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Research Article

Types of Dyslipidemia in Type 2 Diabetic Patients of Haryana Region

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Abstract: Dyslipidemia is elevation of serum total cholesterol (TC), triglycerides (TGs), or both, or low high-density lipoprotein cholesterol (HDL-C) level that contributes to the development of atherosclerosis, which is a hallmark of diabetes. This research determined the influence of type 2 diabetes mellitus (Type 2 DM) on lipid profile of diabetic patients reporting in a tertiary hospital in Haryana. 100 confirmed Type 2 DM patients and 100 non-diabetic control patients were recruited for the study. Fasting blood samples were collected from both study and control patients and analyzed for TC, TG, HDL-C, LDL-C, VLDL-C, plasma glucose and HbA1c. Fifty-three (53%) of diabetic patients were males whilst 47 (47%) were females. The mean plasma glucose levels, HbA1c, TC and TG were significantly raised in the diabetics as compared to those in the controls. TC, HDL-C, LDL-C were significantly different in diabetic males as compared to diabetic females, while this was not so in controls. High TG (56%) was most commonly present in diabetics. HDL-C alone (27%) was the most common dyslipidemia in diabetics followed by combination of TG and HDL-C (20%). There was a significant association of abnormal lipid parameters in type 2 diabetic subjects, TG correlated positively with plasma glucose and HbA1c, while TC, LDL-C correlated only with postprandial plasma glucose only. Thus type 2 diabetes abnormally influences lipids of patients thus denoting the importance of lipid control on diabetes and exposing them to potential associated cardiovascular diseases.
Keywords: Type-2 Diabetes Mellitus, Dyslipidemia, Lipid Profile, Triglyceride, HDL-C, LDL-C, Total Cholesterol.

INTRODUCTION

Diabetes mellitus (DM) is a hereditary, chronic and endocrine metabolic disorder [1]. Certain ethnic and racial groups of Africa and Asia have a greater risk of developing diabetes [2]. India, a developing Asian country with fast industrialization and a modern lifestyle is facing a grave problem in having the largest number of people with diabetes [3, 4] which is estimated to reach 80 million by the year 2030 [5, 6]. It is close to becoming the diabetic capital of the world. The literature on Indian studies showed a threefold rise in the diabetic prevalence in rural as well as urban areas [7, 8]. Haryana state is no exception to the above said rise and it harbours a substantial number of people with diabetes. The prevalence of type 2 DM increases with age such that in developing countries, and most diabetics are in the age bracket of 45 to 64 years [9].

Type 2 DM is caused by relatively impaired insulin secretion and peripheral insulin resistance [10,11]. Lack of insulin or relatively low insulin levels affects the metabolism of carbohydrate, protein, fat, water and electrolyte balance resulting in diabetes [12]. The most common symptom of diabetes is no symptom and by the time the disorder is diagnosed, an abnormal lipid profile, hypertension and retinal changes may be already present often [13].

Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high-density lipoprotein–Cholesterol (HDL-C) level that contributes to the development of atherosclerosis, which may be primary (genetic) or secondary and diagnosed by measuring plasma levels of total cholesterol (TC), TGs, and individual lipoproteins. It is traditionally classified by patterns of elevation in lipids and lipoproteins [14]. Dyslipidemia is a well-recognized and modifiable risk factor that should be identified early to institute aggressive cardiovascular preventive management [15]. Patients with type 2 DM are at greater risk of developing vascular diseases because of lipid changes. Lipid abnormalities and insulin use is critically discussed in diabetics [16]. The most typical lipoprotein pattern reported in diabetes, also known as diabetic dyslipidemia or atherogenic dyslipidemia consists of moderate elevation in TG levels, low HDL-C

cholesterol values, and low density lipoproteins cholesterol (LDL-C) (especially small dense LDL particles) [17]. These variations in lipid profile in DM should not be generalised to all region and should be individualised to specific regions as ethnic, hereditary and environmental factors influence lipid profile. Due to increasing cardiovascular problems in type 2 diabetic patients [16], this study was conducted to observe the co-relation of type 2 DM and type of lipid abnormalities in type 2 DM.

It has been well observed that controlling diabetes and lipid levels provide great benefit to diabetic patients. In spite the presence of currently available medicines it is still impossible to have better control of the increased risk of vascular diseases in type 2 DM. Insulin resistance syndrome also has been widely discussed and found that if it is associated with type 2 DM in which HDL-C is quite reduced [18]. New research suggests that in subset of patients with diabetes, aggressively treating lipid levels in general; without knowledge of the lipid abnormality present, or likely to be present in type 2 DM, just with the aim of preventing cardiovascular diseases and mortality does no better in this regard and may even be harmful so such approaches need to be revised [16]. The remedy is to have a greater knowledge of lipid changes in type 2 DM.

