Assessment of Serum Visfatin Levels in Patients with Chronic Obstructive Pulmonary Diseases in Babylon-Iraq

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Summary

The presented study aims to evaluate of the levels of serum Visfatin, in patients with chronic obstructive Pulmonary diseases in Babylon-Iraq , to find the correlation of Visfatin, with lipid profile in these patients and compared with apparently healthy as case – control study. The study included (50) patients as patients group diagnosed with COPD (G1) and (50)as apparently control group(G2) .The age of all studied groups ranged between (40-65)years and BMI with (18.8-24.4) Kg/m^2.Serum was used in determination of FBS, lipid profile(Ch,TG,HDL-c,LDL-c and VLDL-c) , insulin , CRP and Visfatin , Whole blood was used for determination of HbA1C . The results revealed significant elevation in FBG and HbA1c levels were seen in patients groups when comparing to healthy control .The results indicates a significant elevation in TC, TG ,LDL-c and VLDL-c in G1 comparing to G2. Also , HDL-c significant decreased in G1 comparing to G 2 , Also ,the results showed significant increased in CRP levels in G1 , when comparing to G2, A non significant visfatin and insulin level in G1 when compared with G2(p value = 0.486) , . A non significant correlation was found in G1 between visfatin ,TC,TG, HDL-c and VLDL-c.

The conclusion from this study indicate that G1 which HDL-c is low in this group has the highest value for FBS , HbA1c , TC ,TG,LDL and VLDL .Also, the study found non correlation between lipid profile parameters in G1

Keywords: *COPD , visfatin , Lipid profile, CRP .*

Introduction

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable disease characterized by persistent respiratory symptoms and airflow limitation that is due to airway and or alveolar abnormalities, usually caused by significant exposure to noxious particles or gases. The chronic airflow limitation that characterizes COPD is caused by a mixture of small airway disease (e.g., obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person. Chronic inflammation causes structural changes, small airway narrowing, and destruction of lung parenchyma. A loss of small airways may contribute to airflow limitation and mucociliary dysfunction, a characteristic feature of the disease. Chronic respiratory symptoms may precede the development of airflow limitation and be associated with acute respiratory events [(1)].Visfatin, also called nicotinamide phosphoribosyltransferase (NAMPT) and previously identified as pre-B cell colony-enhancing factor (PBEF) is a 52 kDa protein encoded by the PBEF gene located on chromosome 7**.**It was originally discovered in lymphocytes, bone marrow, liver and muscles[(2)], but later identified in many other organs including the lungs[(3)].Leucocytes, especially granulocytes and monocytes, are the major sources of visfatin[(4)].

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Materials and Methods

This study included one hindered with aged ranged (40-65) years and BMI ranged between (18.8-24.4) Kg/m^2.Subjects were divided into two groups :

Group(1) consist of (50) as Patient group diagnosed with COPD.

Group (2):consist of (50) as apparently healthy control group.

The patients attended the Marjan Medical City in Babylon Province, Hilla city, from November 2018 till March 2019. .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 (15min) to clot then centrifuged at (3000 rpm) for (5min).The serum which obtained was freezed (-20°C) until analysis of lipid profile, visfatin, insulin and CRP .

Laboratory Tests

Glucose was determined after enzymatic oxidation in the presence of glucose oxidase (GOD)[5].Total serum cholesterol determined by utilizing a kit based on the enzymatic hydrolysis [6].The absorbance was recorded for the quinonimine (red complex) at 500 nm . The determination of TG based on the enzymatic hydrolysis. The intensity of the color formed is proportional to the triglycerides concentration in the sample . The lipoproteins of VLDL, and LDL contained in the serum sample were precipitates by the addition by the addition of (4%) phosphotungstic acid solution, which contain (10%) magnesium chloride (PH 6.2) .The supernatant obtained after centrifugation contains the HDL, from which the cholesterol can be determined by complementary kit used in determination of total serum cholesterol as described in reference [7].LDL-cholesterol and VLDLwere estimated indirectly by using Fried Ewald formula [8]:-

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

VLDL-c(mg⁄dl) =TG/5

Insulin and visfatin were determined by using ELIZA technique based on the sandwich method [9] , CRP were determined by using NycoCard CRP single test of solid phase, Sandwich-Format, immunometric assay , HOMA-IR insulin resistance calculated by using the following formula [10].

