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Research Article

ESTIMATION OF LIPID PROFILE IN TYPE 2 DIABETES MELLITUS

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ABSTRACT
Impaired Lipid Metabolism resulting from uncontrolled hyperglycemia has been implicated in cardiovascular complications in diabetes patients. In the present study the impact of glycaemic control on the lipid profile of diabetic patients is examined. Lipoprotein levels are measured in non-diabetic persons and NIDDM (Type 2 DM) patients with good control and poor control of Diabetes mellitus (DM). The present study also reveals that reducing LDL cholesterol is beneficial in reducing CAD risk and lowering of LDL cholesterol being a primary target in the prevention of CAD.

Key Words: *NIDDM (Non-Insulin Dependent diabetes Mellitus), LDL (Low Density Lipoprotein), CAD (Coronary Artery Disease)*

INTRODUCTION
Brown and Goldstein (1994) suggested that Cholesterol is a major component of all mammalian plasma membranes and is vital for cell growth and survival.
However excessive amount is lethal as cholesterol deposition in arterial cell walls potentiates development of atherosclerosis. The mammalian cells are faced with dual problem of providing sufficient cholesterol for membrane growth and replenishment and at the same time avoiding excess accumulation of this insoluble substance.
LDL receptors bind the major cholesterol carrying lipoprotein and regulate the rate at which lipoprotein transfers cholesterol into the cell receptor is under feedback control and is regulated by amount of cholesterol that enters the cells.

Mazzone *et al.,* (1984), shows that Diabetes can affect LDL metabolism in several ways. Insulin is known to increase number of LDL-receptor thus chronic insulin deficiency might be associated with diminished number of LDL receptors, with consequent delayed clearance of this highly atherogenic particle.

Witztum *et al.,* (1982) studied a second factor which may alter LDL binding to its receptor is glycosylation of LDL – Apo B secondary to hyperglycaemia this occurs in lysine residues which are crucial to LDL – binding thus non-enzymatic glycosylation of LDL – Apo B occurs in poor diabetic control and probably interferes with normal LDL catabolism mediated by LDL receptor.

Hiramatsu *et al.,* (1985) demonstrates that LDL – isolated from patients with hypertriglyceridemia showed decreased receptor binding to cultured skin fibroblasts, thus returned to normal following improved glycaemic control which was associated with reduced triglyceride content of LDL particle. Altered LDL composition has been reported in NIDDM. LDL levels have been reported to return to normal on treatment.

Reichi *et al.,* (1986) demonstrates that HDL/LCAT complex is able to act as acceptor of cellular free cholesterol, which is then esterified by LCAT and transferred to core of the particle to become HDL_2 the cholesterol ester can then be transferred to other lipoproteins of lower density by a lipid transfer protein and reach the liver for excretion.

Glomset (1968) reveals that LCAT is synthesized in liver and circulates in plasma associated with HDL. In disorders that obstruct flow of bile, concentration of un-esterified cholesterol and lecithin in plasma are increased. Esterification of cholesterol was decreased in many patients with impaired liver function.

MATERIAL AND METHODS

Selection of Subjects

The study is conducted in three groups of subjects selected from out patients as well as in patients from the Department of Medicine at Owaisi Hospital & Research center and Princess Esra Hospital, Hyderabad.

Group I

Consists of (10) normal adult males and females between the age group of 30 – 50 years selected as control group with no history of diabetes mellitus.

Group II

Consists of (15) subjects of similar age group with good glycaemic control of Diabetes Mellitus.

Group III

Consists of (15) subjects of similar age group with poor glycaemic control of diabetes mellitus.

The following parameters were studied on fasting blood sample.

1. Fasting Plasma Glucose
2. Lipid Profile

Estimation of Glucose

Method: Glucose Oxidase – Peroxidase method

Principle: This is enzymatic method employed in the clinical laboratory for the estimation of glucose. Glucose is oxidized by glucose oxidase to gluconic acid and H_2O_2 is liberated. The colorimetric indicator, quinonemine is generated from 4-amino antipyrene and phenol by H_2O_2 under the catalytic action of peroxidase, intensity of color generated is directly proportional to glucose concentration.

