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A study on lipid Profile of old aged patients of dyslipidemia with and without type 2 Diabetes mellitus: A hospital based observational study

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Abstract

Aims: aim of our study was to evaluate the lipid profile of old aged dyslipidemic patients with and without type 2 diabetes mellitus.

Methods: A detail history, clinical examination and relevant investigations were performed all patients. Plasma glucose estimation was performed by using RFCL kit on the microlab -300 Semi Auto- analyzer supplied by Merck. Total cholesterol was estimated quantitatively by CHOD-PAP technique. Serum Triacylglycerol was estimated quantitatively by GPO-ESPAS technique.

High density lipoproteins (HDL-C) was estimated quantitatively by PEG-PAP method.

Very low density lipoproteins (VLDL-C) was estimated from serum triacylglycerol level using Friedewald formula and low density lipoproteins (LDL-C) was calculated by subtracting serum HDL and VLDL from total serum cholesterol.

Results: Data was analyzed by SPSS software. Mean ± S.D and Person's r value were observed. P value was taken ≤ 0.05 for significant differences.

Conclusions: Old age as well as diabetic state both were affected the lipid profile. All constituent of lipid profile had an increasing trend with age and diabetes except HDL which had a negative trend with advancing age. Hence, diabetes mellitus accelerates age related disturbances in lipid profile.

Keywords: old age, type 2 diabetes mellitus, Plasma glucose, lipid profile

Introduction

Diabetes mellitus type 2 (DM type2) is more common and accounts for 90- 95% of diabetic patient. It is a heterogeneous group of disorder characterized by variable degrees of insulin resistances, impaired insulin secretion [1]. Symptoms of marked hyperglycemia include polyuria, polydypsia, and weight loss. Sometimes polyphagia and blurred vision may also be found. Impairment of growth and susceptibility to certain infections may also accompany chronic hyperglycemia. Acute life threatening consequences of uncontrolled diabetes are hyperglycemia with ketoacidosis or non- ketotic hyperosmolar syndrome [2]. Long-term complications of diabetes mellitus include retinopathy, nephropathy leading to renal failure, peripheral neuropathy and autonomic neuropathy. There is also an increased incidence of atherosclerotic, cardiovascular, peripheral arterial, cerebrovascular disease, hypertension and abnormalities of lipoprotein metabolism.

Diabetes mellitus is a common secondary cause of dyslipidaemia. This dyslipidemic changes are characterised by a high level of plasma triglyceride concentration (particularly post prandial hyperlipidemia), a low level of high density lipoprotein-cholestrol (HDL-c) concentration, increased concentration of small dense LDL particles. These biochemical picture associated with diabetes mellitus has been termed as 'diabetic dyslipidaemia'. Many features of diabetic dyslipidaemia can be explained by reduced action of insulin at the tissue level. This could be due to insulin resistance, although relative insulin deficiency associated with pancreatic beta-cell dysfunction also contributes [3]. Some features of diabetic dyslipidaemia, however, may not be due to insulin resistance. The lipid changes associated with diabetes mellitus are attributed to increased free fatty acid flux secondary to insulin resistance. There is increased free fatty acid release from insulin resistant fat cells. The increased flux of free fatty acid into the liver in presence of adequate glycogen stores promotes Triglyceride (TG) production which in turn stimulates the secretion of apoprotien B (apoB) and VLDL-cholesterol. The impaired ability of insulin to inhibit free fatty acid (FFA) release leads to enhanced hepatic VLDL - cholesterol production [4]. Serum high-density lipoprotein (HDL) cholesterol levels decrease in males during puberty and early adulthood, and thereafter remain lower than in women at all comparable ages. On the other hand, the HDL cholesterol concentrations remain constant in women throughout their lifetime [5]. The triglyeride concentrations increase progressively in men, reaching peak values between 40 and 50 years of age, and decline slightly thereafter. In women, the triglyceride concentrations increase throughout their lifetime, and are always higher in those using estrogens [5]. Essentially before menopause, women usually have total cholesterol levels that are lower than those of men of the same age. As

