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Original Research Article

A Clinical-Epidemiological Assessment of the Lipid Profile in People Diagnosed with Type 2 Diabetes Mellitus

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Abstract

Aim: To assess the lipid profiles in people diagnosed with Type 2 Diabetes Mellitus.

Materials and Methods: This study was conducted in the department of Department of Medicine, PMCH, Patna, Bihar, India from January 2018 to December 2018. The subjects who were enrolled in this study were diabetic patients. A total of 160 diabetic patients (80 males and 80 females) with a history of diabetes for 10 years and 160 healthy controls (80 males and 80 females) were randomly selected and they were examined for dyslipidaemia. Serum total cholesterol was determined by an enzymatic (CHOD-PAP) colorimetric method and triglycerides were determined by an enzymatic (GPO-PAP) method. HDL-Cholesterol was estimated by a precipitant method and LDL-Cholesterol by was estimated by using Friedewald's formula as has been shown below: LDL-C = TC - HDL-C – (TG/5).

Results: The results showed that the mean HDL-C con- centration was non significantly lower in the female diabetics as compared to that in the male diabetics. However, it was on par when it was compared with the controls. The results showed that the frequency of high TC was higher in the diabetic group (10% Vs 1%). The control group had a borderline higher frequency of low HDL-C than that in the diabetic group. The mean TG was highly significant and LDL-C was significant among the lipid profile of the male/female diabetics, whereas TC and LDL-C were significant among the lipid profile of the male/female control groups. The correlation studies showed a negative correlation of FBG with HDL-C and a positive correlation of FBG with TC, TG and LDL-C.

Conclusion: The diabetic patients had a higher prevalence of high serum cholesterol, high triacylglycerol and high LDL-C than the controls, indicating that diabetic patients were more prone to cardiovascular diseases.

Key Words: Diabetes mellitus, Dyslipidaemia, Lipid profile, Triglycerides, HDL-C and LDL-C

Introduction

Type 2 diabetes mellitus (T2DM) represents a significant global health burden, characterized by chronic hyperglycemia resulting from insulin resistance and relative insulin deficiency. Beyond glycemic control, dyslipidemia is a prominent feature in T2DM, contributing significantly to the increased cardiovascular risk observed in these individuals. Dyslipidemia in T2DM is characterized by a distinct pattern of lipid abnormalities, including elevated triglycerides (TG), reduced high-density lipoprotein cholesterol (HDL-C), and often, a predominance of small, dense low-density lipoprotein particles (LDL-P), which collectively contribute to accelerated atherosclerosis and cardiovascular complications. The presence of dyslipidemia in T2DM underscores its clinical significance as a major modifiable risk factor for cardiovascular disease (CVD), the leading cause of morbidity and mortality in this population. [1-6] Individuals with T2DM are prone to a more atherogenic lipid profile characterized by elevated levels of TG-rich lipoproteins, such as very low-density lipoprotein (VLDL) particles, which are a direct consequence of insulin resistance and hepatic overproduction of TG. Concurrently, reduced HDL-C levels, often observed in T2DM, reflect impaired reverse cholesterol transport and contribute to the progression of atherosclerosis. The pathophysiology of dyslipidemia in T2DM is multifactorial, involving complex interactions between insulin resistance, hepatic lipid metabolism, and adipose tissue dysfunction. Insulin resistance leads to increased lipolysis in adipose tissue, releasing free fatty acids into the circulation, which are subsequently taken up by the liver and reassembled into TG-rich VLDL particles. Insulin's suppressive effect on hormone-sensitive lipase activity in adipocytes is impaired, further exacerbating the

release of free fatty acids and promoting dyslipidemia. Additionally, impaired insulin signaling in the liver enhances hepatic production of apolipoprotein B (apoB)-containing lipoproteins, contributing to elevated LDL-P levels and promoting a proatherogenic lipid profile. Effective management of dyslipidemia in individuals with T2DM is crucial for reducing cardiovascular risk and improving long-term outcomes. Current guidelines recommend aggressive lipid-lowering therapy to target specific lipid abnormalities, including statins as first-line agents to lower LDL-C levels and reduce CVD risk. Combination therapies with fibrates or omega-3 fatty acids may be considered to address residual hypertriglyceridemia and low HDL-C levels. Lifestyle modifications, including dietary interventions and increased physical activity, play a pivotal role in improving lipid profiles and overall cardiovascular health in individuals with T2DM. [7-15]