Glucose + O_2 + H_2O + ==========➔ Gluconic acid + H_2O_2

H_2O_2 + 4 Aminoantipyrine + Phenol ==========➔ Quinonemine + 4 H_2O

Reagents

Reagent 1:

Phosphate buffer pH 7.0	100mmol/l
Phenol	5mmol/l
4-Aminoantipyrine	0.5mmol/l
Glucose oxidase	$\geq$ 15 KU/l
Peroxidase	$\geq$ 1 Ku/l

Reagent 2: Glucose standard 100mg / dl

Sample Material

Serum, Heparin – Plasma or Fluoride – Plasma

The stability in serum and plasma is 1 day at 2-8 degree centigrade, serum or plasma must be separated from erythrocyte within 60 minutes of collection.

Assay Procedure

Wave length	Hg 546 nm 500-540nm
Light path	1 cm
Temperature	37 degree centigrade
Measurement	Against reagent block

Reference Range

SERUM / PLASMA	70-110 mg/dl

DILUTION LIMIT

If the concentration exceeds 500 mg/dl samples should be diluted 1 + 4 with NaCl solution (9 g/l) and the result multiplied by 5.

Interferences

No interference is observed by bilirubin up to 10 mg/ U. Fructose galactose, reduced glutathione, ascorbic acid and Xylose do not interfere in the measurement.

REAGENT	BLANK	STANDARD	TEST
Standard	-	10 Ul	-
Sample	-	-	10 Ul
Distilled Water	10 Ul	-	-
Reagent 1	1000Ul	1000Ul	1000Ul

Mix, incubate for approximately 15 minutes at 37 degree centigrade and read the absorbance against reagent blank within 30 minutes.

a) Raabo e, terkildsen tc. On the enzymatic determination of blood glucose Scan J Clin Lab Invest.
b) Trinder P. Glucose oxidase-peroxidase method. Ann Clin Biochem. 1964;6:24

Lipid Profile:

Fasting venous samples are collected in sterile clean and dry bottles. Serum was separated and assays are performed immediately.

Estimation of Total Free and Ester Cholesterol

Estimation of Total Cholesterol:

Method: Colorimetric Method using salkowski reaction.

Reference: Zlatkis. A; Zak-B; & Boyle G.J. (1953) J. LAB CLINICAL MEDICINE 41: 486 (Varley Text Book of Clinical Bio-Chemistry 4th Edition)

Equipment: Bausch & Lomb photo colorimeter (Spectronic 20).

Principle: Proteins in serum are precipitated by ferric chloride. The cholesterol present in protein free filtrate is oxidized and dehydrated with ferric chloride, acetic acid and sulfuric acid to a red colored compound. A measure of the intensity of the color indicates the concentration of cholesterol in the serum.

Reagents:

1. Acetic Acid: (Analytical grade, aldehyde free).
2. Ferric chloride – (10%) 10 grams. Of Feb 13, 6 H2O dissolved in 100 ml of acetic acid.
3. Colour Reagent: 2.5 ml of 10% $FeCl_3$ was added to 500ml of aldehyde free acetic acid.
4. Sulfuric acid (Analar grade)
5. Stock cholesterol standard – 100 mg of cholesterol was added to 100ml of purified acetic acid.
6. Cholesterol working standard solution. The stock cholesterol standard was diluted one ml to 25 ml with color reagent.

Procedure:

In a centrifuge tube take 9.9 ml color reagent and add 0.1 ml serum mix well and allowed to stand for 10 – 15 minutes at room temperature and centrifuged.

II. Take 3 test tubes and label as 'B' (blank) T (Test) and S (standard) and pipette the following reagents in the respective tubes.

S.No.	Reagents	B (in ml)	T (in ml)	S (in ml)
01	Supernatant fluid	-	5	-
02	Color Reagent	5	-	-
03	Working cholesterol std.	-	-	5
04	Concentrated Sulfuric acid	3	3	3

Mix well and keep for 30 minutes at room temperature.

Test and standard are read at 560 nm against blank.

Calculations:

Mg of cholesterol / 100 ml of serum

$$= \frac{\text{Reading of unknown}}{\text{Reading of standard}} \times \frac{0.2}{0.05} \times 100$$

$$\frac{\text{Reading of T}}{\text{Reading of S}} \times 400$$

Estimation of Total Triglycerides

Method: Enzymatic colorimetric method using triglyceride kit

Principle: Determination of triglycerides involves enzymatic splitting with lipoprotein lipase. Indicator is quinonemine, which is generated from 4-aminoantipyrine and 4-chlorophenol by hydrogen peroxide under the catalytic action of peroxidase.

