

LIPID ALTERATION; DIABETIC AND OBESE (NON-DIABETIC) PATIENTS

ORIGINAL
PROF-1760

DR. KAMAL ELDIN AHMED ABDELSALAM

Faculty of Medical Laboratory Sciences,
Omdurman Islamic University-Sudan

DR. SHAZA SALIH TAHA

Faculty of Medical Laboratory Sciences,
Omdurman Islamic University-Sudan

DR. MANAHIL ALI SHARWANI

Faculty of Medical Laboratory Sciences,
Omdurman Islamic University-Sudan

ABSTRACT... Objective: determine serum lipid profile in diabetic and obese non-diabetic patients. **Materials:** 300 subjects comprising three groups, the first group included 100 diabetic patients (non-obese) with duration of diabetes over 5 years, the second group (obese) included 100 obese subjects (non-diabetic) with duration of obesity over 5 years, and the third group (control) included 100 healthy subjects. **Methods:** Lipid profile on the serum was performed with an auto analyzer using standard methods. **Study design and period:** Prospective Analytical Cross Sectional Study conducted in period from September 2006 to January 2008. **Results:** A significant (p value < 0.05) increase in lipid profile results of diabetic and obese patients comparing to control while HDL-C showed significant decreasing. The cholesterol is significantly increased in females than males in diabetic patients, while the triglycerides are significantly decreased in diabetic females. The parameters showed insignificant variations between males and females in obese patients. **Conclusions:** In comparison to control group, DM & obese groups showed significantly increase in TG, TC, and LDL-C, but significantly decrease in HDL-C. Females showed significant increasing in TC and significant decreasing in DM group, while in obese group females showed insignificant decreasing in TG and TC.

Key words: Total cholesterol, triglyceride, low density lipoprotein cholesterol, high density lipoprotein cholesterol, diabetes, obese

INTRODUCTION

Diabetes and obesity currently threaten the health, well-being and economic welfare of virtually every country in the world¹. Today, over 150 million people are suffering from diabetes worldwide (90% of them having type 2 diabetes) while over 300 million people are estimated to be obese. As a result, up to 1.7 billion of the world's population is at an increased risk of other life-threatening diseases such as heart attack and stroke². Diabetes is spreading worldwide as an epidemic. Diabetes mellitus is a group of metabolic diseases characterized by high blood glucose levels, which result from defects in insulin secretion, or action, or both. In type 2 diabetes, patients can still produce insulin, but do so relatively inadequately for their body's needs, particularly in the face of insulin resistance³. It is estimated that about 80% of those who develop type 2 diabetes are obese. When insulin attaches to the receptor protein, the receptor responds by adding a chemical called a phosphate group onto the IRS (insulin receptor substrate) molecules due to which the IRS molecules turn into action⁴.

Obesity means accumulation of excess fat on the body. Obesity is considered a chronic (long-term) disease, like high blood pressure or diabetes. It has many serious

long-term consequences for health, and it is the second leading cause of preventable deaths in many countries (tobacco is the first)⁵. Obesity is defined as having a body mass index (BMI) of greater than 30. The BMI is the body weight in kilograms divided by the square of the height in meters ($\text{weight} / \text{height}^2$). Healthy weight is defined as a BMI between 19 and 25 kg/m^2 . The National Institutes of Health recommends four BMI Categories:

Underweight	(BMI < 18.5).
Normal weight	(BMI = 18.5-24.9).
Overweight	(BMI = 25-29.9).
Obesity	(BMI = 30 or greater) ⁶ .

Although the exact biochemical mechanisms responsible for the association between obesity and the above diseases have not been completely elucidated, it is known that increase in triglyceride stores is associated with a linear increase in the production of cholesterol which in turn is associated with increased cholesterol secretion in bile and an increased risk of gallstone formation and the development of gall bladder diseases⁷. Similarly, increased levels of circulating triacylglycerol in obesity are associated with decreased concentrations of high-density lipoprotein, which may account for the increased risks for cardiovascular disease and heart

attack in obese patients⁸. Cardiovascular risk factor reports in obese individuals have recently demonstrated a remarkable number of metabolic abnormalities that embrace differences in lipids, glycemia, insulin, blood pressure, and hematologic function⁹. Diabetes and obesity, both, accelerate hardening of the arteries (atherosclerosis) of the larger blood vessels, leading to coronary heart disease (angina or heart attack), strokes, and pain in the lower extremities¹⁰.

The aim of this study was to determine and compare serum T.C, TG, HDL-C and LDL-C levels between diabetic and obese non-diabetic patients.