Thus this research aims to determine the type of dyslipidemia of affected subjects with type 2 DM in Haryana region.

MATERIALS AND METHODS

Study Area and Design

The study was carried out at a tertiary government medical hospital in Haryana with a very active bi-weekly diabetic clinic. 100 type 2 diabetic patients were recruited after their consent had been sought.

Patients Selection Criteria

The study targeted type 2 diabetic patients, medically diagnosed by American Diabetes Association (ADA) criteria, above 18 years of age on diabetic treatment, scheduled to visit the hospital at regular intervals for routine medical review. Randomly selected age and sex matched individuals, with no history of diabetes or any type of illness and not on statins were used as controls.

Patients Exclusion Criteria

Subjects with type 1 DM, other ailments, metabolic disorders and other causes of hyperlipidemia were not included in this study. Pregnant women, patients on statins for abnormal lipid treatment (both for type 2 DM and controls) were also excluded.

Table 1: ATP III Classification of LDL-C, TC, HDL-C and TG (mg/dL) [25]

LDL- Cholesterol	
<100	Optimal
100-129	Near optimal/above optimal
130-159	Borderline high
160-189	High
>190	Very high
Total Cholesterol	
<200	Desirable
200-239	Borderline high
>240	High
HDL- Cholesterol	
<40	Low
>60	High
Triglyceride	
<150	Normal
150-199	Borderline high
200-499	High
≥500	Very high

Samples and Investigations

Venous blood samples were taken from both fasting diabetic and control patients. Investigations carried out were, blood glucose fasting and 2 hrs postprandial blood glucose, glycated haemoglobin (HbA1C) and fasting lipid profile including TC, TG, HDL-C, LDL- C and very low density lipoprotein cholesterol (VLDL-C).

Serum TC was determined by an enzymatic (CHOD-PAP) colorimetric method [19] and TG were determined by an enzymatic (GPO-PAP) method [20].

HDL-C was estimated by a precipitant method [21] and LDL-C by was estimated by using Friedewald's formula [22] as has been shown below:

LDL-C = TC - HDL-C – (TG/5), where TG/5 is approximately equal to VLDL-C.

Plasma glucose was determined by using the glucose oxidase enzymatic method [23]. HbA1c was done by immunoinhibition method [24]. Dyslipidemia (abnormal lipid profile) was defined using the National Cholesterol Education Programme – Adult Treatment Panel III (NCEP – ATP III) (National Cholesterol Education Programme, 2002) criteria as shown in table 1 [25]. Normal values of fasting lipid profile were taken as TC desirable <200mg/dl, TG <150 mg/dl, HDL-C > 40 mg/dl, LDL-C near optimal <130 mg/dl.

Data Analysis

Data analysis was performed using SPSS 21.0 software. The values of all the parameters were given in mg/dl and they were expressed as mean ± SE (standard error). Descriptive analysis was done whilst the statistical significance of the difference between the control and the study groups were evaluated by the student's t-test. Pearson's correlation test was performed to examine various correlations. Pearson's Chi-square was used to test association between diabetes and abnormal lipid parameters. P≤0.05 was considered as significant while P≥0.05 as not significant.

RESULTS

One hundred type 2 diabetic subjects and 100 control subjects were enrolled in the study. The mean age of type 2 diabetic cohort is 55.82±1.29 yrs, with range of 32-88 years. In controls mean age was 55.46±1.30 yrs, ranged 30-85 years. Of the diabetic patients 100 (53%) were males and 47 (47%) were females with equal number of male and females in controls. Table 2 shows age distribution of type 2 DM and control patients. Among the patients mean duration of diabetes was 5.9 years and average duration ranged from newly diagnosed to 30 years.

