History of Medicine, 2029, 9(2): 356–361
DOI: 10.17720/2409-5834.v9.2.2023.049

Study the Effect of Dyslipidemia in Iraqi Diabetes Patients with Both Gender

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Received: 11 May 2023 **Accepted:** 10 June 2023

Citation: ALShaban SAW, Hashim RK, Bohan AH, Akram RS (2023) Study The Effect of Dyslipidemia In Iraqi Diabetes Patients with Both Gender. History of Medicine 9(2): 356–361. https://doi.org/10.17720/2409-5834.v9.2.2023.049

Abstract:

The aim of this article to Study The Effect of Dyslipidemia In Iraqi Diabetic Patients with Both Gender. Twenty hindered participant (50) male with dyslipidemia compared with (50) male without dyslipidemia and (50) female with dyslipidemia compared with (50) female without dyslipidemia. There was a highly significant increase of TC,TG and LDL-c in Male with dyslipidemia when compared with Male without dyslipidemia, while a significant decreased of HDL-c in Male with dyslipidemia when compared with Male Control, and a highly significant incased of TC,TG and LDL-c in female with dyslipidemia when compared with female without dyslipidemia .while a significant decreased of HDL-c in female with dyslipidemia when compared with female without dyslipidemia. It can concluded increased TC, TG and LDL-C levels, while decreased level of HDL-C in male and female with dyslipidemia these due to Lipids are a major role in the development of CVD, and controlling these factors has become a top priority for health policymakers of both genders.

Keyword:

Dyslipidemia, cardio vascular disease, T2DM, Gender.

In patients with type 2 diabetes, dyslipidemia is one of the most important modifiable risk factors for cardiovascular disease. The increased flux of free fatty acids in patients with DM2 is linked to insulin resistance, which causes dyslipidemia. Despite the significant frequency and associated complications of dyslipidemia in T2DM patients (1), Hypertriglyceridemia, decreased HDL cholesterol levels, and a larger concentration of LDL particles were the most common dyslipidemia patterns in T2DM patients (2, 3). Insulin resistance is connected to dyslipidemia in DM2, as is an increase in the flow of free fatty acids as a result of insulin resistance. Insulin resistance and hyperglycemia create hypertriglyceridemia in DM2, which results in overproduction of triglyceride-rich lipoproteins in the liver, poor clearance of triglyceride-rich lipoproteins, and, in some cases, a shift in postprandial lipoprotein metabolism (4, 5). Insulin resistance in DM is connected to a decreased ability of insulin to inhibit hormone-sensitive lipase in adipose tissue, resulting in increased lipolysis and, as a result, increased portal flow of free fatty acids to the liver. Increased FFA can cause lipoprotein lipase to shed from the endothelium membrane, causing it to change its function (6,7). As the availability of free fatty acids in the liver increases, apoB breakdown diminishes, resulting in an excess of very low-density lipoproteins in insulin resistance. Increased levels of triglyceride-rich lipoproteins are linked to lower levels of HDL and higher

levels of low-density LDL (8, 9). Because of growing consumption of poor foods, less physical activites, and urbanzation, obesity, the global burden of dyslipidemia in diabetics is constantly elevating (10,11).

Dyslipidemia is one of the risk factors for diabetic vascular problems because it increases the flow of free fatty acids as a result of insulin resistance, which is exacerbated by elevated levels of inflammatory adipokines (12). According to the Framingham Heart Study, among diabetics, high cholesterol was prevalent in 13% of men and 24% of women, and high plasma triglyceride levels were prevalent in 19% of men and 17% of women (13).

Young Arab populations have a high prevalence of CVD risk factors such as dyslipidemia and smoking. According to the Gulf Registry of Acute Coronary Events (Gulf RACE) 2, this group experiences acute myocardial infarction (AMI) 10-12 year earlier than their Western counterparts. In numerous communities around the world, high levels of low-density lipoprotein (LDLC) are a substantial independent risk factor for CVD. As a result, a number of guidelines consider LDLC to be the primary target of cholesterol-lowering medication (14, 15). However, a considerable proportion of patients receiving lipid-lowering medications (LLDs) in the Arabian Gulf, primarily those in the high-risk groups for CVD, are not meeting the recommended therapeutic goals for LDLC. The Centralized PanMiddle East Survey of Hyperchole sterolemia Subtreatment (CEPHEUS), which included 5,276 individuals with LLD from six Arabian Gulf nations, found this. In 91.1 percent of low-risk patients, 52.7 percent of high-risk patients, and 32.0 percent of extremely high-risk patients, the LDLC objective was met (16). The aim of this article is to investigate the effects of dyslipidemia in both males and females Iraqi diabetes patient.

Material s and Methods:

Two hindered participant (50) male with dyslipidemia compared with (50) male without dyslipidemia and (50) female with dyslipidemia compared with (50) female without dyslipidemia who visited national diabetes center/mustansiriyah university aged ranged 35-60 years .