MATERIALS AND METHODS

Study area

The study was conducted in Khartoum state, Sudan.

Types of study

Prospective Analytical Cross Sectional Study

Sample size and Study population

This study was approved by the ethical committee of International Africa University. Informed consent was obtained from each participant.

Samples were taken from 300 subjects comprising three groups. The first group (DM) included 100 diabetic patients (non-obese) with duration of diabetes over 5 years, the second group (obese) included 100 obese subjects (non-diabetic) with duration of obesity over 5 years, and the third group (control) included 100 healthy subjects (non-diabetics and non-obese). The age of all participants was between (31-59) years, chosen among male and female with no history of smoking or biochemical evidence of hypertension, hyperlipidaemia, renal or liver disease or cancer.

Study period

September 2006- January 2008

Samples collection

5ml of overnight fasting blood collected from the antecubital vein, by venipuncture without venous stasis, in serum separator tube. Serum was separated after

20minutes and analyzed immediately after separation.

Methods

The concentration of total cholesterol (TC), triglyceride (TG), low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C) were measured using direct kit methods (from Bio-Diagnostics Kits, Spain)

Statistical analyses of data

Statistical analyses were performed using SPSS (Statistical Package for Social Sciences). Differences in mean values between groups were evaluated by a one-way analysis of variance (ANOVA) and Student's t-test. Two-tailed P-values were used and statistical significance was considered at $P < 0.05$.

RESULTS

Table-I. Lipid profile levels in DM, obese and control group

Parameters (m.mol/L)	Control	DM	Obese
Triglyceride	1.4	2.2	2.5*
Cholesterol	3.1	6	5.4*
HDL	1.3	1	0.9*
LDL	1.5	3.4	3.6*

* Significant changes

Table-II. Compare the results of lipid profile in DM and obese subjects according to sex

Parameters (m.mol/L)	DM		Obese	
	Male (N=60)	Female (N=40)	Male (N=71)	Female (N=29)
Triglyceride	2.3	4.7	5.7	5.5
Cholesterol	5.3	6.1	5.7	5.2
HDL	1	1.1	0.9	0.9
LDL	3.4	3.3	3.6	3.6

DISCUSSION

Hyperlipoproteinemia is regarded as one of the most important risk factors for the development of atherosclerotic diseases especially when cofounded

with diabetes and/or obesity¹¹. Studies indicate that in addition to the routine determinations of triacylglycerol and cholesterol, quantitative determination of the corresponding apolipoproteins is also important¹². It has been stated that these lipid abnormalities are often present before the clinical onset of diabetes and are known to become worse with the development of diabetic long-term complications such as nephropathy¹³.

In this study, in comparison to control group, both study groups (DM & obese) showed significant ($p < 0.05$) increased levels of triglyceride (TG), total cholesterol (TC), and low density lipoprotein cholesterol (LDL-C), while the level of high density lipoprotein cholesterol (HDL-C) significantly ($p < 0.05$) decreased. Our results were in agreement with those reports of Must et al¹⁴, and McNamara et al¹⁵.

Also as in table (I) the comparison between the DM and obese subjects revealed that there were no significant changes in HDL-C and LDL-C, while the TC is significantly increased in DM patient, and a significant increased in TG in obese group. Some studies were agreed with these findings¹⁶ but Wendy et al¹⁷ reported that the hyperlipidaemia in obesity is less severity than in diabetes mellitus and Sniderman et al¹⁸ reported that HDL-C does not decreased significantly in obese persons compared to normal healthy persons

The study groups (DM and obese) are classified according to sex. As consistent with other studies¹⁹ this study demonstrated that insignificant variations ($p > 0.05$) were observed in HDL-C and LDL-C in the serum of both study groups between men and women. In DM group, males showed significant higher results in TG while TC in females is significantly increased. But in obese group the cholesterol in men is significantly raised than in female, while TG showed insignificant changes. Smith et al²⁰ report were agreed with our findings.

CONCLUSIONS

In comparison to control group, DM & obese groups showed significantly increased in TG, TC, and LDL-C, but significantly decreased in HDL-C. Females showed significant increasing in TC and significant decreasing in

DM group, while in obese group females showed insignificant decreasing in TG and TC.

ACKNOWLEDGMENT

We gratefully acknowledge the help and support from laboratory of Sudanese Armed Forces Hospital for providing facility to work for this project.

Copyright© 03 August, 2011.