Materials and Methods

This study was conducted in the department of Department of Medicine, PMCH, Patna, Bihar, India from January 2018 to December 2018. The subjects who were enrolled in this study were diabetic patients. A total of 160 diabetic patients (80 males and 80 females) with a history of diabetes for 10 years and 160 healthy controls (80 males and 80 females) were randomly selected and they were examined for dyslipidaemia. Patients with other ailments and metabolic disorders were excluded from the study. Laboratory tests were used to confirm the absence of diabetes in the control group and also by asking questions about the sings of diabetes such as polyuria, polydipsia and recent weight loss. Ethical clearance was sought and obtained for the study from the hospital. The aim of the study was explained to the subjects and those who gave their consent were included in the study. In both the patients and the controls, about 5 ml of fasting blood was obtained by venipuncture by using sterile disposable syringes and needles. The blood was collected into centrifuge tubes. It was allowed to clot and it was then centrifuged at 3000 rpm for 15 min at room temperature. The serum which was obtained was pipetted into a clean blood sample bottle and analysed on the day of col- lection for serum sugar and lipid profile tests. Serum total cholesterol was determined by an enzymatic (CHOD-PAP) colorimetric method[11] and triglycerides were determined by an enzymatic (GPO-PAP) method. [12] HDL-Cholesterol was estimated by a precipitant method [13] and LDL-Cholesterol by was estimated by using Friedewald's formula [14] as has been shown below: LDL-C = TC - HDL-C – (TG/5).

Serum glucose was determined by using the glucose oxidase enzymatic method.[15] All the parameters which were under investigation were determined in the serum of the subjects by using commercially available reagent kits. The lipid profile of the subjects was classified, based on the ATP III model. [16]

The values of all the parameters were given in mg/dl and they were expressed as mean ± SD. 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.

Results

The mean age of the subjects were 51.04 ± 11.79 and 47.20 ± 10.65 years for the diabetic and the control groups respectively. The sex distribution showed an equal number of males and fe- males in all the groups. [Table 1] shows the mean total cholesterol, triacylglycerols, LDL-C and the fasting blood sugar levels which were highly significant in the diabetics as compared to those in the controls.[Table 2] shows the comparison between the mean biochemical variables with respect to gender in the diabetics and the controls respectively. The results showed that the mean HDL-C con- centration was non significantly lower in the female diabetics as compared to that in the male diabetics. However, it was on par when it was compared with the controls. [Table 3] shows the frequencies of the TC, TG, HDL-C and the LDL-C concentrations in both the diabetic and the control groups. The results showed that the frequency of high TC was higher in the diabetic group (10% Vs 1%). The control group had a borderline higher frequency of low HDL-C than that in the diabetic group. The mean TG was highly significant and LDL-C was significant among the lipid profile of the male/female diabetics, whereas TC and LDL-C were significant among the lipid profile of the male/female control groups. The correlation studies showed a negative correlation of FBG with HDL-C and a positive correlation of FBG with TC, TG and LDL-C.

Table 1: Biochemical Parameters of Diabetic and Control Groups

Parameters	Diabetics (n=160) Mean ± SD	Control (n=160 Mean ± SD	t table value
Total cholesterol (mg/dl)	184.27 ± 35.82	160.37 ± 27.34	6.69**
Triacylglycerols (mg/dl)	198.18 ± 111.02	131.98 ± 53.08	6.78**
HDL-C (mg/dl)	37.44 ± 4.47	37.68 ± 5.99	0.39
LDL-C (mg/dl)	106.96 ± 35.10	96.53 ± 25.71	3.02**
FBS (mg/dl)	170.29 ± 57.75	82.14 ± 12.83	18.79**

[Table -2]: Comparison of the Biochemical Parameters in the Males and Females in both Groups.