The mean plasma glucose levels and HbA1c were significantly raised in the diabetics as compared to those in the controls as shown in table 3. The mean serum TG in our study was 186.97 ± 9.28 mg/dL as compared to 102.54 ± 2.77 mg/dL of controls. The mean serum TC was 178.35 ± 5.27 mg/dL as compared to 162.90 ± 3.06 mg/dL of controls. VLDL-C was 38.18 ± 2.04 mg/dL which was significantly higher to 20.76 ± 0.55mg/dL of controls. LDL-C was 102.78 ± 3.94 in comparison to 98.75 ± 2.78 mg/dL of controls, while HDL-C was 41.34 ± 3.10 mg/dL which was non-significantly lower to that of controls as shown in table 4. Serum TC, LDL-C were significantly higher in diabetic females as compared to diabetic males, while serum HDL-C was significantly lower, but this was not seen in controls in whom parameters of lipid profile were comparable in both gender as shown in table 5. Further analysis of results showed that raised TC was seen in 34% (n=34) patients. TG was increased in 56% (n=56) of patients, HDL-C was decreased in 52% (n=52) patients, while LDL-C was raised in 23% (n=23) patients (table 7). The control group had higher frequency of low HDL-C as in the diabetic group.

The highest abnormal lipid analyte is HDL-C in both males 17% and females 10%. Abnormal combined TC, TG and LDL-C was equally present in females in diabetic patients (10%) as HDL-C alone (Table 8). In the correlation studies (table 9) serum TG, serum TC and LDL-C correlated positively with the post prandial plasma glucose while only serum TG correlated positively with the fasting plasma glucose and HbA1c.

Table 2: Age Distribution of Diabetics and Control Patients Diabetics Control

Age range (yrs)	Pateints	Male	Female	Controls	Male	Female
30	0	0		2	2	0
31-40	14	6	8	16	7	9
41-50	26	13	13	21	11	10
51- 60	25	16	9	26	16	10
61-70	25	11	14	23	9	14
71-80	7	5	2	8	5	3
81-90	3	2	1	4	3	1
Total	100	53	47	100	53	47
Mean age	55.82±1.29			55.46±1.30		
Range	32 – 88			30 – 85		

Table 3: Fasting, post prandial plasma glucose, HbA1c of type 2 diabetic patients and controls (Mean ± S.E.)

Parameters	Patients	Controls	p value	Significance
Fasting plasma glucose (mg/dL)	150.42 ± 5.68	84.89 ± 1.66	<.001	HS
Post Prandial plasma glucose (mg/dL)	246.48 ± 8.30	147.60 ± 2.08	<.001	HS
HbA1c (%)	8.99 ± 0.23	4.94 ± 0.05	<.001	HS

Table 4: Lipid profile in type 2 diabetic patients and controls (Mean ± S.E)

Lipid Profile Parameters (mg/dL)	Patients	Controls	p value	Significance
Serum TG	186.97 ± 9.28	102.54 ± 2.77	<.001	HS
Serum TC	178.35 ± 5.27	162.90 ± 3.06	.012	HS
Serum HDL-C	41.34 ± 3.10	39.66 ± 1.07	.609	NS
Serum LDL-C	102.78 ± 3.94	102.40 ± 2.79	.937.	NS
Serum VLDL-C	38.18 ± 2.04	20.76 ± 0.55	<.001	HS

TC= Total Cholesterol; HDL-C= High Density Lipoprotein- Chiolesterol; LDL-C= Low Density Lipoprotein-Cholesterol; TG=Triglyceride.

Table 5: Lipid profile in type 2 diabetic patients and controls according to gender

Lipid profile parameter (mg/dL)	Type 2 diabetes patients (mean ± S.E.)			Controls (mean ± S.E.)		
	Males (n=53)	Females (n=47)	p (Mann-Whitney)	Males (n=53)	Females (n=47)	p (Mann-Whitney)
Serum TG	184.91±12.89	189.30±13.50	.793	97.89+3.58	107.79+4.20	.087
Serum TC	165.23±6.36	193.15±8.16	.005*	159.30+4.09	166.96+4.56	.236
Serum HDL-C	36.09±1.08	40.89±1.34	.004*	38.28+1.41	41.21+1.62	.145
Serum LDL-C	92.19±4.69	114.72±6.08	.002*	100.89+3.72	104.11+4.23	.738
Serum VLDL-C	38.53±3.06	37.79±2.68	.904	19.83+5.17	5.68+0.83	.073

TC= Total Cholesterol; HDL-C= High Density Lipoprotein- Cholesterol; LDL-C= Low Density Lipoprotein-Cholesterol; TG=Triglyceride. * = Significant (P<0.05)

Table 6: Frequency of measured abnormal lipid analyte in diabetic patients

Lipid Analyte measured parameter	Male frequency (%)	Female frequency (%)	Combined frequency (%)
TC+TG+HDL-C+LDL-C	0	3	3
TC+TG+HDL-C	2	0	2
TC+TG+LDL-C	6	10	16
TC+TG	2	5	7
TC+LDL-C	1	3	4
TG+HDL-C	15	5	20
HDL-C	17	10	27
TC	0	2	2
TG	4	4	8
TOTAL	47	42	89
No Abnormality	11		