REFERENCES

1. **Diabetes Prevention Program Research Group. 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study.** *Lancet*. November 2009; 374: 1677-1686.
2. Westlund K, Nicolaysen R. **Ten³/year mortality and morbidity related to serum cholesterol.** *Scand J Clin Lab Invest* 1972; ((Suppl)) 30: 1-24.
3. **Centers for Disease Control & Prevention. Differences in prevalence of obesity among black, white, and Hispanic adults – United States, 2006 – 2008.** *MMWR Morb Mortal Weekly Rep* 2009;58(27):740-744.
4. Miettinen H, Lehto S, Salomaa V, Mahonen M, Niemela M, Haffner SM, Pyorala K, Tuomilehto J, for the FINMONICA **Myocardial Infarction Register Study Group: Impact of diabetes on mortality after the first myocardial infarction.** *Diabetes Care* 21: 69-75,1998.
5. Bray GA. **Pathophysiology of obesity.** *Am J Clin Nutr* 1992; 55: 4885-4945.
6. Kim AH, Brian JC, Sean K, et al. **The Association between Body Mass and Health Care Expenditures.** *Clin Ther.* 1997; 19: 811-820.
7. Finkelstein EA, Trogon JG, Cohen JW, Dietz W. **Annual medical spending attributable to obesity: payer and service estimates.** *Health Affairs* 2009;28(5)w822-w831.
8. Ian J Martinsa, Trevor G Redgraveb. **Obesity and post-prandial lipid metabolism.** *Feast or famine?* (March 2004) 15(3): 130-141.
9. Barnes P. 2009 National Health Interview Survey, **Centers for Disease Control & Prevention**, National Center for Health Statistics.
10. Garrow JS, Webster J: **Outelets index (w/H2) as a measure of fatness.** *International Journal of obesity* 1985, 9:147-153.

11. Syvanne M and Taskinen MR. **Lipids and lipoproteins as coronary risk factors in non-insulin dependent diabetes mellitus.** Lancet, 1997; 350: 20–23.
12. Akanji AO. **Diabetic Dyslipidaemia in Kuwait.** Med. Principles Pract., 2002; 11: 47–55.
13. Albers JJ, Marcovina SM, Imperatore G, Snively BM, Stafford J, Fujimoto WY, Mayer-Davis EJ, Petitti DB, Pihoker C, Dolan L and Dabelea DM. **Prevalence and determinants of elevated apolipoprotein B and dense lowdensity lipoprotein in youths with type 1 and type 2 diabetes.** 1: J. Clin. Endocrinol. Metab., 2008; 93: 735–742.
14. MustA, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. **The disease burden associated with overweight and obesity.** JAMA. 1999;282:1523–1529.
15. McNamara JR, Shah PK, Nakajima K, Cupples LA, Wilson PW, Ordovas JM, Schaefer EJ. **Remnant lipoprotein cholesterol and triglyceride reference ranges from the Framingham Heart Study.** Clin Chem. 1998;44(pt 1):1224–1232.
16. Krauss RM. **Relationship of intermediate and low density lipoprotein subspecies to risk of coronary artery disease.** Am Heart J. 1987;113:578–581.
17. Wendy M. Miller, Katherine E. Nori-Janosz, Martin Lillystone, Jose Yanez and Peter A. McCullough. **Obesity and lipids.** Current Cardiology Reports. 7(6); 465–470.
18. Sniderman AD, Pedersen T, Kjekshus J. **Putting low-density lipoproteins at center stage in atherogenesis.** Am J Cardiol. 1997;79:64–67.
19. Palumbo PJ: **Metformin: effects on cardiovascular risk factors in patients with non-insulin-dependent diabetes mellitus.** J Diabetes Compl 12:1 10–119, 1998.
20. Smith JW, Marcus FI, Serokman R: **Prognosis of patients with diabetes mellitus after acute myocardial infarction.** Am J Cardiol54: 718–721,1984.

Article received on: 17/03/2011

Accepted for Publication: 03/08/2011

Received after proof reading: 02/12/2011

Correspondence Address:

Dr. Kamal Eldin A Abdelsalam
 Assistant Professor
 Department of Chemical Pathology
 Faculty of Medical Laboratory Sciences
 Omdurman Islamic University, Sudan
 PO Box: 504 Khartoum - Sudan
 kamaleldin55@yahoo.com

Article Citation:

Abdeislam KEA, Sharwani MA, Taha SS. Lipid alteration; Diabetic and obese (non-diabetic) patients. Professional Med J Dec 2011;18(4): 663-666.

**BEWARE OF LITTLE EXPENSES.
 A SMALL LEAK WILL SINK
 A GREAT SHIP.**

BENJAMIN FRANKLIN