Parameters	Diabetics			Controls		
	Male (n=80) Mean ± SD	Female (n=80) Mean ± SD	T Table value	Male (n=80) Mean ± SD	Female (n=80) Mean ± SD	T Table value
Total cholesterol (mg/dl)	184.18 ± 38.59	184.36 ± 32.81	0.03	154.95 ± 25.90	165.79 ± 27.67	2.49
Triacylglycerols (mg/dl)	228.05 ± 137.09	168.30 ± 63.82	3.51**	129.16 ± 58.04	134.80 ± 47.44	0.67
HDL-C (mg/dl)	37.28 ± 6.17	37.08 ± 5.75	0.26	37.44 ± 5.06	37.45 ± 3.79	0.02
LDL-C (mg/dl)	100.29 ± 36.67	113.63 ± 32.08	2.63*	91.68 ± 25.08	101.38 ± 25.41	2.45
FBS (mg/dl)	175.61 ± 68.61	164.96 ± 43.63	1.16	80.90 ± 13.74	83.39 ± 11.72	1.22

Table-3]: Frequency of the Biochemical Variables in the Diabetic and Control Groups according to the ATP III classification

Parameter Total cholesterol (mg/dl	Diabetics (%)	Controls (%)
Desirable (<200)	113 (71%)	145 (91%)
Borderline high (200-239)	31 (19%)	13 (8%)
High (□240)	16 (10%)	2 (1%)
Triacylglycerols (mg/dl)		
Normal (<150)	61 (38%)	130 (81%)
Borderline high (150-199)	39 (24%)	17 (11%)
High (200-249)	60 (38%)	21 (8%)
HDL-C (mg/dl)		
Low (<40)	113 (71%)	100 (63%)
Borderline high (40-59)	45 (28%)	54 (34%)
High (□60)	2 (1%)	6 (3%)
LDL-C (mg/dl)		
Optimal (<100)	69 (43%)	103 (65%)
Near optimal (100-129)	51 (32%)	42 (26%)
Borderline high (130-159)	26 (17%)	11 (7%)
High (160-189)	7 (4%)	2 (1.25%)
Very high (□190)	7 (4%)	1 (0.75%)

Discussion

Diabetic patients have many complications which include elevated levels of LDL-C and triacylglycerols, low levels of HDL-C and a preponderance of abnormalities in the composition of the smaller, dense particles [17]. 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 that they were in agreement with the findings of Idogun et al., [18] and Albrki et al., [19]. This study also showed that while the mean (± SD) of the variables were separated for the male and the female subjects, TG 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., [20]. However, Vinter-Repalust et al., [21] reported no significant differences in the prevalence of type 2 diabetes mellitus between males and females. The prevalence rates for high TC, combined high and very high LDL-C and low HDL-C in the diabetic subjects were 10%, 8% and 71% respectively. The prevalence rates of high TC and TG in this study were 10% and 38% respectively. The correlation studies showed a negative non significant correlation (r= -0.024) between FBG and HDL-C, whereas positive significant correlations were recorded between FBG and TC (r = 0.584) and FBG and TG (r = 0.514). This study revealed that dyslipidaemia was observed in the diabetic population, but that HDL-C was not significantly decreased.

Conclusion

The diabetic patients had a higher prevalence of high serum cholesterol, high triacylglycerol and high LDL-C than the controls, indicating that diabetic patients were more prone to cardiovascular diseases.

References

1. Kolhar U, Priyanka P. Study of serum lipid profile in type 2 diabetes mellitus patients and its association with diabetic nephropathy. Int J Adv Med 2017;4:1513-6..

