# A STUDY OF SERUM LIPID PROFILE IN OBESE NIDDM PATIENTS

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**Abstract**

Diabetes mellitus is a complex disease where the carbohydrate and fat metabolism is impaired. Most cases of hyperlipidaemia are secondary to an abnormal dietary intake ,or to underlying diseases in which lipid metabolism is affected ;these including obesity ,since obesity dose contribute in a considerable way to complications of diabetes .We though it would be worthwhile to examine the serum lipid profile in obese Type II diabetics with the control. This study was conducted on 56 obese NIDDM and 52 obese control with normal glucose tolerance test .Age range of the patients and controls were 30-65 years and their body mass indexes (BMI)>27Kg/$m^2$. lipid profile was estimated including total cholesterol ,triglyceride HDL-cholesterol, LDL-cholesterol, and VLDL-cholesterol. It was found a significant increases ($p<0.001$) in the lipid profile except in HDL-cholesterol which is decreased significantly ($p<0.01$).The result showed that most of the NIDDM diabetic patient, have elevated cholesterol while some of them have elevated triglycerides. The consequence is usually an elevation cholesterol, triglycerides and reduced HDL-cholesterol. This effect is amplified by obesity.

## Introduction

The first systematic description of diabetes mellitus was written by the Arelaeus of cappadosis in Asia minor ,probably in the first century AD, the disease as "A melting down of flesh into the urine " Van Mering and Minikowaki in 1889 reported that pancreactomy cases a metabolic disorder called Diabetes mellitus is a result of insulin deficiency [1]. Diabetes mellitus (DM) is a multifactor disease which is characterized by hyperglycemia, lipoprotein abnormalities and altered intermediary metabolism of major food substrates [2]. It characterized by either the absence of insulin that is NIDDM-Type I or which is insensitive to the insulin that is IDDM or type II [1]. It is a complex disease where the carbohydrate and fat metabolism is impaired [3]. Insulin affects many sites of mammalian lipid Metabolism. It stimulates synthesis of fatty acid in liver, adipose tissue and in the intestine. The insulin has also been reported to increase the cholesterol synthesis. The activity of lipoprotein lipase in white adipose is also increased [4].Some of the obese persons express resistance to insulin and increase fat synthesis and up taken. The obese person responds to a carbohydrate meal with increased insulin and decreased utilization of free fatty acids [5]. The interrelationship between obesity and insulin resistance , dyslipema ,and hypertension may contribute to cardiovascular risk, but is not known to what extent[6].Many studies have revealed, for instance ,that insulin resistance is not a necessary component of obesity [7,8]. In this study we examined the serum lipid profile in obese with NIDDM Type II diabetics and compared with a healthy control.

### Materials and methods

This study was conducted on 56 (26 females, 30 males) obese NIDDM patients and 52 (26 females,26 males) control with normal glucose tolerance test (GTT) [9]. Age of the patients &controls was 30-65 years. Patients with any concurrent sickness like chronic liver disease, hypothyroidism were excluded. Patients on drugs like diuretics, oral contraceptives (women) were excluded from the study. Body mass index (BMI) was calculated from the formula;

BMI=weight in kilograms/ (height in meters)$^2$, according to world healthy organization(WHO). Criteria patients were taken as obese if their BMI >27Kg/$m^2$ [10]. The fasting and postprandial blood sample, 2 ml in floride bulb for sugar estimation and 5 ml in plain bulb for lipid profile estimation were collected from the cubital vein. Serum

was separation within 30 minutes of sample collection by centrifugation at 3000 xg for 1-2 minutes.

**Blood Sugar**:Enzymatic, GOD-POD, endpoint colorimetric, single reagent chemistry. (autospan kit method).

**Total cholesterol**: Enzymatic Colorimetric Kit method, BioMerieux-France

**Serum Triglycerides**: Enzymatic Colorimetric Kit method, BioMerieux-France.

**HDL- cholesterol**:CHOD-POD kit method, BioMerieux- France.

**LDL-Cholesterol:** concentration was determined from the following equation:

LDL-cholesterol=Totalcholesterol-

(Triglycerides/2.825)-HDL cholesterol

**VLDL cholesterol** concentration was determined by dividing triglyceride value obtained on 2.825 when concentration given in mmol/L [11].