Test Concentrations:

Reagent:

Good's buffer pH 7.2	50nmol/l
4-Chlorophenol	4 nmol/l
ATP	2 nmol/l
Mg^{2+}	15 nmol/l
Glycerokinase (GK)	$\geq$ 0.4 kU/l
Peroxidase (POD)	$\geq$ 2.0 kU/l
Lipoprotein lipase (LPL)	$\geq$ 2.0 kU/l
4-Aminoantipyrine	0.5 nmol / l
Glycerol-3-phosphate oxidase (GPO)	$\geq$ 1.5 kU/l

Reagent 2: Triglycerides

Sample Material:

Serum, heparin plasma or EDTA plasma

The stability in serum or plasma is 2 days between 20-25^{o}C, 7 days between 4-8^{o}C and min. 1 year – 20^{o} C.

Assay Procedure:

Wave length 550nm, Hg 546 nm

Light path 1 cm

Temperature 20 – 25^{o} C/37^{o} C

Measurement Against blank

Reference Range {2}

Desirable	: <200 mg/dl (2.3nmol) fasting
Borderline	: 200-400 mg/dl (2.3-4.5nmol/l)
Elevated	: > 400mg/dl (4.5nmol/I)

Clinical interpretation {3}

Epidemiological studies have observed that a combination of plasma triglycerides >180 mg/dl (>2.0nmol/l) and HDL-Cholesterol < 40 mg/dl (1.0nmol/l) predict a high risk of CHD, Borderline levels (> 200 mg/dl) should always be regarded in associated with other risk factors for CHD.

Performance Characteristics

Measuring range

The test has been developed to determine triglyceride concentrations within a measuring range from 1-1000 mg/dl (0.01 – 11.3nmol/l).

When values exceed this range samples should be diluted 1 + 4 with NaCl solution (9g/l) and the result multiplied by 5.

Interferences

No interference is observed by bilirubin up to 40 mg/dl, Ascorbic acid interferes starting with a concentration of 6 mg/dl. Hemoglobin interferes starting with a concentration of 250 mg/dl.

Limit of Detection

The lower limit of detection is 1 mg / dl.

REAGENT	BLANK	STANDARD	TEST
Standard	-	10 Ul	-
Sample	-	-	10 Ul
Distilled Water	10 Ul	-	-
Reagent 1	1000Ul	1000Ul	1000Ul

Mix, incubate 10 minutes at 25 degree centigrade or 5 minutes at 37 degree centigrade read the absorbance against reagent blank within 60 minutes.

A) Recommendation of the second joint task force of European and other societies on Coronary Prevention. Prevention of coronary heart disease in clinical practice. European Heart Journal 1998; 19 1434-503.
B) Handbook of Lipoprotein testing. Nader Rifai, G. Russel Wamick, and Marek H.Dominiczack, eds.Washington D.C.: AACC Press, 1997: P 115-26.

Estimation of HDL Cholesterol

Phosphotungstate Method

Intended Use

This reagent kit is intended form vitro quantitative determination of HDL – Cholesterol (High Density Lipoprotein-Cholesterol) in serum or plasma.

Clinical Significance

HDL – Cholesterol and coronary heart disease are inversely related. Low concentrations of HDL – Cholesterol are associated with higher risk of coronary heart disease. Thus HDL-Cholesterol in combination with total cholesterol determination is a good index of the risk of coronary heart disease. Coronary Heart Disease (CHD) risk factor can be calculated using total lipid profile, as suggested by Castelli *et al.,* The risk factor gives the most accurate and definite assessment of heart disease risk.

The factors are calculated by the ratio of Total Cholesterol to HDL – Cholesterol and by the ratio of LDL – Cholesterol (Low Density Lipoprotein-Cholesterol to HDL – Cholesterol).