HOMA-IR: [fasting glucose × fasting insulin] / 22.5

This work was done in the Department of Biochemistry, College of Medicine /University of Babylon and in Hilla city Iraq.

Statistical Analysis

The results expressed as mean ± SD. Students t-test was applied to compare the significance of the difference between all the studied groups. P-value ($p<0.05$), ($p<0.001$) and ($p>0.05$) considered statistically significant, highly significant and non-significant respectively. The correlation coefficient (r) test is used for describing the association between the different studied parameter

Ethical Issues:

A-Approval by scientific committee of the Clinical Biochemistry Department, College of Medicine/ University of Babylon, Iraq.

B-Approval by Babylon Health Directorate, Ministry of Health and Information Centre for Research and Development of Babylon Province .

C-The objectives and methodology were explained to all subjects and verbal consent had been taken.

-Study Design

This study design was a case – control study.

The Results

The mean ± SD and T-test of descriptive parameters for COPD in the studied groups with aged ranged (40-65) years and BMI ranged between (18.8- 24.4) Kg/m^2 are presented in table (1).

Table (1): Descriptive parameter for G1 (patients) and G2 (controls).

Group parameter	G1)No. 50(Mean ± SD	G2)No. 50(Mean ± SD	T- Test,
FBS (mg/dL)	105.86 ± 20.0	89.25 ± 9.94	0.00001(S)*
HbA1c (%)	5.98 ± 0.52	5.76 ± 0.34	0.013(S)*
TC(mg/dL)	201.94 ± 53.86	173.46 ± 23.28	0.001(S)*
TG(mg/dL)	126.67 ± 73.91	89.21 ± 36.57	0.002(S)*
HDL(mg/dL)	36.87 ± 9.13	43.82 ± 10.13	0.001(S)*
LDL(mg/dL)	136.43 ± 49.47	111.79 ± 28.01	0.003(S)*
VLDL (mg/dL)	25.31 ± 14.78	17.84 ± 7.31	0.002(S)*

***(S) significant differences**.

The results in table (1) revealed significant elevation in FBS and HbA1C levels was found in patients groups when comparing to healthy control . The results indicate significant elevation in TC,TG, LDL and VLDL in G1 comparing to G2. Also The results , also, illustrated significant decreased in HDL in G1 when comparing to G2.

Systemic inflammation and steroid use could be important contributory factors responsible for both COPD and hyperglycemia[9]. Similarly, Mannino et al.,3 also found out that COPD patients with severe and very severe airflow obstruction had a higher prevalence of diabetes[10]. Dyslipidemia, a major risk factor for CHD and metabolic syndrome, is characterized by a cluster of lipid abnormalities such as an elevated level of triglyceride (TG), a reduced level of high-density lipoprotein cholesterol (HDL) and an increased level of low-density lipoprotein cholesterol (LDL). A number of studies have evaluated the relationship between COPD and blood lipid profiles with inconsistent results. While some authors reported reduced serum levels of HDL or increased serum levels of TG in COPD patients [13], [14], The objective of the present study was to investigate the association between COPD and the serum levels of FBS ,total cholesterol (TC), triglyceride (TG),HDL, LDL and VLDL. The prevalence of lipid profile abnormalities in COPD are different in studies and range of 9-50%have been reported. Smoking affects the lipid profile in the following ways. The plasma β-lipoprotein, cholesterol and triglycerides concentration are higher and HDL- c is lower in smoker than in nonsmokers [15].