**Statistical analysis**

The results were expressed as mean ± SD and analyzed statistically, the different between the results of patients and control group were assessed by students t test. Significant variation was considered when the p value was less than 0.05.

**Results**

The study was carried out on 56 obese NIDDM patients and 52 healthy obese non-diabetic control subjects in chemistry department, science college, Kufa uneversity and, Alhakem hospital, in Najaf, in Iraq. Incidence of diabetes mellitus is 57.143% in age group 40-49 yrs, and 23.214% of diabetes in age group 30-39 yrs.

***Table (1)***
***Age and sex distribution of study subjects.***

| Age (years) | Sex | | Total | Perce. (%) |
|---|---|---|---|---|
| | Male | Female | | |
| 30-39 | 8 | 3 | 11 | 19.64 |
| 40-49 | 19 | 13 | 32 | 57.14 |
| 60&above | 7 | 6 | 13 | 23.21 |
| Total | 34 | 22 | 56 | 100.01 |

***Patients were taken as obese if their BMI >27Kg/m$^2$***

***Table (2)***
***Anthropometric parameters of the subjects.***

| | | Weight /kgs (mean ±SD) | Height/m (mean ±SD) | BMI (mean ±SD) |
|---|---|---|---|---|
| Obese NIDDM | Male | 78.35 ± 6.95 | 1.86 ± 0.09 | 27.75 ± 1.04 |
| | Female | 80.34 ± 8.3 | 1.72 ± 0.05 | 27.05 ± 0.74 |
| Obese control | Male | 85.95 ± 7.6 | 1.77 ± 0.11 | 27.46 ± 0.66 |
| | Female | 79.05 ± 9.3 | 1.68 ± 0.08 | 27.79 ± 1.82 |

The mean value of total cholesterol, serum triglyceride, LDL-cholesterol and VLDL-cholesterol in diabetic group is increased significantly ($p < 0.001$) compared to control group . Mean value of serum HDL- cholesterol is decreased statistically significant ($p < 0.01$), in diabetic group compared to control.

***Table (3)***
***Serum lipid profile in diabetic patients and control group.***

| Lipid fractions | Controls (n=53) | Diabetics (n=56) | P Value |
|---|---|---|---|
| Total cholesterol (mmol/L) | 4.50±0.38 | 7.74±3.68 | P<0.001 |
| Triglyceride (mmol/L) | 1.16±0.22 | 3.20±2.25 | P<0.001 |
| HDL-cholesterol (mmol/L) | 0.92±0.08 | 0.75±0.58 | P<0.01 |
| LDL-cholesterol (mmol/L) | 2.87±0.35 | 4.86±2.78 | P<0.001 |
| VLDL-cholesterol (mmol/L) | 0.71±0.21 | 1.45± .11 | P<0.001 |

**Discussion**

Hyperlipidaemia as a metabolic abnormality is frequently associated with diabetes mellitus .Its prevalence is variable, depending on the type and severity of diabetes, glycaemic control, nutritional status ,age and other factors. The most characteristic lipid abnormality in diabetics is hypertriglyceridaema, with or without associated increase