women and men get older, their blood cholesterol levels rise until about 60–65 years of age. In women, menopause often causes an increase in their LDL cholesterol and a decrease in their HDL cholesterol level, and after the age of 50, women often have higher total cholesterol levels than men of the same age. This may well be one factor as to why the incidence of atherosclerotic disease rises dramatically in postmenopausal women [5]. A mechanism that could explain the age-related changes in lipid metabolism is pseudo-capillarization of the liver sinusoidal endothelial cells dysfunction. This physiologic phenomenon has been described in elderly humans and some animals. It leads to decreased endocytosis, increased leukocytes adhesion, decreased hepatic perfusion, and potentially affects passage of chylomicron remnants to hepatocytes [9]. Total cholesterol level gradually increases with age. After the age of about 55 years, women consistently have higher total LDL and HDL cholesterol level than do men of the same age. The value of total cholesterol as predictor of the relative risk of coronary disease with increasing age in both men and women. In the Framingham study, Cholesterol levels over 275 mg/dl (7.1 mmol/L) were associated with a fourfold increase in risk of recurrent infarction or in coronary death, and almost a threefold increase in risk from all-cause mortality compared with Cholesterol levels less than 200 mg/ dl [7]. Lipoproteins are altered in diabetes. The quantitative changes, most commonly seen are an increase in the TG-rich lipoproteins and a decrease in HDL [6, 8]. These changes can be seen at and even before the diagnosis of diabetes. Serum high-density lipoprotein (HDL) levels decrease in males during puberty and early adulthood, and thereafter remain lower than in women at all comparable ages. On the other hand, the HDL-c concentrations remain constant in women throughout their lifetime [7]. Triglyceride concentration increases progressively in men, reaching peak values between 40 and 50 years of age, and declines slightly thereafter. In women, the triglyceride concentration increases throughout their lifetime, and are always higher in those using estrogens. Aim of this present study was to evaluate the lipid profile of old aged patients of dyslipidemia with and without type 2 diabetes mellitus.

Materials and Methods

This study was conducted in department of Biochemistry in Katihar Medical College and Hospital in collaboration with the Department of Medicine during a period from February 2012 to November 2012. The patients were in uniformity in socioeconomic status, culture and food habits. Entire subjects signed an inform consent approved by institutional ethical committee of Katihar Medical College, Katihar, Bihar, India was sought.

Study Subjects: A total of 72 cases of dyslipidemia were enrolled in this study. All patients were devided into two groups (case group I and case group II). Case group I (DDO) were 36 old aged (50-60) diabetes patients with dyslipidemia. And case group II (NDDO) were 36 old aged (>60 years) dyslipidemia without diabetes. Inclusion criteria of this study was on phase of medication, tobacco chewer, and chain smoker (consuming ≥5 cigarettes per day and were smoking continuously for a minimum of six months prior to being enrolled). Exclusion criteria: Subjects were suffered from any other hormonal disorders, benign or malignant disorders, diabetic ketoacidosis, febrile conditions, renal failure and other renal diseases, gastroenterological conditions, liver diseases, transplant rejection, diseases of the central nervous system and pregnant ladies were excluded from this study.

Study Design: Randomly selected diabetic patients were subjected to evaluation for lipid profile, clinically and biochemically. The diagnosis of diabetes mellitus was based on World Health Organization (WHO) criteria i.e. Fasting plasma glucose of 126 mg/dl (7.0 mmole/L) or more, after a minimum of 12-hour fasting, with symptoms of diabetes, And 2 hours - post prandial plasma glucose level of equal or more than 200 mg/dl (11.1mmole/L). All the biochemical estimations were done by using RFCL kit on the microlab - 300 Semi Auto- analyzer supplied by Merck. Fasting and postprandial serum glucose were estimated quantitatively by GOD/POD technique as described by Trinder (1969). Total cholesterol was estimated quantitatively by CHOD-PAP technique as described by Allian C.C (1974). Serum Triacylglycerol was estimated quantitatively by GPO-ESPAS technique as described by Buccolo G and David M (1973). High density lipoproteins (HDL-C) was estimated quantitatively by PEG-PAP method. Very low density lipoproteins (VLDL-C) was estimated from serum triacylglycerol level using Friedewald formula. Low density lipoproteins (LDL-C) was calculated by subtracting serum HDL and VLDL from total serum cholesterol [9, 10].

Estimation of plasma Glucose [11].