Nicotine stimulates the release of adrenaline from the adrenal cortex leading to increased serum concentration of free fatty acids (FFA) which further stimulates hepatic synthesis and secretion of cholesterol as well as hepatic secretion of very low density lipoprotein (VLDL) and hence increased TGL.

Smoking decreases estrogen levels and further leads to decreased HDL cholesterol concentration. Also, HDL concentration was inversely related to VLDL concentration in serum.

Smoking increases insulin resistance and thus, causes hyperinsulinemia. LDL, VLDL and TGL are elevated in hyperinsulinemic conditions due to decreased activity of lipoprotein lipase. R. Gupta et al. study shows significantly higher LDL and significantly lower VLDL levels when compared to controls [16].

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

Table (2): Levels of insulin, HOMA-IR, hs-CRP& Visfatin in patients and controls.

groups parameters	G1 (No. 50) Mean ± SD	G2 (No. 50) Mean ± SD	T- Test
insulin	9.91 ± 4.63	9.40 ± 3.92	0.553(NS)*
HOMA - IR	2.57 ± 1.29	1.98 ± 0.89	0.009(S)*
hs-CRP	21.52 ± 29.55	4.01 ± 2.41	0.0001(HS)*
Visfatin	7.63 ± 4.19	8.31 ± 5.43	0.486(NS)*

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

The results in table (2) , also , showed significant increase in CRP levels for G1 when comparing to G1, but visfatin levels non- significant elevation was found in G1 when comparing to G2 .

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 . Other study results suggest that increased HOMA-IR index has significant association with COPD[17] , this study agreement with our result .

Results also, showed highly a significant increase in hs-CRP levels in G1 when compared with G2 (p value = 0.0001) , The patients with COPD had significantly higher leukocytes and CRP levels. Pinto-Plata et al. showed that CRP levels in COPD patients are significantly higher compared to the control group and the CRP level[18]. Two studies, one each reported by Smith et al. and Varma et al. have suggested that the serum visfatin levels are not associated with parameters of body composition or the insulin resistance , Taken together, it would appear that there is no relation between visfatin and insulin resistance[19,20] this study agreement with our result .

Relationships and correlation coefficients :

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

Table (3) : Correlation coefficient and p-value between visfatin levels and other parameters for patients and controls

Group parameters	G1 (No. 50)		G2 (No. 50)	
	r*	P-value	r	P-value
Visfatin vs TC	0.106	0.465(NS)	-0.320	0.024(S)
Visfatin vs TG	0.130	0.370(NS)	0.049	0.737(NS)
Visfatin vs HDL	0.207	0.150(NS)	-0.095	0.511(NS)
Visfatin vs LDL	0.054	0.711(NS)	-0.244	0.088(NS)
Visfatin vs VLDL	0.130	0.367(NS)	0.049	0.737(NS)
Visfatin vs insulin	0.093	0.521(NS)	-0.026	0.860(NS)
Visfatin vs HOMA-IR	0.119	0.412(NS)	0.016	0.914(NS)
Visfatin vs CRP	0.204	0.156(NS)	-0.253	0.076(NS)

*(S) Significant differences,(NS)No significant differences.

A non-significant correlation was found between visfatin levels and (TC) in G1(r = 0.106)), as shown in figure (1) ,and negative significant between visfatin with G2 (r =-0.320(.