in plasma cholesterol [13-15]. In our study, obese diabetics when compared to obese control subjects showed statistically significant increase in levels of serum total cholesterol ($p<0.001$), serum triglycerides($p<0.001$), serum LDL-cholesterol ($p<0.001$),and VLDL-cholesterol ($p<0.001$). Serum HDL-cholesterol levels decreased significantly in obese patients when compared with control subjects ($p<0.01$). Cohen *et al* (1979) showed significant increase in the level of serum cholesterol and LDL-cholesterol in obese diabetics when compared with obese controls. In their study, serum HDL- cholesterol levels did not differ significantly in the two groups [16]. Sharma (1970) and Jain (1980) observed increase in the levels of serum total lipids, total cholesterol, serum triglycerides and serum phospholipids in diabetic subjects as compared to normal controls [17,18] .In contrast, there are studies which seem to suggest that the lipoprotein distribution in Type II diabetes mellitus is not significantly altered by the degree of metabolic control [19-21].We have found that serum cholesterol is increased in group of diabetes when compared with the controls. Some of the possible reason of higher concentration of serum cholesterol in diabetes may be attributed to decrease muscular exercise or inhibition of cholesterol catabolism. It has been suggested that the increase in triglyceride may be due to insulin deficiency which results faulty glucose utilization, causes hyperglycemia and mobilization of fatty acids from adipose tissue .In diabetes blood glucose is not utilized by tissue resulting in hyperglycemia. The fatty acid from adipose tissue are mobilized for energy purpose and excess fatty acid are accumulated in liver ,which are converted to triglyceride [22]. The present study indicate that insulin increases the number of LDL receptor ,so chronic insulin deficiency might be associated with a diminished level of LDL receptor. This causes the increase in LDL particles and result in the increase in LDL-cholesterol value in diabetes mellitus. It seems that atherosclerosis begins with tiny tears at stressed places in the walls of the arteries [23]. Low density lipoproteins from the blood enter these tears, where their chemistry changes enough to leave cholesterol behind. The cholesterol causes irritation; the body responds with inflammation; damage and scarring follow .Eventually the artery gets so diseased blood cannot flow through it. Strokes and heart attack are the result [24].But if there are lots of HDL in the blood, the cholesterol is rapidly picked up and not allowed to cause problems. The present data of serum lipid profile measurement indicated a atypical pattern of mixed hyperlipidemia. The glysimic status has a serious influence of serum lipid profile diabetics [23]. The evaluation of the results of this study indicated that generally hyperlipidemia is more aggressive in obese NIDDM patients .It was some what less aggressive in obese subjects. Realizing that in an individual who is obese and diabetic (two strong risk factors for coronary artery disease) their lipid abnormalities be properly taken care of ,if morbidity and a mortality in diabetic is to be significantly altered. Thus the detection of risk factor in the early stage of the disease will help the patient to improve and reduce the morbidity rate.

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**الخلاصة**

داء السكري هو مرض معقد فيه يضطرب أيض الكاربوهيدرات و أيض والدهون، من ناحية أخرى معظم حالات فرط الدهنيه في الدم هي حاله ثانوية لحالات التناول غير الطبيعي للغذاء أو نتيجة للأمراض التي تؤثر في ايض الدهون ،والتي تتضمن زيادة الوزن .ولان زيادة الوزن تساهم بشكل مؤثر في مضاعفات السكر .لذلك وجدنا إنه من المجدي إجراء فحص مستويات الدهون المصلية لدى المرضى المصابين بالسكري من النوع الثاني المتصفين بزيادة الوزن ،مع مراعاة مجموعة السيطرة . تضمنت الدراسة 56 مريض مصاب بداء السكري من النوع الثاني غير المعتمد على الأنسولين والمتصفين بزيادة الوزن و52 شخص متصف بزيادة الوزن شرط كون فحص تحمل السكر طبيعي لديهم .أعمار المرضى والأصحاء تتراوح بين 30−65 سنة ومعدل كتلة الجسم لديهم أكبر من 27 كغم/م$^2$.تم تقدير الكلسريدات الثلاثية المصلية، الكوليستيرول، الكوليستيرول HDL، الكوليستيرول LDL، والكوليستيرول VLDLفي الأشخاص الأصحاء والمرضى .بينت النتائج زيادة معنوية ( $p<0.001$) في تركيز الدهون المصلية جميعها فيما عدا الكوليستيرول HDL والذي قل بصورة

معنوية ( $p<0.01$). تشير النتائج أنه معظم الأشخاص المصابين بالسكري يرتفع مستوى الكوليستيرول وبعضهم يزداد مستوى الكلسريدات الثلاثي كذلك عن المستوى الطبيعي. والسبب في ذلك يرجع إلى تغير طريقة الجسم في الحصول على الطاقة التي يحتاجها وكذلك تتغير طريقته في معالجة الدهون .بالتالي فمن الطبيعي زيادة الكوليستيرول والكلسريدات الثلاثية ونقص الكوليستيرول HDL،هذة النتائج تزداد في حالات زيادة الوزن.