Risk	Ratio: Total/HDL-Cholesterol		Ratio : LDL/HDL – Cholesterol	
	Men	Women	Men	Women
½ Avg.	3.43	3.27	1.00	1.47
Avg.	4.97	4.44	3.55	3.22
2 x Avg.	9.55	7.05	6.25	5.03
3 x Avg.	23.99	11.04	7.99	6.14

The value of LDL – Cholesterol can be calculated as follows. If the value of the Triglycerides is known, LDL-Cholesterol can be calculated based on Friedewald's equation.

$$\text{LDL CHOL mg/dl} = \text{Total CHOL} - \frac{\text{Triglycerides}}{5} + \text{HDL CHOL}$$

Principle

Chylomicrons, VLDL (Very Low Density Lipoproteins) and LDL fractions in serum or plasma are separated from HDL by precipitating with Phosphotungstic Acid and Magnesium Chloride. After

centrifugation, the cholesterol in the HDL fraction which remains in the supernatant is assayed with enzymatic cholesterol method, using Cholesterol Esterase, Cholesterol Oxidase Peroxidase and the chromogen 4-Aminoantipyrine/Phenol.

Sample Collfction, Storage & Stability

Serum is preferred. EDTA or heparinised plasma can also be used.

As far as possible, use samples on the same day. Samples are stable for a week when stored tightly capped at $2^oC/8^oC$.

Do not use hemolysed or grossly contaminated samples.

Reagents:

Reagent 1 (Enzymes / Chromogen):	
Cholesterol Esterase	$\geq$ 200 U/l
Cholesterol Oxidase	$\geq$ 200 U/l
Peroxidase	$\geq$ 1000 U/l
4-Aminoantipyrine	0.5nmol / L
Reagent 1A (Buffer):	
Pipes buffer, pH 6.90	50nmol / L
Phenol	24nmol / L
Sodium Cholate	0.5nmol / L
Reagent 2 (Precipitating Reagent):	
Phosphotungstic Acid	2.4nmol / L
Magnesium Chloride	39nmol / L
Standard (HDL Cholesterol 50 mg/dL):	
Cholesterol	0.5 g/L

Precaution:

Good laboratory safety practices should be followed when handling any laboratory reagent.

Storage & Stability of the Reagents

When stored at $2^oC/8^oC$ and protected from light, the reagents are stable until the expiry dates stated on the labels.

Reagent Reconstitution

Allow the reagents to attain room temperature. Dissolve the contents of one bottle of reagent 1 into one bottle of reagent 1A. Mix by gentle swirling till completely dissolved.

Reagent Reconstitution

Allow the reagents to attain room temperature. Dissolve the contents of one bottle of reagent 1 into one bottle of reagent 1A. Mix by gentle swirling till completely dissolved.

Wait for 5 minutes before using.

Reconstituted Reagent Storage & Stability

The reconstituted reagent is stable for 3 months when stored at 2^oC - 8^oC.

Procedure

The samples, the precipitating reagent 2 and the reconstituted reagent should be brought to room temperature prior to use.

1 Precipitation Dispense into Centrifuge Tube:

	Test
Sample	0.20mL (200uL)
Precipitating Reagent 2	0.20mL (200uL)

Mix well.
IV11X WC11.
Centrifuge at 1500 g or 3500-4000 rpm for 10 minutes. Separate the clear supernatant immediately and determine the cholesterol content as given in the Section II.

Note

The 50 mg/dL HDL-Cholesterol standard should not be subjected to the precipitation step.

II Cholesterol Assay

The following general system parameters are to be used with this kit:
Reaction Type: End point
Reaction Slope: Increasing
Wavelength: 500nm (4952- 500nm)
Flowcell Temp.: 30^{o}C
Incubation: 5 minutes 37^{o}C
Sample Vol. (Supernatant): 20uL
Reagent Volume: 1.0mL
Standard Connection: 100 mg/dL (The standard of 50mg/dL is to be fed as 100mg/dL to account for the dilution of sample in the precipitation step)
Zero setting: Reagent Blank
Set the instrument using above system parameters.
Dispense into test tubes:

	Blank	**Standard**	**Test**
Reconstituted Reagent	1mL	1mL	1mL
Standard	-	20uL	-
Supernatant	-	-	20uL

Incubate for 5 minutes at 37^{o}C Mix and read.
Application Sheets for most of the commonly used Chemistry Analyzers are available on request,

Quality Control

To ensure adequate quality control, the use of commercial reference control serum is recommended with each assay batch.
It should be realized that the use of quality control material checks both, the instrument and the reagent functions together.

