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The Relationship between Lipid Profile Levels and Cardiovascular Disease

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Abstract: Cardio vascular disease has become a great problem throughout the world. It is connected to increase morbidity and mobility and decrease quality of life in patients compared to the general population. many cases of disease has been described in cardiovascular disease. The study conducted on too Iraqi patient with chronic atherosclerosis at age rang (29-70years) in Baquba teaching hospital in (center care unit) during the period from 25 September 2020 to may 2021. The patients divided in (55 mans and 45 patients) the total patient divided in tow groups according the treatment (30) of total patient under go to treatment for two to three days after atherosclerosis and 70 patient in same day of diagnosis of atherosclerosis and observed increasing in the serum of total cholesterol, HDL cholesterol, VLDL, Triglyceride.

Introduction

Total Cholesterol

Cholesterol levels are precisely managed among the body's cells, as (50 percent) of cholesterol is produced in eukaryotic cells in the liver and small intestine, with the remaining amount received from food [1]. An abnormality in cholesterol metabolism might contribute to a plethora of disorders, the most serious of which is probably cardiovascular disease. In addition, cholesterol can be detected in the blood among the lipoprotein particles [2].

Low Density Lipoprotein (LDL):

Medium-density lipoproteins with all of their contents are changed into low-density lipoproteins, with VLDL forming the majority of the LDL generated by IDL. There is evidence that some of such particles are formed directly by the liver [1]. LDL, or low-density lipoprotein, is high in cholesterol, accounting for (40-50 percent) by weight; hence, LDL is an atherogenic lipoprotein.

Low-density lipoproteins connect to protein receptors found on the exterior of target cells for these particles. It enters the cell via the process of cellular endocytosis. Lysosomal lipases generate cholesterol, which either binds or binds to the cell membrane or is re-esterified for storage [2].

High Density Lipoproteins (HDL):

High-density lipoproteins are produced in the liver and intestine and expelled. They are the tiniest forms of lipoproteins and contain numerous varieties (such as HDL2, HDL3) that may be isolated from one another using a refrigerated centrifuge based on density differences[3, 4]. Nascent HDL is a newly produced high-density lipoprotein (HDL) composed of phospholipid and free cholesterol bilayers. Cholesterol is not degraded to provide energy, and it is taken from tissues by HDL when it

travels to the liver after being transformed into cholesterol ester by the enzyme lecithin: cholesterol acyltransferase (this is known as reverse cholesterol transport [5]. As a result of these processes, the level of plasma cholesterol which could be deposited in the arteries is reduced, and there is a significant inverse association between HDL cholesterol and the risk of numerous illnesses, including cardiovascular disorders [4].

Triglycerides (TGs):

TGs are the major storage site for highly concentrated metabolic energy produced from adipose tissue in the state of fatty acids and transferred to target tissues through blood. Dietary TGs are hydrolyzed in the gut, re-esterified in the enterocytes, conjugated with cholesterol and proteins to create chylomicrons, and then released into the bloodstream. Furthermore, TGs can be produced endogenously in the liver and discharged as very-low-density lipoproteins (VLDL). Elevated TG concentrations reported over the course of dyslipidemia are a well-known risk factor for cardiovascular disease [6].

Experimental Part

Determination of Human CHOLESTEROL:

PRINCIPLE:

Enzymatic method described by Allain and al., which reaction scheme

is as follows:

Cholesterol esters → Cholesterol + free fatty acids

Cholesterol + O2 → Cholesten 4 one 3 + H2O2

2 H2O2 + Phenol + PAP → Quinoneimine (pink) + 4 H 20

REAGENTS PREPARATION:

Use a non-sharp instrument to remove aluminum cap. Add promptly the content of vial R2 into vial R1. Mix gently until complete dissolution. Vial R3: Ready to use .

Assay PROCEDURE:

Manual method:

Let stand reagent and specimens at room temperature.

Reagent	1000 uL
Blank, Standard, Control or specimen	10 uL
Mix. Let stand for 10 minutes at room temperature or 5 minutes at 37°C. Record absorbances at 500 nm (480-520) against reagent blank. Color is stable for 1 hour.	

1- Performances with manual procedure should be validated by user.

2- Kenza applications and other applications proposal are available on request.

Determination of Human LDL-CHOLESTEROL

PRINCIPLE:

This technique ,uses a separation method based on the specific precipitation of low-density lipoproteins (LDL) by polyvinyl sulfate in whole serum, sedimentation of the precipitant by centrifugation, and subsequent test as residual cholesterol of the rest of lipoproteins (VLDL+ HDL) remaining in the clear supernatant. LDL-cholesterol is calculated by substracting the supernatant cholesterol fraccions from the total cholesterol of the sample.