TC= Total Cholesterol; HDL-C= High Density Lipoprotein- Cholesterol; LDL-C= Low Density Lipoprotein-Cholesterol; TG=Triglyceride

Table 7: Frequency occurrence and correlation of abnormal values of lipid parameters in diabetic and control patients

Parameter Abnormal Value	Frequency in Diabetics (%)	Frequency in control (%)	p
High TC	34	10	<.001
High TG	56	7	<.001
Low HDL-C	52	52	NS
High LDL-C	22	18	.381 (NS)

TC= Total Cholesterol; HDL-C= High Density Lipoprotein- Cholesterol; LDL-C= Low Density Lipoprotein-Cholesterol; TG=Triglyceride. Significant (P<0.05)

Table 8: Frequency of the lipid profile parameters in the type 2 diabetic and control groups according to the ATP III classification

Parameter	Diabetics (%)	Controls (%)
Total cholesterol (mg/dl)		
Desirable (<200)	66	90
Borderline high (200-239)	21	10
High (≥240)	13	0
Triglycerides (mg/dl)		
Normal (<150)	44	93
Borderline high (150-199)	16	7
High (200-499)	40	0
Very High (≥500)	0	0
HDL-C (mg/dl)		
Low (<40)	52	52
Borderline high (40-59)	48	46
High (≥60)	0	2
LDL-C (mg/dl)		
Optimal (<100)	50	48
Near optimal (100-129)	27	34
Borderline high (130-159)	13	18
High (160-189)	8	0
Very high (≥190)	1	0

HDL-C= High Density Lipoprotein- Cholesterol; LDL-C= Low Density Lipoprotein- Cholesterol.

Table 9: Pearson's correlation of lipid profile with diabetic status

Lipid profile	Fasting plasma glucose		Postprandial plasma glucose		HbA1c	
	r	p	r	P	r	p
Serum TG	.336**	<.001	.319**	<.001	.399**	<.001
Serum TC	.120	.090	.226**	.001	.111	.118
Serum HDL-C	.018	.798	.100	.161	.090	.206
Serum LDL-C	-.004	.952	.139*	.049	-.020	.784
Serum VLDL-C	.284**	<.001	.295**	<.001	.392**	<.001

TC= Total Cholesterol; HDL-C= High Density Lipoprotein- Cholesterol; LDL-C= Low Density Lipoprotein-Cholesterol; VLDL-C= very low density lipoprotein; TG=Triglyceride. Significant (*=p<0.05, **= p<.001)

DISCUSSION

Two hundred subjects were recruited for the study made up of 100 diabetics and 100 controls. Of the diabetic patients there was a no significant difference between the number of male (53) and female (47) subjects recruited. Majority (76%) of the diabetic patients were aged 41-70. The age of diabetic patients observed to be ≥40yrs confirmed earlier works that proves that age plays a significant role in the risk of developing type 2 DM especially after 40yrs [26].

In the present study, the results showed that the lipid and the lipoprotein profiles of the diabetics were higher than that of the controls and is similar as shown by Masum *et al*. [27] and Huang *et al*. [28]. The absolute LDL-C concentration here is not altered significantly as it does not directly reflect the increased number of TG rich-small dense LDL particles which actually increase in number [29].

This study revealed that dyslipidemia was observed in the diabetic population, but that HDL-C and LDL-C were not significantly decreased and increased respectively. This study also showed that when the mean (± SE) of the variables were separated for the male and the female subjects, TC, HDL-C and LDL-C were significantly different in the diabetic group. The results showed a gender difference in the lipid metabolism between the diabetic and the non-diabetic males and females, which was in agreement with the findings of Gustafsson *et al*. [30]. However, Vinter-Repalust *et al*. [31] reported no significant differences in the prevalence of type 2 diabetes mellitus between males and females.

Dyslipidemia was found in 89% diabetic patients studied, which is similar to prevalence found in other studies like 89.1% in Lagos in Nigeria and 90.3% in South Africa [32-34]. The high prevalence observed in this study could be attributed to urbanization in the population from the surrounding villages. Increasing urbanization has been observed to be associated with modernization of life style, which is largely characterized by physical inactivity, change in diet pattern and consequently development of obesity that is greatly considered as a risk factor for developing type 2

DM. Fifty two Percent (52%) of the dyslipidemic subjects had two lipid values outside the normal range of which the most frequent combination was low HDL-C and high TG (20%). This was similar to an earlier work undertaken by Cook et al [35] who observed that 54% of studied subjects had two lipid values, though parameters were reduced HDL-C and increased LDL-C as the most frequent combination outside the normal range in their study.