Method: Glucose oxidase peroxidise (GOD-POD) end point colorimetry (RFCLkit)

Principle: Glucose is oxidised to gluconic acid and hydrogen peroxide in the presence of glucose oxidase. Hydrogen peroxide further reacts with phenol and 4- amino antipyrine by the catalytic action of peroxidise to form a red colored quinonimine dye complex. Intensity of the colour is directly proportional to the amount of glucose present in sample.

Estimation of lipid profile: Estimation of serum cholesterol by CHOD/PAP method.

Principle [12, 13]. Cholesterol esters are hydrolysed by cholesterol esterase to give free cholesterol and fatty acids. Then cholesterol oxidase (CHOD) oxidises 3-OH group of free cholesterol to form cholest-4-en-3-one to liberate hydrogen peroxide. Next, peroxidase reduces the hydrogen peroxide to water and releases nascent oxygen (O). This (O) oxidises the chromogenic substance, 4-aminoantipyrine in presence of phenol to purple colored Quinoleimine dye.

Absorbance of colored dye is measured at 505 nanometer, which is proportional to the amount of total cholesterol concentration in the sample.

For HDL Cholesterol: LDL cholesterol, VLDL and Chylomicron frations are precipitated by addition of Polyethylene Glycol 6000 (PEG). After centrifugation, HDL fraction remains in the supernatant and is determined with CHOD- PAP method.

For Total Cholesterol: Thousand micro litres of CHOD-PAP reagent was taken in three test tubes marked as blank, standard and test. Then 10µl of distilled water was added in test tube marked as Blank, 10 µl of standard (200 mg/dl) in test tube marked as Standard and 10 µl serum in test tube

marked as Test (unknown)
The solutions were mixed properly and incubated at 37°C for 10 minutes. Then the concentration of cholesterol in serum was measured in photoelectric colorimeter at 505 nm against reagent blank.
For HDL Cholesterol: Two hundred micro litres of precipitating reagent was taken in a clean centrifuged tube. The same amount of serum was added to it than the solutions were mixed properly and kept at 15-30 minutes. Then the solution was centrifuged for 15 minutes at 2000 rpm. Then 1000 µl of CHOD-PAP reagent was taken in three test tubes marked as Blank, Standard and Unknown. Then 100 µl of distilled water as blank, 100µl of standard 100 µl of clear supernatant in test tube marked as unknown was added. The solutions were mixed properly and incubated at 37 °C for 10 minutes. Then the concentration of HDL cholesterol in serum was measured in photoelectric colorimeter at 505 nm against reagent blank.

Calculations for total cholesterol

$$\text{Cholesterol concentration (mg/dl)} = \frac{\text{Absorbance of unknown X conc. of standard(200mg/dl)}}{\text{Absorbance of standard}}$$

Calculations for HDL cholesterol

$$\text{HDL cholesterol concentration (mg/dl)} = \frac{\text{Absorbance of unknown X conc. Of Standard}}{\text{Absorbance of Standard}} \quad 50X2^*$$

(The standard concentration is multiplied by 2 as diluents factor as sample was diluted 1:1)

Calculations for LDL Cholesterol [9, 10]

LDL-c = Total serum cholesterol- (VLDL-c +HDL-c)
VLDL was calculated from serum TG by using Friedewald `s equation.
Estimation of plasma triacylglycerol (TG) level by end point method [9]: Glycerol -3 Phosphate oxidase and peroxidise method (GPOGAP)

Principle

They are hydrolysed by lipoprotein Lipase (LPL) to produce glycerol and free fatty acid (FFA). In presence of glycerol Kinase (GK), Adenosine Triphosphate (ATP) phosphorylates glycerol to produce glycerol -3 phosphate and Adenosine Diphosphate. Glycerol -3 phosphate is further oxidised by glycerol -3 phosphate oxidase (GPO) to produce dihydroxy acetone (DAP) and hydrogen peroxide. In presence of peroxidase (POD), H_2O_2 couples with 4-AAP and 4-chlorophenol to produce red Quinoleimine dye. Absorbance of colored dye is measured at 505 nm, which is proportional to triacylglycerol concentration in the sample.

Procedures

Thousands microlitre triglyceride Mono- Reagent was taken in Three test tubes marked as blank, standard and unknown. Then 10 µl distilled water in test tube marked as Blank, 10 µl standard (200 mg/dl) in test tube marked as standard, 10 µl in test tube marked as unknown were added. Then the solutions were mixed properly and were incubated at 37 °C for 10 minutes. Then the concentration of cholesterol in serum was measured in photoelectric colorimeter at 505 nm against the reagent blank.