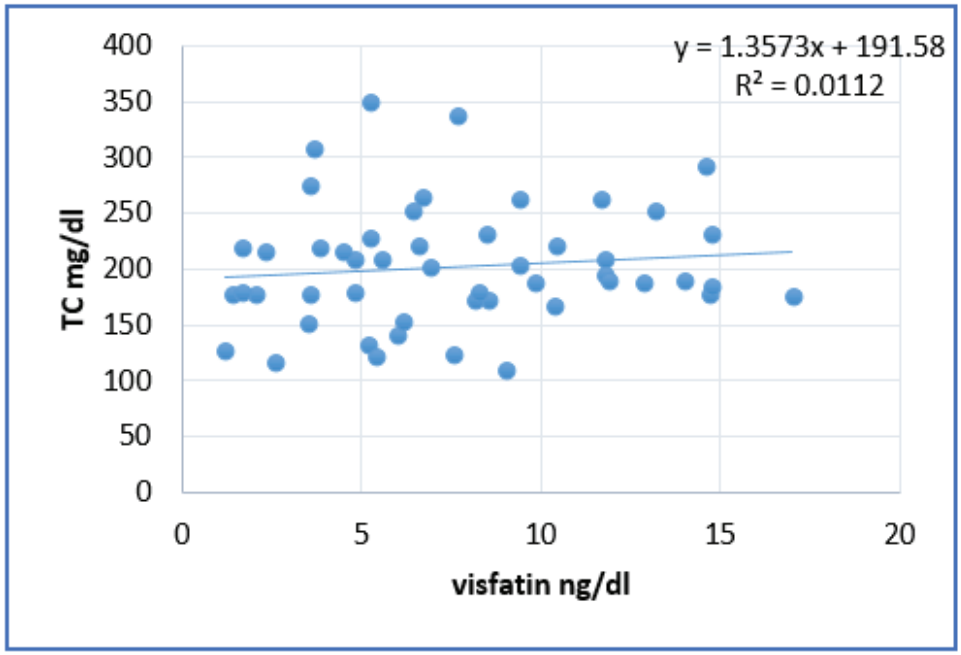

Figure (1) :Correlation between visfatin & TC in G1

A a non significant correlation coefficients was found between visfatin and TG in two studied groups (r = 0.130, r = -0.049) respectively

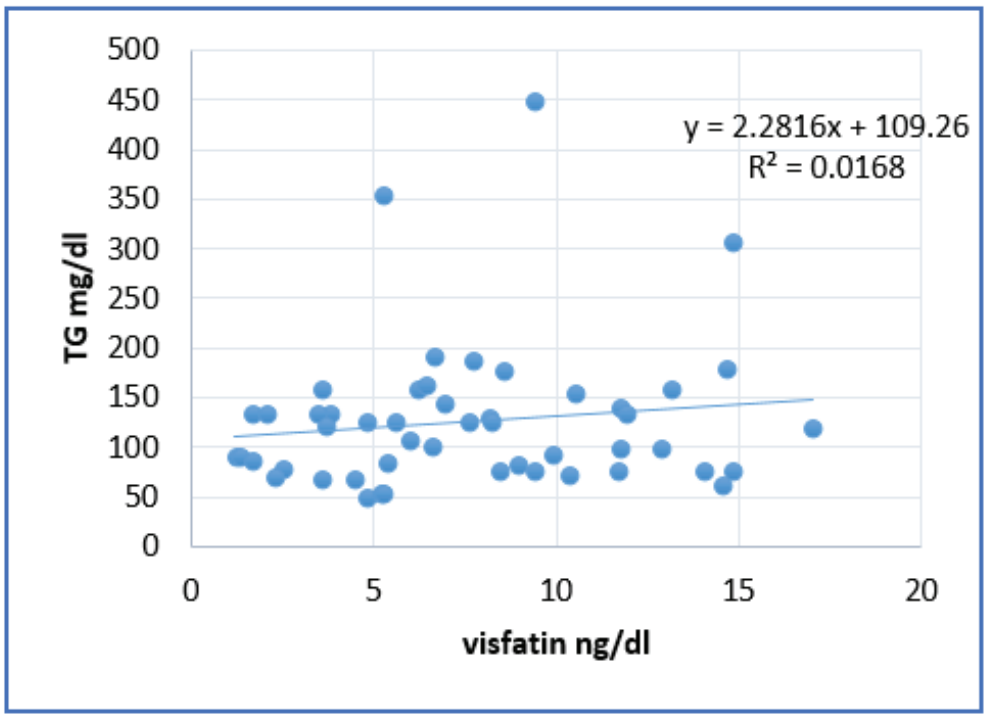

Figure (2) Correlation between visfatin and TG in G1

Results also , showed a non significant correlation between visfatin levels and HDL for all studied groups (r_1= 0.207, r_2 = 0.095)respectively , Results in table (3-3) illustrated a non significant correlation in visfatin levels with LDL in G1 (r = 0.054) and also in G2 (r = 0.244) . Also non significant correlation was found between visfatin levels with VLDL in G1 (r= 0.130) and G2 (r=0.049) .