Our study revealed that 34% type 2 diabetics have high TC level and 56% had high TG. LDL-C was high in 22% patients and HDL-C was low in 52% diabetics, which indicate that the diabetics are prone in future for developing cardiovascular, cerebrovascular complications and malignancies [36, 37] like prostate, colorectal, and breast [38-40]. Although, levels of HDL-C in diabetic individuals are reportedly comparable with that found in non-diabetics, low levels of HDL-C along with elevated TG have been reported in type 2 DM patients as probable cause of CVD [41]. The study showed that type 2 DM influence abnormal lipid profile in diabetes when compared with controls in all the lipid analytes except HDL-C.

The most frequent dyslipidemia entity in diabetics in this study was reduced HDL-C alone (27%) which is similar to previous research works [42]. This same pattern was also observed in African-American diabetics studied in USA using the ADA criteria [43]. Life style, environment, occupation and level of education may account for these differences [34].

TG in diabetic patients of type 2 are proportional to plasma glucose and HbA1c confirming earlier work [44, 45], while TC and LDL-C are proportional to post prandial plasma glucose, proving the importance of better metabolic control for diabetic patients [46].

The abnormal lipid profile observed in type 2 DM is said to be related to insulin resistance as reported in previous studies, which leads to increased release of free fatty acids from fatty tissue, impaired insulin dependent muscle uptake of free fatty acids and increase fatty acid release to the hepatic tissue [47] which has been closely associated with diabetic dyslipidemia, hypertension [48] and enormous risk to vascular diseases.

Chronic hyperglycemia causes glycation of apolipoproteins and interferes with the normal pathways of lipoprotein metabolism [49]. Free fatty acid levels especially from abdominal deposits with direct delivery to the liver, hyperinsulinemia, and hyperglycemia are all stimulators of VLDL-C production in the liver. Turnover of plasma VLDL-C particles may be increased. The consequence may be elevation of plasma VLDL-C concentrations and reduction of plasma HDL-C concentrations [50]. Furthermore, hepatic insulin resistance may result in increased lipoprotein secretion. It has also been identified that insulin can inhibit the assembly and secretion of VLDL-C by increasing posttranslational degradation of apoB and reducing the expression of microsomal TG transfer protein in the liver [51]. These metabolic abnormalities induce not only hyperglycemia, but also dyslipidemia (elevated VLDL-C and reduced HDL-C values) [50].

Lipoprotein lipase an insulin dependent enzyme which together with insulin resistance leads to increase in TG levels, result in type 2 diabetics having high levels of TG. HDL-C levels may be further reduced in DM due to elevated hepatic lipase activity that catalyzes HDL-C [52]. In diabetic patients, inability of insulin to upregulate the apoA-I production (owing to insulin resistance) might also contribute to low HDL-C levels. Furthermore, insulin resistance and low HDL-C level have a common mediator TNF (tumor necrosis factor) [29]. The higher level of fat also prevents the action of insulin or down regulates its receptors and so produce insulin resistance [53]. All these suggest correlation between lipid parameters and plasma glucose levels, their interdependence and importance of better metabolic control in diabetics.

Management of high cholesterol in diabetes has improved in last few years and further hard work is required [54]. If awareness of complications of DM could be increased through media, then blood sugar levels can be tightly controlled resulting into good control of lipid levels which will result into less coronary artery disease and other complications.

CONCLUSION

Type 2 DM patients in this study had elevated levels of TG, TC with slightly elevated levels of LDL-C and reduced levels of HDL-C. This indicates the influence of type 2 DM on abnormal lipid profile of patients with its associated danger of elevated CVD risk. Serum TC, HDL-C and LDL-C were significantly higher in diabetic female in comparison to males, which is not so in controls, indicating gender influence on lipid in diabetics. TG was increased in most diabetic subjects, while increased HDL-C alone was the most common type of dyslipidemia in diabetics. Plasma glucose correlates positively with TG, while TC and LDL-C correlates positively with postprandial plasma glucose only.

Findings from this research will raise awareness on the need for routine lipid profile analysis and abnormal lipid analytes in clinic reviews and treatment of type 2 DM patients in Haryana region. Thus lipid profile analysis must be made an integral part of type 2 DM patients' clinical reviews. Type 2 DM and other diabetics must be educated on the risks they face as a result of their abnormal lipid levels and the necessary steps they need to manage it.

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