Calculation

$$\text{Triglyceride}_{\text{conc}} \text{ (mg/dl)} = \frac{\text{Absorbance of unknown X conc. of standard (200mg/dl)}}{\text{Absorbance of standard}}$$

Statistical Analysis

Data was analyzed by using SPSS software. Mean ± S.D and Person's r value were observed. P value was taken equal to or less than 0.05 for significant differences.

Observations

A total 72 subjects were included in this study. Out of 72 cases, 36 old age (50-60 years) patients of type 2 diabetes mellitus with dyslipidemia were enrolled in case group I and 36 old age (>60 years) patients of non diabetic with dyslipidemia were enrolled in case group II.

Table 1: Demographic details of case I and case II group patients.

Groups		Numbers (n)	Mean Age(Yrs) ± SD	Sex ratio (M/F)
Case I	DDO	36	56.77 ± 0.392	3.50
Case II	NDDO	36	66.75 ± 0.637	1.00

As shown in table 1, mean age of overall case group was 56.51 ± 0.865 yrs while sex ratio was 1.57. Mean age of DDM, DDO and NDDO groups were 46.02 ± 0.422 yrs., 56.77 ± 0.392 yrs. and 66.75 ± 0.637yrs respectively. While the sex ratio in these three groups were 1.25, 3.50 and 1.00 respectively. Mean age of control group was 35.19 ± o.437 yrs. and sex ratio was 2.60.

Table 2: Blood sugar levels and lipid profiles in old aged Diabetic and Dyslipidemic Patients (DDO) (n=36).

	Mean (mg/dl)	SD	SEM	R value
FBS	178.4167	31.16076	5.19346	-0.736
PPBS	245.1389	43.85962	7.30994	-0.728
TC	314.7222	36.82903	6.13817	0.360
HDL	28.6944	2.05384	.34231	-0.129
LDL	214.6611	36.45781	6.07630	0.175
VLDL	78.8778	3.05054	.50842	0.681
TG	358.6389	37.63065	6.27178	0.216

As shown in table 2, FBS and PPBS levels were 178.4167 ± 31.16076 mg/dl and 245.1389 ± 43.85962 mg /dl respectively in DDO group. Mean TC, HDL, LDL, VLDL and triglyceride level were 314.7222 ± 36.82903 mg/dl, 28.6944 ± 2.05384 mg/dl, 214.6611 ± 36.45781mg/dl, 78.8778 ± 3.05054mg/dl and 358.6389 ± 37.63065 mg/dl respectively. There is a positive correlation between age and TC, LDL, VLDL and triglyceride with r values 0.360, 0.175, 0.681 and 0.216 respectively. HDL was negatively correlated with age with r value –0.129

Table 3: Blood sugar levels and lipid profiles in DDO group with history of DM < 5 Yrs. (n=18)

	Mean (mg/dl)	SD	SEM
FBS	166.4737	27.72357	6.36022
PPBS	219.0526	28.68502	6.58080
TC	306.8947	35.98131	8.25468
HDL	29.4211	2.00875	0.46084
LDL	210.7789	36.81285	8.44545
VLDL	78.2316	4.14260	0.95038
TG	336.8947	32.38980	7.43073

Table 4: Blood sugar levels and lipid profiles in DDO group with history of DM > 5 Yrs. (n=18)

	Mean (mg/dl)	SD	SEM
FBS	191.7647	30.01152	7.27886
PPBS	274.2941	39.63705	9.61340
TC	323.4706	36.83089	8.93280
HDL	27.8824	1.83311	0.44459
LDL	219.0000	36.67533	8.89507
VLDL	79.6000	.00000	0.00000
TG	382.9412	26.98951	6.54592

In table 3 and 4 we compared lipid profiles of DDO group with history of diabetes < 5 yrs and > 5 yrs. Mean total cholesterol LDL, VLDL and TG were significantly higher in DDO with history of DM > 5 yrs. HDL level was significantly lower in later group.