Measurements

Anthropometric Measurement

1 Calculation of BMI was computed by dividing the subjects' weight (kg) by their height (m) (m2). (17).

Biochemical Evaluation:

1 Serum Total Cholesterol (TC) Measurement: Serum TC was measured using an enzymatic approach utilizing a cholesterol kit (18).

2 Determination of serum triacylglycerol (TG): Serum TG was determined using an enzymatic technique using a TAG kit (19).

3 Serum HDL-C was tested using an enzymatic technique using an HDL-C kit (20).

4 Cholesterol Concentration in Serum Low Density Lipoprotein (LDL-C)

Friedewald's equation (21) is approach for determining low-density lipoproteins that is used indirectly. It only applies when TG levels are less than (400 mg/dL).

LDL-C is calculated as TC - [HDL-C + TAG/5].

5 Blood urea determination: an enzymatic approach is used to determine blood urea (22)

6 Serum creatinine determination: an enzymatic approach is used to determine serum creatinine levels (23)

7 Uric acid determination: enzymatic measurement of serum creatinine

Discussion

Over the previous decade, CVD-related mortality has risen internationally, and it is now the leading cause of death, accounting for more than 40% of all deaths in 2014. (24). Controlling blood lipids, which are a major contributor to the onset of CVD, has risen to the top of health authorities' agendas around the world (25). The findings of this study show that gender specific patterns of dyslipidemia should be carefully examined in population-levels lipid control and CVD prevention efforts (26). The results of this study demonstrated that the prevalence of abnormal HDL-C differed significantly from that of TC, TG, and LDLC in terms of age (27).Males had a larger prevalence of low HDL-C than females, with a largely steady absolute difference, and both genders had two parallel curves with no discernible increasing or decreasing tendency with age (28,29). Hypertriglyceridemia raises the

risk of cardiovascular disease by 32% in men and 76% in women [30,31]. Diabetic dyslipidemia frequently occurs several years before type 2 diabetes, implying that impaired lipid disorder is an early event in the development of (CVD) in diabetes [32]. Insulin resistance reduces the activity of lipoprotein lipase, a key mediator of VLDL clearance. Although this action has a minimal impact on plasmatic triglyceride levels, it is a mechanism that is affected. Hepatic uptake of VLDL, IDL, and LDL is reduced in type 2 diabetes patients, resulting in longer plasma residence times for these lipoproteins [33]. The study discovered that multiple types of dyslipidemia were widespread among patients, with hypo-HDL-cholesterolemia being the most common. In this study, gender was a significant determinant of hypercholesterolemia, hypertriglyceridemia, and hypo-HDL-cholesterolemia; age was a significant determinant of hypercholesterolemia, hypertriglyceridemia, and hyper-LDL-cholesterolemia; and nationality was a significant determinant of hypertriglyceridemia (34).The link between hereditary variables and differences in LDL-C levels has been studied in depth. Diet, smoking, diabetes type 2, physical inactivity, and medications account for around half of the variation in HDL-C levels, while acquired variables such as diet, smoking, diabetes type 2, physical inactivity, and pharmaceuticals account for the other half. Furthermore, decreased apolipoproteins in HDL-C could explain why isolated low HDL-C is so common in people who eat low-fat diets. Our findings imply that medical and natural science students had a higher frequency of dyslipidemia than students in social science faculties (35,36). This could be related to the type of education offered at these institutions, which may encourage students to sit for long periods of time in order to read or work on computers. The findings of another study are similar to those of this one. According to another study,(37,38) , no significant link between dyslipidemia and eating behaviors or the intake of majority of the foods studied, a finding that is consistent with earlier research (39). Finally, diabetes is a risk factor for dyslipidemia and atherosclerosis, and there is evidence that atherosclerosis can start in childhood and progress to adulthood (40). It may be concluded that men and women with dyslipidemia have higher TC, TG, and LDL-C values, but their HDL-C levels are lower. as a result of lipids are a major determinant in the development of CVD, and controlling these parameters has become a top priority for health policymakers of both genders.

Results

Table (1): Demographic characteristics between Male with dyslipidemia and Male without dyslipidemia.

Parameter	Male with dyslipidemia No(50)	Male without dyslipidemia No(50)	P-Value
	Mean±SD	Mean±SD	
Age (Years)	46.34±5.61	52.14±9.57	0.126
High (cm)	167.74±6.11	1683.26±10.0	0.181
Weight (Kg)	79.46±7.70	70.98±8.85	0.143
BMI(Kg/m^2)	29.55±5.50	23.44±1.27	0.008

$p<0.05$ is significant, $p<0.01$ is high significant.

There was a significant of urea in Male with dyslipidemia when compared with control and a highly significant of FBS and HbAIC in Male with dyslipidemia when compared with Male without dyslipidemia while, no different appear in creatinin and uric acid between different group

Table(2): Biochemical measurement between Male with dyslipidemia and Male without dyslipidemia

Parameters	Male with dyslipidemia No(50)	Male without dyslipidemia No(50)	p-value
	Mean±SD	Mean±SD	
FBS (mg/dl)	196.66±19.35	176.48±35.21	0.01
HbA1c %	8.89±1.59	6.89±2.19	0.01
urea (mg/dl)	32.60±8.82	36.24±6.60	0.05
creatinine (mg/dl)	0.95±0.097	0.96±0.09	0.150
Uric acid (mg/dl)	5.58±0.90	5.21±0.76	0.136

There was a highly significant increase of TC,TG and LDL-c in Male with dyslipidemia when compared with Male Control, while a significant decreased of HDL-c in Male with dyslipidemia when compared with male without dyslipidemia in table 3.

