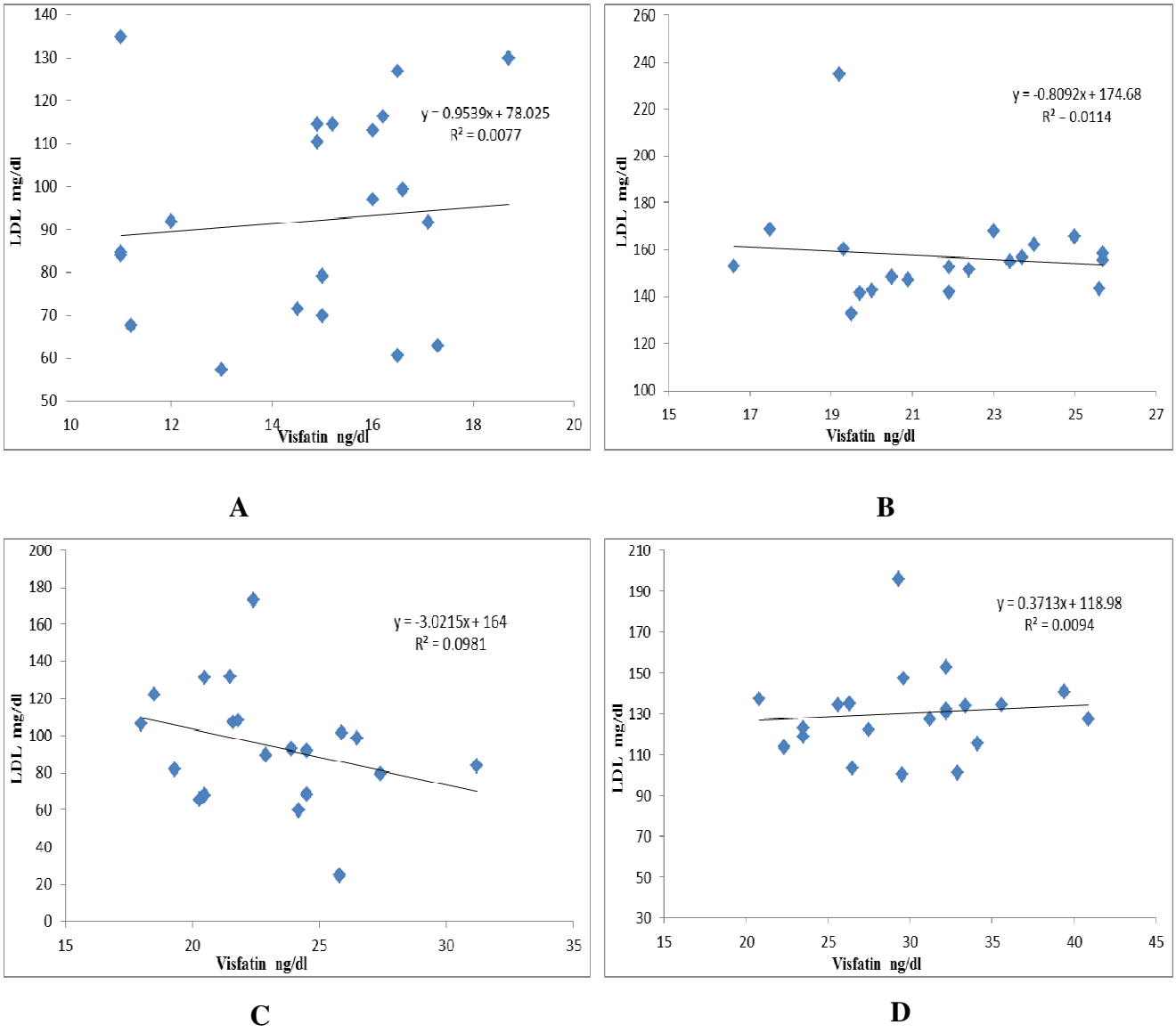
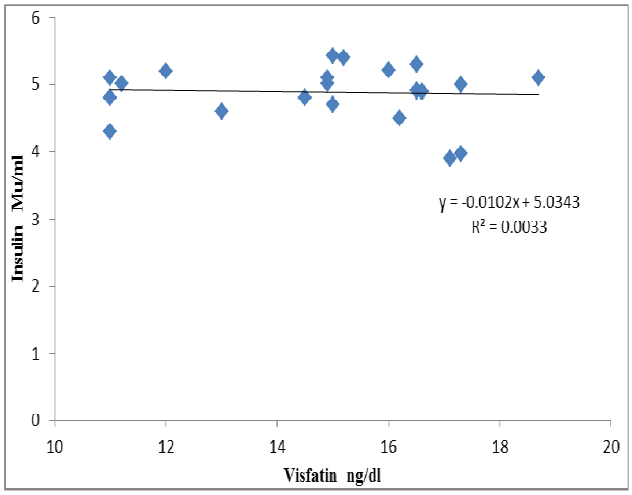
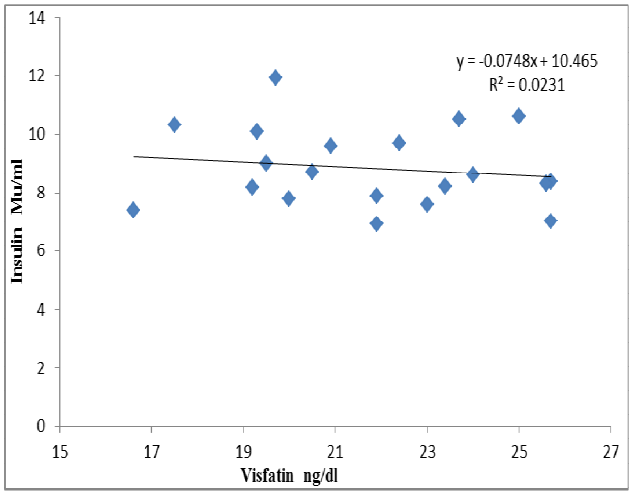