Table 5: Blood sugar levels and lipid profiles in non Diabetic Dyslipidemic (NDDO) Patients (n=36)

	Mean (mg/dl)	SD	SEM	R values
FBS	90.8889	12.39073	2.06512	0.408
PPBS	130.8056	12.89921	2.14987	0.534
TC	330.2222	40.54987	6.75831	0.364
HDL	31.3333	2.48424	.41404	-0.156
LDL	224.5833	39.52407	6.58734	0.451
VLDL	70.9389	6.21883	1.03647	0.275
TG	359.8333	28.34935	4.72489	0.206

As shown in table 5, mean FBS and PPBS levels were 90.8889± 12.39073 mg/dl and 130.8056± 12.89921 mg /dl respectively in NDDO group. Mean TC, HDL, LDL, VLDL and triglyceride level were 330.2222± 40.54987 mg/dl, 31.3333± 2.48424 mg/dl, 224.5833 ± 39.52407 mg/dl, 70.9389 ± 6.21883 mg/dl and 359.8333 ± 28.34935 mg/dl respectively. There is a positive correlation between age and TC, LDL, VLDL and triglyceride with r values 0.364, 0.451, 0.275 and 0.206. Respectively. HDL was negatively correlated with age with r value -0.156

Table 6: Comparison of DDO group with NDDO

		Mean (mg/dl)	SD	SEM	P Values
FBS	DDO	178.4167	31.16076	5.19346	0.00
	NDDO	90.8889	12.39073	2.06512	
PPBS	DDO	245.1389	43.85962	7.30994	0.00
	NDDO	130.8056	12.89921	2.14987	
TC	DDO	314.7222	36.82903	6.13817	0.09
	NDDO	330.2222	40.54987	6.75831	
HDL	DDO	28.6944	2.05384	.34231	0.00
	NDDO	31.3333	2.48424	.41404	
LDL	DDO	214.6611	36.45781	6.07630	0.27
	NDDO	224.5833	39.52407	6.58734	
VLDL	DDO	78.8778	3.05054	.50842	0.00
	NDDO	70.9389	6.21883	1.03647	
TG	DDO	358.6389	37.63065	6.27178	0.87
	NDDO	359.8333	28.34935	4.72489	

As shown in table 6, FBS and PPBS were significant higher in DDO group as compared to NDDO group. Mean TC, HDL, LDL, VLDL and triglyceride level were 330.2222 ± 40.54987 mg/dl, 31.3333 ± 2.48424 mg/dl, 224.5833 ± 39.52407 mg/dl, 70.9389 ± 6.21883 mg/dl and 359.8333 ± 28.34935 mg/dl respectively in NDDO group. While in DDO group mean TC, HDL, LDL, VLDL and triglyceride level were 314.7222± 36.82903mg/dl, 28.6944± 2.05384 mg/dl, 214.6611 ± 36.45781mg/dl, 78.8778± 3.050 54mg/dl and 358.6389± 37.63065 mg/dl respectively. In NDDO group TC, LDL, and Triglyceride levels were higher but not statistically significant while VLDL levels were significantly lower

Discussion

Diabetes mellitus (DM) is a group of metabolic disorders characterized by hyperglycemia resulting from defect in insulin secretion, insulin action or both [14]. There are various genetic as well as environmental factors that can influence the occurrence of this disease [15, 16]. DM is characterized by either the absence of insulin that is insulin dependent DM (Type 1 DM [T1DM]) and which is of insensitivity to the insulin i.e., non-insulin dependent DM (Type 2 DM [T2DM]). It is a complex disease where the carbohydrate and fat metabolism is impaired [17].

Type 2 Diabetes Mellitus. Insulin has im-portant effects on key steps in the metabolism of lipids and lipopro-teins, which are altered in diabe-tes, possibly leading to dyslipidemia [18].

This present study was carried out on total of 72 cases. Out of 72 cases, The patients were divided into two groups, one group case I comprising 36 patients in old age group (50-60 years) with diabetes and Dyslipidemia (DDO) and second group (n=36) case II comprised of old age non –diabetic but dyslipidemic patients (NDDO).