Reference Values

It is recommended that each laboratory establish its own reference values. The following values may be used a guideline:
HDL – Cholesterol:
Serum – Plasma: 30 – 70 mg/dL

A) Lopes – verelia MF, Stone P, Elnis & Colweh JA (1977). Cholesterol determination in high-density lipoproteins separated by three different methods. *Clinical Chemistry* **23** 882-884.
B) Allain CA, Poon LS, Chan CS, Richmond W and Fu PC (1974). Enzymatic determination of total serum cholesterol. *Clinical Chemistry* **20** 470-5
C) Richmond W (1973). Preparation and properties of a cholesterol oxidase from Nocardia sp and its application to the enzymatic assay of total cholesterol in serum. *Clinical Chemistry* **19**(12) 1350-1356.
D) Miller NE, Thelle DS, Forde OH and Mjos OD (1977). The Tromso heart-study. High-density lipoprotein and coronary heart-disease: a prospective case – control study. *Lancet* **1**(8019) 965-968.

Table 1: Fasting plasma glucose (mg/dl)

S.No.	Group I Normal Control	Group II DM with Good Glycemic Control	Group III DM With Poor Glycemic Control
1.	88	130	194
2.	96	140	194
3.	96	111	186
4.	92	142	182
5.	90	118	188
6.	100	139	166
7.	98	114	169
8.	89	120	192
9.	94	124	192
10.	101	160	170
11.		148	188
12.		148	182
13.		162	230
14.		118	226
15.		126	200
Mean	94.40	133.33	189.00
± SD	4.57	16.45	18.70
±SE	1.44	4.25	4.82

ANOVA:

Table 1.1: Fasting plasma glucose

Source	DF	Sum of Squares	Mean Squares	F	Significance
Between Groups	2	56674.24	28337.12	118.208	P < 0.01
Within Groups	37	8889.73	239.72		
Total	39	85543.97			

Table 2: Total cholesterol (mg%)

S.No.	Group I Normal Control	Group II DM with Good Glycemic Control	Group III DM With Poor Glycemic Control
1.	171	221	264
2.	169	231	328
3.	186	204	280
4.	179	234	280
5.	184	197	280
6.	210	239	256
7.	188	211	276
8.	196	196	336
9.	179	224	264
10.	180	273	256
11.		255	296
12.		260	288
13.		275	320
14.		207	301
15.		242	294
Mean	184.20	231.27	289.00
± SD	12.01	26.11	24.95
±SE	3.80	6.74	6.44

ANOVA:

Table 2.1: Total cholesterol

Source	DF	Sum of Squares	Mean Squares	F	Significance
Between Groups	2	68242.56	34121.28	84.53	P < 0.01
Within Groups	37	19562.53	528.71		
Total	39	87805.10			

Table 3: Serum trigly ceride (mg%)

S.No.	Group I Normal Control	Group II DM with Good Glycemic Control	Group III DM With Poor Glycemic Control
1.	112	216	232
2.	99	196	292
3.	139	224	241
4.	124	292	212
5.	135	248	226
6.	110	180	199
7.	90	172	204
8.	155	246	296
9.	136	248	194
10.	149	176	192
11.		188	249
12.		265	236
13.		224	291
14.		214	288
15.		278	226
Mean	124.90	224.47	238.53
± SD	21.57	37.83	37.27
±SE	6.82	9.76	9.62

ANOVA:

Table 3.1: Serum triglyceride

Source	DF	Sum of Squares	Mean Squares	F	Significance
Between Groups	2	80390.73	40195.38	23.88	P < 0.01
Within Groups	37	62318.36	1884.28		
Total	39	142709.10			

Table 4: HDL – cholesterol (mg%)

S.No.	Group I Normal Control	Group II DM with Good Glycemic Control	Group III DM With Poor Glycemic Control
1.	58	39	44
2.	49	46	36
3.	64	52	30
4.	66	44	30
5.	71	38	28
6.	41	49	32
7.	69	36	26
8.	74	51	30
9.	62	44	24
10.	54	41	30
11.		54	32
12.		37	28
13.		34	24
14.		46	26
15.		50	24
Mean	60.80	44.07	29.60
± SD	10.37	6.35	5.24
±SE	3.28	1.64	1.35

ANOVA:

Table 4.1: HDL – cholesterol

Source	DF	Sum of Squares	Mean Squares	F	Significance
Between Groups	2	5877.64	2938.82	56.63	P < 0.01
Within Groups	37	1920.13	51.89		
Total	39	7797.77			

Table 5: LDL – cholesterol (mg%)