2. Sadeghi E, Hosseini SM, Vossoughi M, Aminorroaya A, Amini M. Association of Lipid Profile with Type 2 Diabetes in First-Degree Relatives: A 14-Year Follow-Up Study in Iran. Diabetes Metab Syndr Obes. 2020 Aug 5;13:2743-2750. doi: 10.2147/DMSO.S259697. PMID: 32801820; PMCID: PMC7415448. Gordon DJ, Probstfield JL, Garrison RJ, et al. High-density lipoprotein cholesterol and cardiovascular disease. Four prospective American studies. Circulation. 1989;79(1):8-15.
3. Sharahili AY, Mir SA, ALDosari S, Manzar MD, Alshehri B, Al Othaim A, Alghofaili F, Madkhali Y, Albenasy KS, Alotaibi JS. Correlation of HbA1c Level with Lipid Profile in Type 2 Diabetes Mellitus Patients Visiting a Primary Healthcare Center in Jeddah City, Saudi Arabia: A Retrospective Cross-Sectional Study. *Diseases*. 2023; 11(4):154. https://doi.org/10.3390/diseases11040154.
4. Krauss RM. Lipids and lipoproteins in patients with type 2 diabetes. Diabetes Care. 2004; 27 (6):1496-1504.
5. Austin MA, King MC, Vranizan KM, et al. Atherogenic lipoprotein phenotype. A proposed genetic marker for coronary heart disease risk. Circulation. 1990;82(2):495-506.
6. Hu FB, Stampfer MJ, Solomon C, et al. Physical activity and risk for cardiovascular events in diabetic women. Ann Intern Med. 2001;134(2):96-105.
7. Ginsberg HN, Elam MB, Lovato LC, et al. Effects of combination lipid therapy in type 2 diabetes mellitus. N Engl J Med. 2010;362 (17):1563-1574.
8. Goldberg RB, Mellies MJ, Sacks FM, et al. Cardiovascular events and their reduction with pravastatin in diabetic and glucose-intolerant myocardial infarction survivors with average cholesterol levels: subgroup analyses in the cholesterol and recurrent events (CARE) trial. Circulation. 1998;98(23):2513-2519.
9. Gharib, A.F.; Saber, T.; EL Askary, A.; Alharthi, A.; Alsalmi, N.A.; Alhashmi, S.T.; Al-Asiri, R.F.; Shafie, A. Relation of Hypoxia Inducible Factor, Dyslipidemia and CAD Saudi Patients with Type 2 Diabetes. *In Vivo* **2022**, *36*, 2481–2489
10. The American Diabetes Association. The management of dyslipidemia in adults with diabetes. Diabetes Care 1999; 22 (Suppl. I):S 56-S59.
11. Allain CC, Poon IS, Chan CHG, Richmond W. Enzymatic determina- tion of serum total cholesterol. Clin. Chem. 1974; 20:470-71.
12. Jacobs NJ, Van Denmark PJ. Enzymatic determination of serum trig- lycerides. Biochem. Biophys 1960; 88:250-55.
13. Gordon T, et al. An enzymatic method for the determination of the serum HDL-cholesterol. Am.J.Med 1977; 62:707-08.
14. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concen- tration of LDL-cholesterol. Clin. Chem 1972; 18(6):499-515.
15. Trinder P. Determination of blood glucose by using 4-aminophenazone as an oxygen acceptor. J.Clin.Path 1969; 22:246.
16. The National Cholesterol Education Program (NCEP). Expert panel on the detection, evaluation and the treatment of high blood cholesterol in adults (Adults Treatment Panel III). JAMA 2001; 285:2486-97.
17. Haffner SM. Management of dyslipidemia in adults with diabetes.Diabetes Care 1998; 21: 9(1):1600-78.
18. Idogun ES, Unuigbe EI, Ogunro PS, Akinola OT, Famodu AA. Assess- ment of the serum lipids in Nigerians with type 2 diabetes mellitus complications. Pak. J. Med. Sci. (Part 1) 2007; 23(5):708-12.
19. Albrki WM, Elzouki AN Y, EL-Mansoury ZM, Tashani OA. Lipid profiles in Libian type 2 diabetes. J.Sci.Appls 2007; 1(1):18-23.
20. Gustafsson I, Brendorp B, Seibaek M, Burchardt H, Hildebrandt P. The influence of diabetes and the diabetes – gender interaction on the risk of death in patients who were hospitalized with congestic heart failure. J.AM.Coll.Cardiol 2004; 43(5):771-77.