Table 1 shown demographic details of all cases. Mean age of DDO and NDDO groups were 56.77 ± 0.392 yrs. and 66.75 ± 0.637 years respectively. While sex ratio in these two groups were 3.50 and 1.00 respectively. Higher incidence of diabetes mellitus in males is consistent with the findings of many

authors who stat that there is increased prevalence of diabetes in males especially in older age groups [19]. As shown in table 2, in DDO group lipid profile was associated with age. There is a positive correlation between age and TC, LDL, VLDL and triglyceride with r values 0.360, 0.175, 0.681 and 0. 216 respectively. HDL was negatively correlated with age with r value –0.129. These findings can again be explained by the studies of Miller *et al*. [20]. And Framingham *et al.* [21]

Our findings were also supported by the study of Walter *et al*. [22] who were found that the blood- lipid profile worsened with increasing age. Specifically, triglycerides, total cholesterol, and LDL cholesterol increased within each age-group (31, 16, and 15%, respectively). No consistent effect of age was noted on HDL cholesterol and the total cholesterol–to–HDL cholesterol ratio. In table 3 and 4 we compared lipid profiles of DDO group with history of diabetes < 5 yrs and > 5 yrs. Mean total cholesterol LDL, VLDL and TG were significantly higher in DDO with history of DM > 5 yrs. HDL level was significantly lower in later group.

Similar studies have been reported by Harno *et al.,* [23] in which they reported a reduction in HDL cholesterol in Type 2 diabetic patients due to the increased activity of hepatic lipase, which plays an important role in HDL metabolism. However, controversial studies also have been reported where no significant change in HDL cholesterol levels in diabetic patients were reported [24].

As shown in table 5, in NDDO group mean FBS and PPBS levels were well within normal range, they were 90.88 ± 12.39 mg/dl and 130.80 ± 12.89 mg /dl respectively. As in DDO group mean TC, LDL, VLDL and triglyceride level in NDDO were correlated with age. There was a positive correlation between age and TC, LDL, VLDL and triglyceride with r values 0.364, 0.451, 0.275 and0 0.206 respectively. HDL was negatively correlated with age with r value -.156. When we compared DDO with NDDO group to find out the effects of DM over lipid profiles (table 6). FBS and PPBS were significant higher in DDO group as compared to NDDO group. Mean TC, HDL, LDL, VLDL and triglyceride level were 330.22 ± 40.54 mg/dl, 31.33 ± 2.48 mg/dl, 224.58 ± 39.52 mg/dl, 70.93 ± 6.21 mg/dl and 359.83 ± 28.34 mg/dl respectively in NDDO group. While in DDO group mean TC, HDL, LDL, VLDL and triglyceride level were 314.7222 ± 36.82903mg/dl, 28.6944± 2.05384 mg/dl, 214.6611 ± 36.45781mg/dl, 78.8778± 3.05054mg/dl and 358.6389± 37.63065 mg/dl respectively. In NDDO group TC, LDL, and Triglyceride levels were higher but not statistically significant while VLDL levels were significantly lower as compared to DDO. Age and diabetic state both are known to affect the lipid profiles. Above picture of lipid profile can be explained by presence of one factor in each group. Effects of old age (>60 yrs) on lipid profile (except VLDL) in NDDO might have been antagonized by the effects of DM on lipid profiles. In nutshell above result analysis established a significant correlation of lipid profile with aging in diabetic and nondiabetic individuals. Effects of age on lipid profile derangement were greater than diabetic state.

Summary

1. Total cholesterol, LDL, VLDL and triglyceride levels were increased in old aged group (DDO) of diabetic patients as compared to non-diabetes dyslipidemic NDDO patients group.
2. HDL level was decreased in diabetic with dyslipideic groups (DDO) as compared to non-diabetic with dyslipidemic (NDDO) patients group.
3. Age as well as diabetic state both were affected factors of lipid profile.
4. In diabetic with dyslipidemic patient (DDO) lipid profile was more severely affected as compared with non-diabetic with dyslipidemic patients group.
5. Age had stronger effects on lipid profile than the effects of diabetic state which was evident during comparison of diabetic patients with older (more than 60 years) non diabetic subjects.
6. Duration of diabetes mellitus had also a deleterious effect on lipid profile which was evident from the comparison of subjects having history of DM more than 5 years and less than 5 years.

Conclusion

This study was concluded that age as well as diabetic state both were affected the lipid profile. All constituent of lipid profile had an increasing trend with age and diabetes except HDL which had a negative trend with advancing age. Hence we were concluded that diabetes mellitus accelerates age related disturbance in lipid profile.

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