Figure (4) Correlation between visfatin and LDL in G1(A),G2(B),G3(C),G4(D)

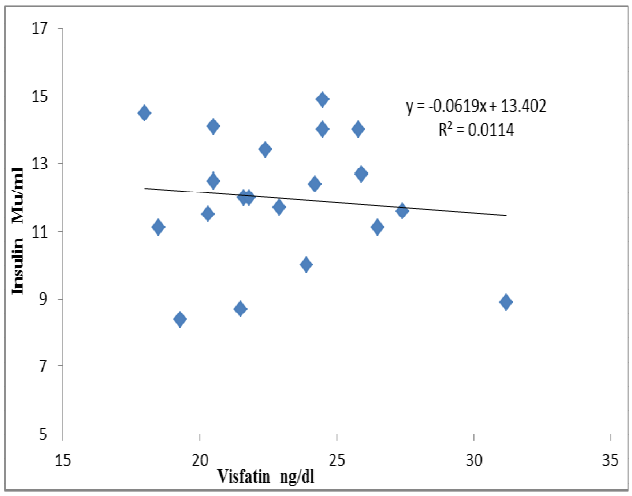
A significant negative correlation between visfatin and insulin levels in illustrated group (r_1=- 0.057, r_2= - 0.152 , r_3= - 0.1066 and r_4= - 0.349) respectively as shown in figures (5 A,B,C,D) .



A



B



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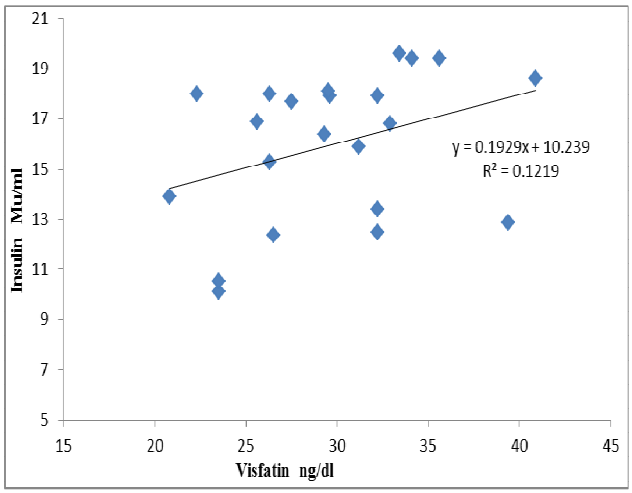

D

Figure (5) Correlation between visfatin and insulin in G1(A),G2(B),G3(C),G4(D)

A significant negative correlation between visfatin and HOMA-IR levels in illustrated group (r_1=-0.314, r_2 = -0.0387, r_3 = -0.04559 and r_4 = -0.0147) respectively as shown in figures (6 A, B,C and D) .