A non-significant correlation between visfatin and insulin levels was found in G1(r=0.093) as shown in figures (3),A significant negative correlation between visfatin and insulin levels was found in G2 which (r = -0.026).

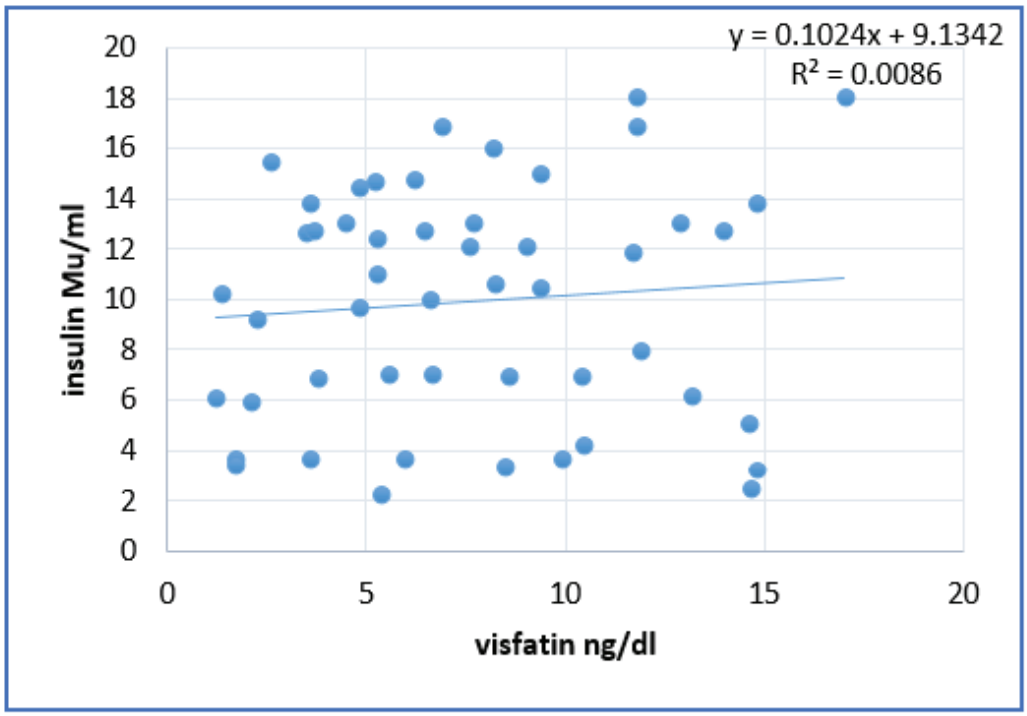

Figure (3) :Correlation between visfatin & Insulin in G1.

Also a non significant correlation between visfatin and HOMA-IR levels are shown in G1 which (r =0.119), while positive significant correlation between visfatin and HOMA-IR levels are shown in G2 which (r = 0.016). A non significant correlation was found in visfatin and hs-CRP levels in all studied groups which (r= 0.204, r = -0.253) respectively .

Conclusion

The conclusion could be drown from this study that visfatin non changed in G1 comparing to G2. Also, there are non-significant correlation between visfatin levels with insulin and CRP , while positive significant correlation between visfatin and HOMA-IR levels are shown in G2 which (r = 0.016) . As far as to our knowledge this is the first study determined the levels of visfatin in COPD patient with normal BMI and find its non-relation with lipid profile in patients with COPD

Source of Funding- Self

Conflict of Interest : the authors have nil conflict of interest

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