REAGENT PREPARATION:

All the kit compounds are stable until the expiry date stated on the label. Store the vials tightly closed, protected from light and prevented contaminations during the use.

PROCEDURE:

I. Precipitation

1. Bring reagents and samples to room temperature.
2. Pipette into labelled centrifuge tubes:

Sample or Standard	0.2 mL
Precipitating reagen	0.1 m

Ratio = Sample / Reagent = 1/0.5
Dil. factor = 1.5

3. Vortex and allow to stand for 10 minutes at room temperature.
4. Centrifuge for 10 minutes at 6000 r.p.m., or 2 minutes at 12000 r.p.m.
5. Remove an aliquot of the supernatant for measurement of cholesterol.

Colorimetry

1. Bring the components of the kit and the components of the Cholesterol MR to room temperature
2. Prepare 2 series of tests to measure in parallel the total cholesterol of the sample and the remaining cholesterol in the supernatant. Follow for total cholesterol the instructions of the insert.
3. Pipette into labeled tubes:

TUBES	Blank	Sample Supernat	Standard Supernat
Monoreagent	1.0 mL	1.0 mL	1.0 mL
Supernate	_	50 µL	_ 50 µL
Standard	_	_	

The color is stable for at least 30 minutes protected from light.

Determination of Human TRIGLYCERIDES MR

PRINCIPLE:

The method1, 2 is based on the enzymatic hydrolysis of serum or plasma triglyceride to glycerol and free fatty acids (FFA) by lipoprotein lipase (LPL). The glycerol is phosphorylated by adenosin triphosphate (ATP) in the presence of glycerolkinase (GK) to form glycerol-3-phosphate (G-3-P) and adenosine diphosphate (ADP). G-3-P is oxidized by glycerophosphate oxidase (GPO) to form dihydroxyacetone phosphate (DHAP) and hydrogen peroxide. A red chromogen is produced by the peroxidase (POD) catalyzed coupling of 4-aminoantipyrine (4-AA) and phenol with hydrogen peroxide (H2O2), proportional to the concentration of triglyceride in the sample.

Triglycerides + 3 H2O → Glycerol + 3 FFA

Glycerol + ATP → Glycerol- 3-P + ADP

Glycerol-3-P + O2 → DHAP + H2O

4-AA + 4 Phenol → Quinoneimine + H2O

REAGENT PREPARATION:

The Monoreagent and the Standard are ready-to-use.

PROCEDURE:

1. Bring reagents and samples to room temperature.

2. Pipette into labelled tubes:

TUBES	Blank	Sample	CAL.Standard
R1. Monoreagent	1.0 mL	1.0 mL	1.0 mL
Sample	-	10 µL	-
CAL. Standard	-	-	10 µL

3. Mix and let the tubes stand 15 minutes at room temperature (16-25ºC) or 5 minutes at 37ºC.
4. Read the absorbance (A) of the samples and the standard at 500 nm against the reagent blank.

* The color is stable for at least 1 hour protected from light.

Result and discussion

The results of serum lipid profile in study groups (patients compared with controls)

The lipid profile (TRTCHIL, CHOL, VLDL, LDL, HDL) showed increased significantly in patients (169.49 ± 4.67, 255.53 ± 3.61, 40.69 ± 6.19, 187.83 ± 3.18, 35.63 ± 0.93 respectively) when compared with control (123.52 ± 6.40, 153.80 ± 4.57, 29.78 ± 2.13, 29.78 ± 2.13, 171.65 ± 8.58, 171.65 ± 8.58, and 31.77 ± 1.73 respectively) p-value <0.05 figure (1), (2) ,(3) , (4) and (5) table (1) , this result was agree with Maha Radhi Abess , et al.(2017) [7] were observed increasing in the serum of total cholesterol , HDL cholesterol , VLDL , Triglyceride .

In other study Dabei Fan ,*et al.(*2018) [8] observed relationship between high levels of lipid profile and type 2 diabetes to developing cardiovascular disease .

Table (1) lipid profile in study groups

Parameters	Group	Mean ± Std.	P-value
T.G	Control	123.52 ± 6.40	<0.000
	Patients	169.49 ± 4.67	
CHOL	Control	153.80 ± 4.57	<0.000
	Patients	255.53 ± 3.61	
VLDL	Control	29.78 ± 2.13	0.034
	Patients	40.69 ± 6.19	
LDL	Control	171.65 ± 8.58	0.031
	Patients	187.83 ± 3.18	
HDL	Control	31.77 ± 1.73	0.045
	Patients	35.63 ± 0.93	