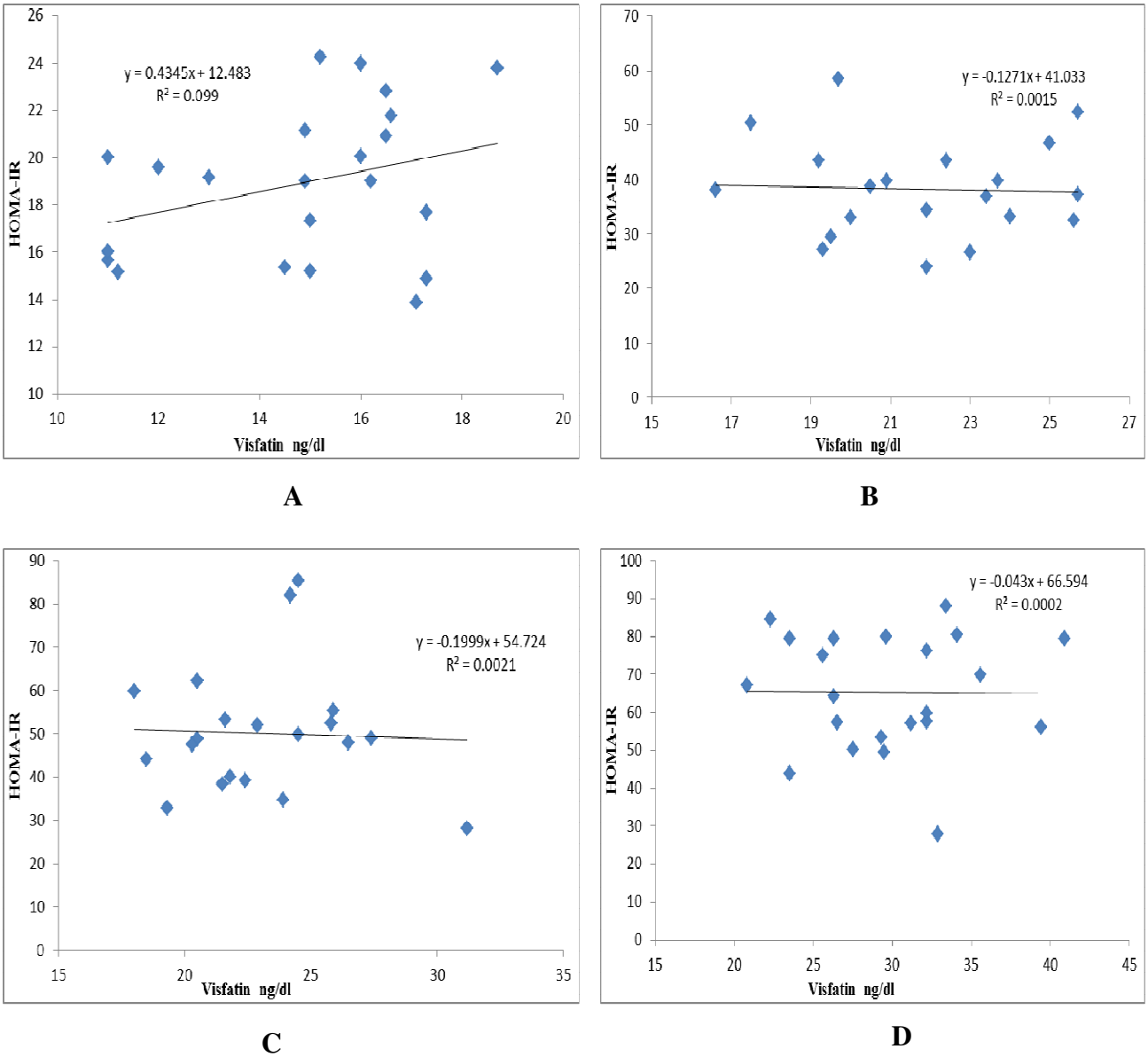

Figure (6)Correlation between visfatin and HOMA-IR in G1(A),G2(B),G3(C),G4(D)

A highly significant correlation was found in visfatin and CRP levels in all studied groups which (r1= - 0.1813 , r2 = 0.2477, r3 = - 0.4212 and r4 = -0.0829) respectively, as shown in figures (7 A ,B ,C and D) .