S.No.	Group I Normal Control	Group II DM with Good Glycemic Control	Group III DM With Poor Glycemic Control
1.	91	139	174
2.	100	146	234
3.	94	107	218
4.	88	132	208
5.	86	109	207
6.	147	154	184
7.	101	141	205
8.	91	96	245
9.	90	130	201
10.	96	197	188
11.		163	214
12.		170	213
13.		196	238
14.		92	217
15.		136	225
Mean	98.40	140.53	211.40
± SD	17.75	32.19	19.80
±SE	5.61	8.31	5.11

ANOVA:

Table 5.1: LDL – cholesterol

Source	DF	Sum of Squares	Mean Squares	F	Significance
Between Groups	2	82790.04	41395.02	67.04	P < 0.01
Within Groups	37	22845.73	617.45		
Total	39	105835.77			

Table 6: VLDL – cholesterol (mg%)

S.No.	Group I Normal Control	Group II DM with Good Glycemic Control	Group III DM With Poor Glycemic Control
1.	22	43	46
2.	20	39	58
3.	28	45	48
4.	25	58	42
5.	27	50	45
6.	22	36	40
7.	18	34	41
8.	31	49	59
9.	27	50	39
10.	30	35	38
11.		38	50
12.		53	47
13.		45	58
14.		43	58
15.		56	45
Mean		44.93	47.60
± SD		7.62	7.44
±SE		1.96	1.92

ANOVA:

Table 6.1: VLDL – cholesterol

Source	DF	Sum of Squares	Mean Squares	F	Significance
Between Groups	2	3445.36	1722.68	36.20	P < 0.01
Within Groups	37	1760.53	47.58		
Total	39	5205.90			

RESULTS AND DISCUSSION

Serum cholesterol levels in diabetic patients are elevated. The levels in diabetic patient with poor glycemic control are much more elevated when compared to diabetic patients with good control (Table 1 & 1.1). The levels being in good control (231.27 $\pm$ 26.11) and in poor control (289.00 $\pm$ 24.95). These levels are significantly raised (P<0.01) as compared to the normal control in whom the serum cholesterol levels are 184.20 $\pm$ 12.01 (Table 2 & Table 2.1).

Serum triglyceride levels in diabetic patients are elevated in both the groups with good and poor glycemic control much more in patients with poor glycemic control. The levels being in good glycemic control (224.47 $\pm$ 37.83) and in poor glycemic control (231.86 $\pm$ 13.48) respectively and it is significantly raised (P<0.01) as compared to the normal control values (124.90 $\pm$ 21.57) (Table 3 & Table 3.1).

HDL Cholesterol levels in normal control are 60.80 $\pm$ 10.37. In diabetic patients with good glycemic control it is decreased (44.07 $\pm$ 6.35). It is further decreased in diabetic patients with poor control, the levels being 29.60 $\pm$ 5.24 (P<0.01 (Table 4 & Table 4.1).

LDL Cholesterol level in diabetic patients with good control are (140.53 $\pm$ 32.19) and in poor control are 211.40 $\pm$ 19.80. These are significantly higher than in normal control group 98.40 $\pm$ 17.75 (P < 0.01).

VLDL Cholesterol level in diabetic patients with good control are 44.93 $\pm$ 7.62 and in poor control 47.60 $\pm$ 7.44. (P<0.01)

The type of lipoprotein abnormalities in diabetes depend on many factors, type of diabetes, endogenous insulin reserve, degree of obesity and insulin resistance, type of therapy, degree of glycaemic control and the presence of absence of nephropathy.

Diabetes mellitus type 2 in particular is a progressive macro vascular disease, with universally established excessive predilection for CAD irrespective of race, ethnicity, gender or geography. NIDDM is an independent risk factor for coronary artery disease and risk of coronary disease is three to four fold increased in patients with NIDDM compared with non-diabetic population. These patients often have abnormalities of both quantity and quality of lipoprotein that among other factors might be responsible for the increased incidence of vascular complication.

The most characteristic lipid abnormality in diabetics is hypertriglyceridaemia which is a risk factor for major vascular events in diabetics.

It has been conclusively shown that reducing LDL cholesterol is beneficial in reducing CAD risk, with lowering of LDL cholesterol being a primary target in the prevention of CAD.

Results of the study indicate derangement of lipoprotein metabolism in patients with NIDDM. There was increase in serum triglycerides, cholesterol, LDL and VLDL levels. HDL cholesterol levels are lowered.

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