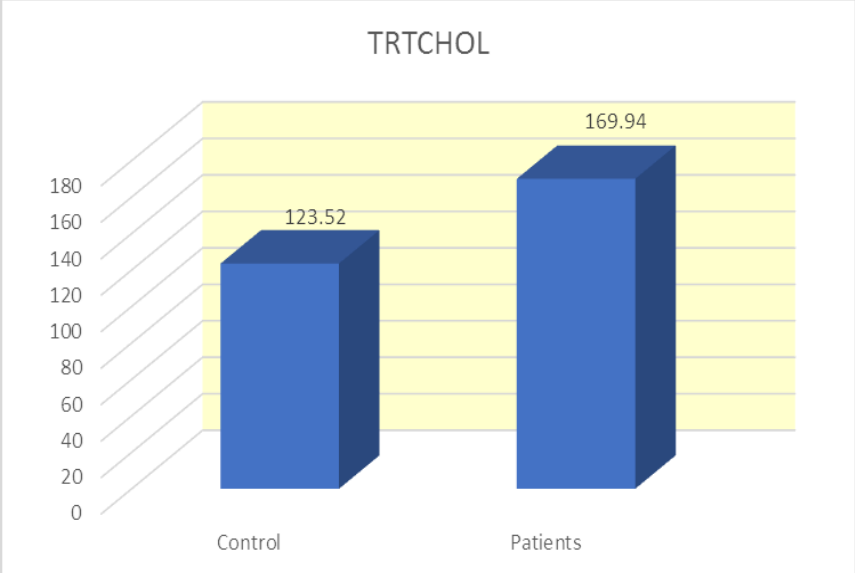

Figure (1) serum Tri Cholestrol level in study groups

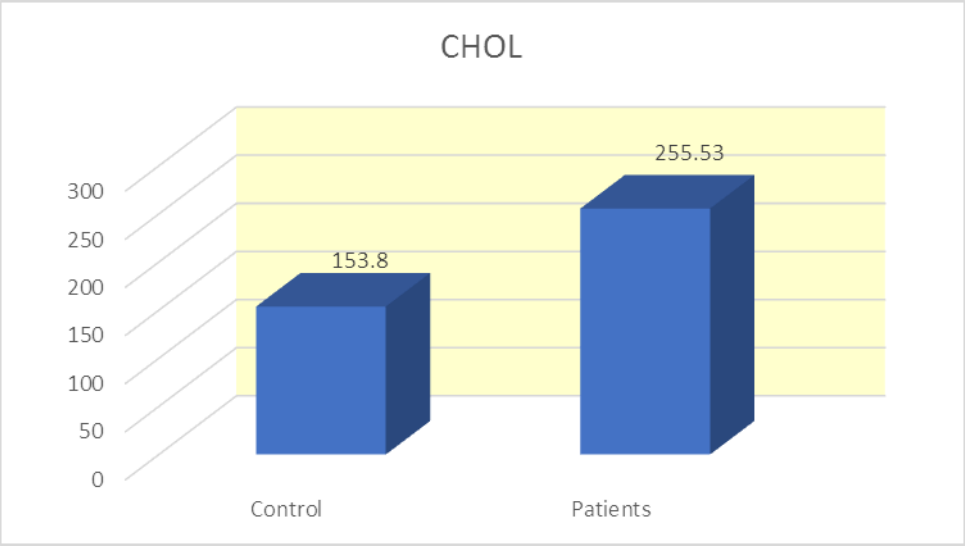

Figure (2) Serum CHOL level in study groups

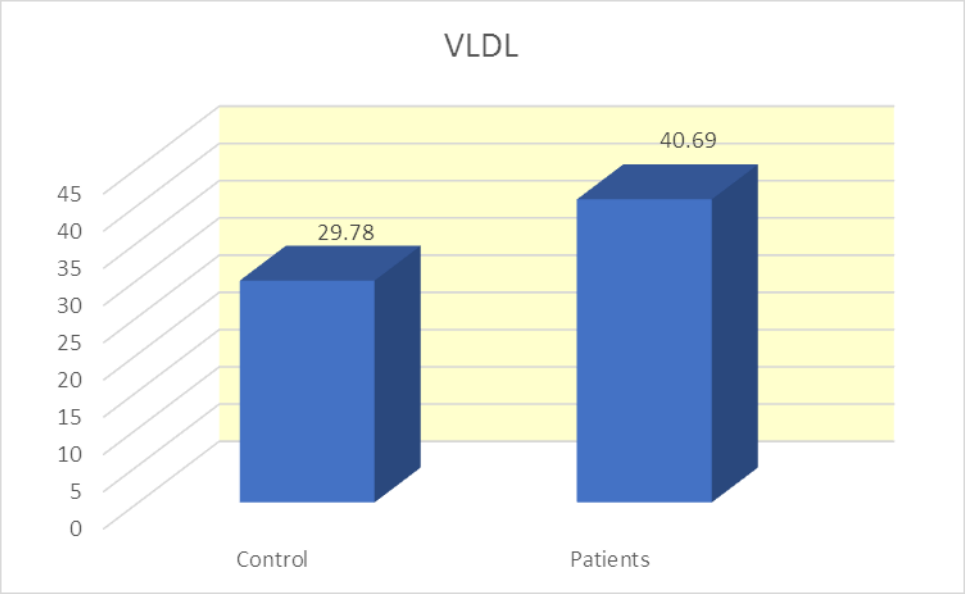