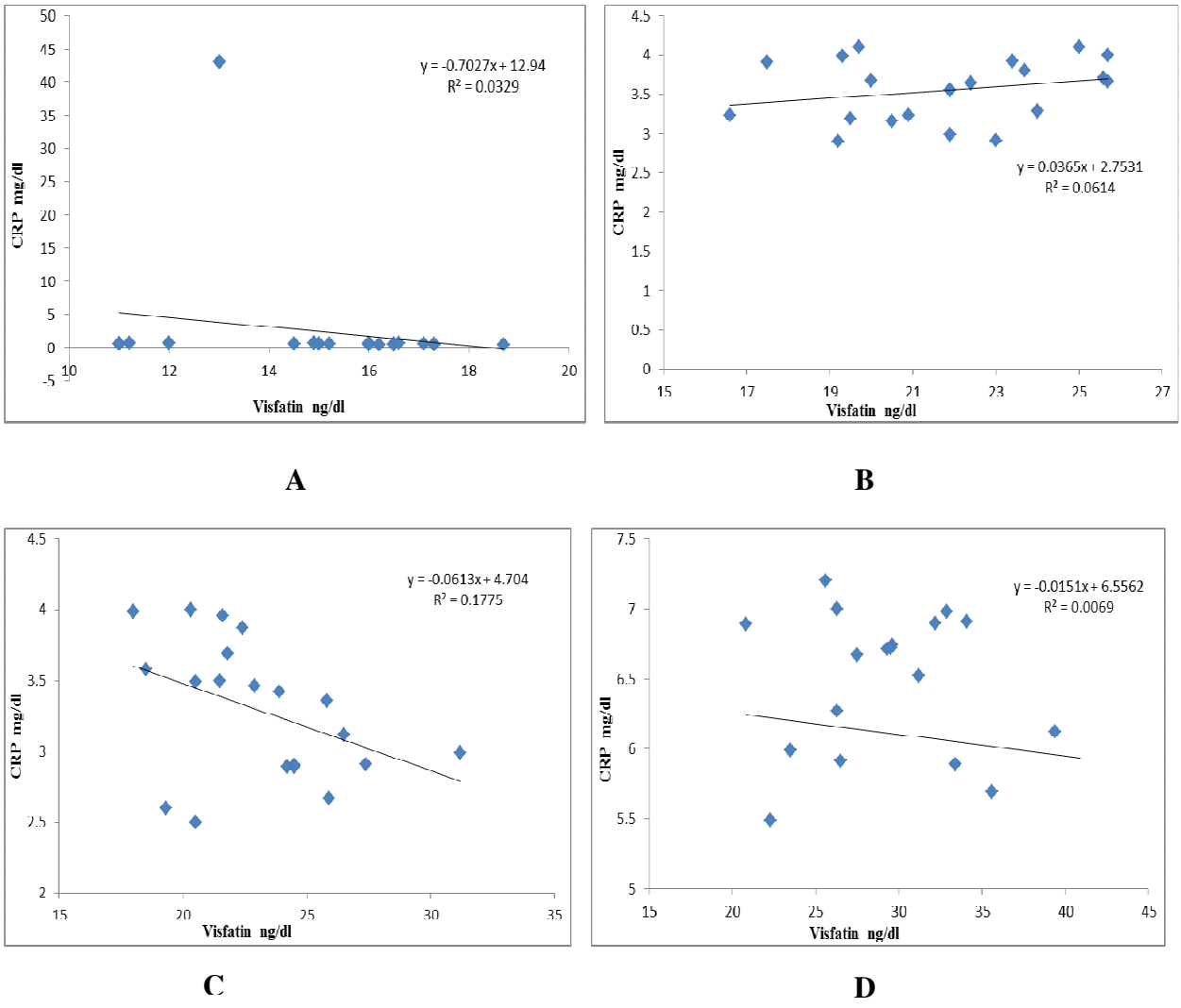

Figure (7) Correlation between visfatin and CRP inG1(A),G2(B),G3(C),G4(D)

The conclusion could be drown from this study that visfatin increased in G4 more than other groups comparing to G1 . Also, there are significant correlation between visfatin levels with insulin , HOMA-IR and CRP levels . As far as to our knowledge this is the first study determined the levels of visfatin in young male with normal BMI and find its relation with lipid profile in patients with hyperlipoproteinemia which divided according to Fredrecsons classification .

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