Table(3): Lipid Profile measurement between Male with dyslipidemia and male without dyslipidemia.

Parameters	Male with dyslipidemia No(50)	male without dyslipidemia No(50)	p-value
	Mean±SD	Mean±SD	
TC(mg/dl)	258.46±33.28	208.0±45.50	0.01
TG(mg/dl)	201.46±76.622	151.0±36.01	0.01

HDL-c (mg/dl)	33.44±6.92	46.50±11.18	0.05
LDL-c (mg/dl)	173.55±38.81	123.0±44.89	0.01

p<0.05 is significant, p<0.01 is high significant.

In table 4 shows no significant scour of age ,high, weight and BMI in female with dyslipidemia when compared with Female without dyslipidemia.

Table (4): Demographic characteristics between Patients and control groups.

Parameter	Female with dyslipidemia No(50)	Female without dyslipidemia No(50)	P-Value
	Mean±SD	Mean±SD	
Age (Years)	46.89±6.11	46.34±5.61	0.122
High (cm)	167.56±6.44	167.74±6.11	0.155
Weight (Kg)	70.03±8.59	70.98±8.85	0.243
BMI(Kg/m^2)	27.70±2.10	23.80±2.12	0.05

p<0.05 is significant, p<0.01 is high significant.

As shown in table 5 there was a highly significant increased of FBS and HbA1c in female with dyslipidemia when compared with female Control, while no significant of urea and creatinin in female with dyslipidemia when compared with Female without dyslipidemia

Table(5): Biochemical measurement between Female with dyslipidemia and without dyslipidemia.

Parameters	Female with dyslipidemia No(50)	Female without dyslipidemia No(50)	p-value
	Mean±SD	Mean±SD	
FBS (mg/dl)	202.06±46.49	176.48±1.59	0.01
HbA1c %	7.78±1.47	5.89±1.59	0.05
urea (mg/dl)	36.26±7.23	40.36±0.95	0.06
creatinine (mg/dl)	0.93±0.14	0.95±0.09	0.153
Uric acid (mg/dl)	5.46±0.90	5.01±0.90	0.109

In table 6 shows a highly significant incased of TC,TG and LDL-c in female with dyslipidemia when compared with female Control .while a significant decreased of HDL-c in female with dyslipidemia when compared with female without dyslipidemia .

Table(6): Lipid Profile measurement between Female with dyslipidemia and Female without dyslipidemia.

Parameters	Female with dyslipidemia No(50)	Female without dyslipidemia No(50)	p-value
	Mean±SD	Mean±SD	
TC(mg/dl)	233.56±42.26	202.22±22.09	0.01
TG(mg/dl)	174.52±39.66	145.86±19.03	0.01
HDL-c (mg/dl)	34.66±7.24	44.44±6.90	0.05
LDL-c (mg/dl)	125.32±48.94	118.60±22.50	0.01

p<0.05 is significant, p<0.01 is high significant.

Comparison according age in male and female dyslipidemia in figure(1,2) shows when the age increased the dyslipidemia increased in both genders.

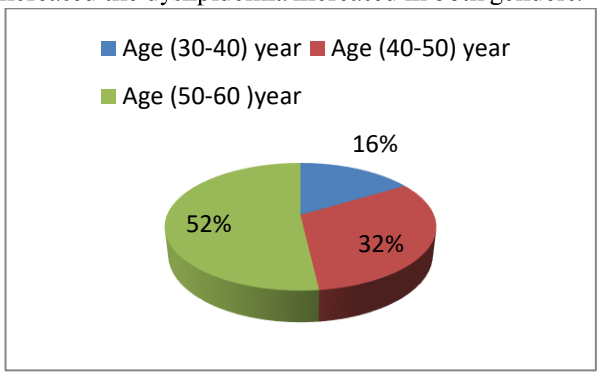

Figure (1): Comparison according age in male dyslipidemia.

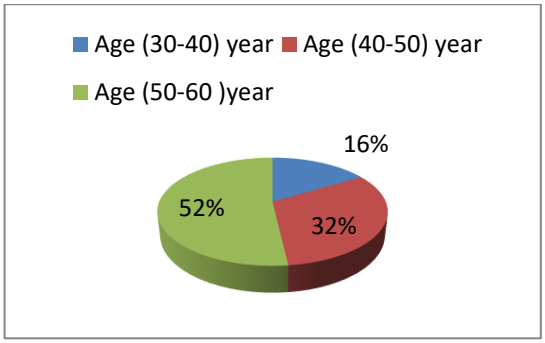

Figure (2): Comparison according age in Female dyslipidemia.

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