Figure (3) Serum VLDL level in study groups

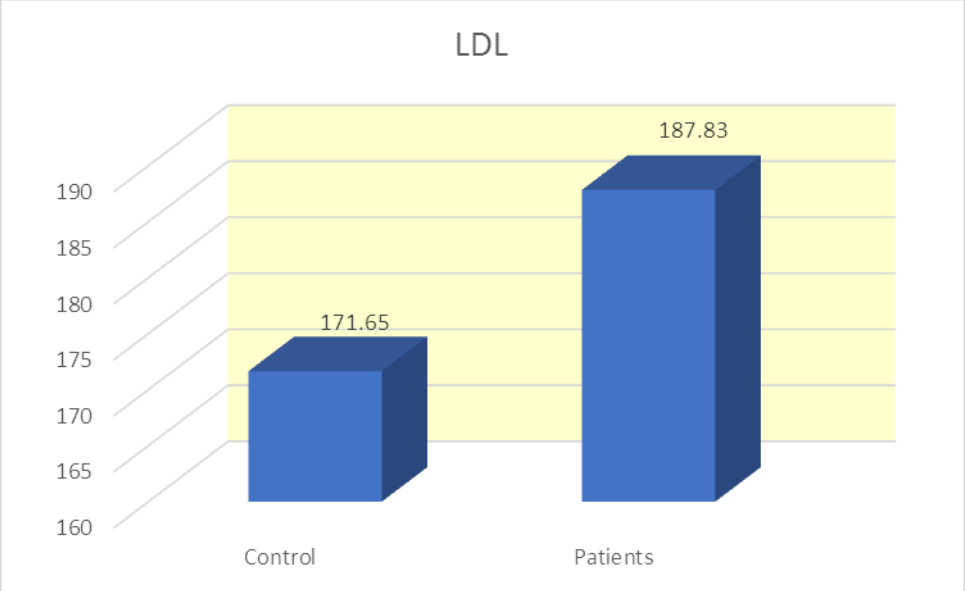

Figure (4) serum LDL level in study groups

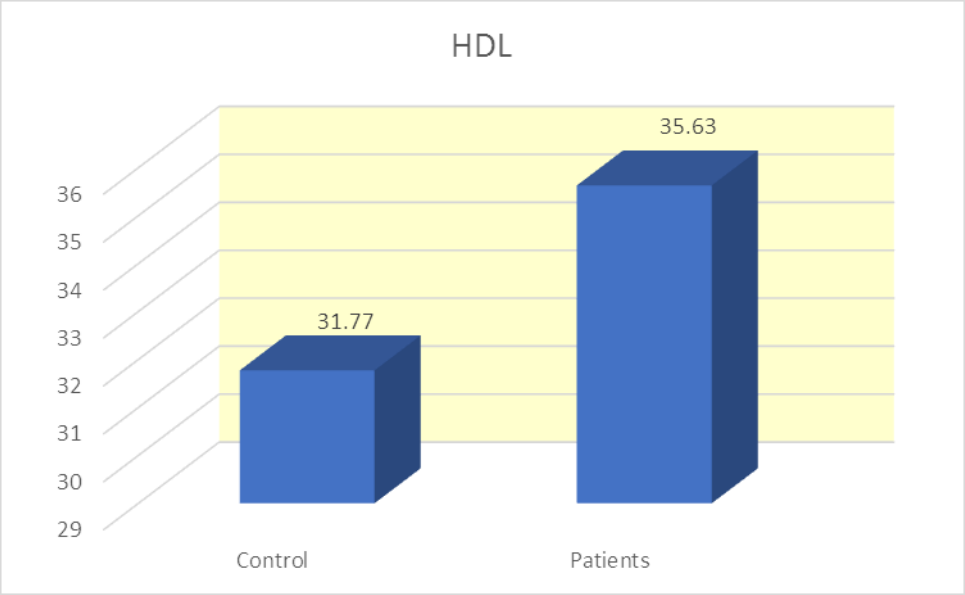

Figure (5) serum HDL level in study groups

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