

STUDY OF TOTAL CHOLESTEROL, TRIACYLGLYCEROLS, HIGH-DENSITY LIPOPROTEIN CHOLESTEROL IN TYPE 2 DIABETES MELLITUS

ANITA P MANDARE¹, NEELAM DEOKAR², SMITA V PATIL^{3*}, GAIKWAD PANDURANG B⁴

¹Department of Physiology, RCSM Government Medical College & Hospital, Kolhapur Maharashtra, India. ²Department of Physiology, Government Medical College, Miraj, Maharashtra, India. ³Department of Physiology and Biochemistry, Bharati Vidyapeeth Deemed University Dental College & Hospital, Sangli, Maharashtra, India. ⁴Department of Physiology, Prakash Institute of Medical Science and Research, Islampur, Sangli, Maharashtra, India. Email: mailmesmita.patil@rediffmail.com

Received: 06 August 2016, Revised and Accepted: 20 October 2016

ABSTRACT

Objective: There are probably 100 million people in the world with diabetes mellitus (DM), and incidences of diabetes are on the rise. Dyslipidemia is one of the common disorders which are seen in most of the diabetes patients, which causes cardiovascular disorders. The aim of this study is to investigate the total cholesterol (TC), triacylglycerols (TGs), high-density lipoprotein cholesterol (HDL-C) in Type 2 DM, and healthy controls.

Methods: The study was conducted on 50 controls and 50 Type 2 diabetic subjects between age group of 30 and 60 years. Serum TC was determined by an enzymatic (cholesterol oxidase/phenol-aminophenazone [PAP]) colorimetric method and TGs were determined by an enzymatic (glycerol phosphate oxidase-PAP) method, and HDL-C was estimated by a precipitant method. Statistical analysis was done using unpaired *t*-test.

Results: The mean value of TC, TGs, and HDL-C in normal subjects is 165.5±24.24, 118.7±41.58, 28.38±7.85 mg/dl, respectively, and the value of TC, TGs, and HDL-C in diabetic patients is 179±31.69, 164.35±27.93, 25.4±6.86 mg/dl, respectively. The observed difference in the means of TC, TGs, and HDL-C in normal and diabetic are statistically significant (*p*<0.05).

Conclusion: From the present study, it is concluded that TC, TGs, and HDL-C levels for all persons with Type 2 DM should be done as a routine test. Furthermore, early diagnosis and treatment of dyslipidemia can be used as a preventive measure for the development of cardiovascular disease in Type 2 diabetes.

Keywords: Diabetes mellitus, Dyslipidemia, Lipid profile, Total cholesterol, Triglycerides, High-density lipoprotein cholesterol.

© 2017 The Authors. Published by Innovare Academic Sciences Pvt Ltd. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>) DOI: <http://dx.doi.org/10.22159/ajpcr.2017.v10i2.14535>

INTRODUCTION

In India, diabetes carries a high risk of atherosclerosis and cardiovascular disease (CVD), especially coronary heart disease (CHD) and stroke, is the most leading cause of death among patients with Type 2 diabetes. Diabetes mellitus (DM) is the most common metabolic disorder affecting the people all over the world. It is a group of metabolic disorders characterized by hyperglycemia resulting from defect in insulin secretion, insulin action, or both [1]. It is a clinical syndrome characterized by hyperglycemia, caused by a complicated interplay of genes, obesity, environment, increased glucose production in liver, increased fat breakdown, and an absolute or relative deficiency of insulin. DM arises when insufficient insulin is produced, or when the available insulin does not function correctly. Without insulin, the amount of glucose in the blood stream is abnormally high, causing unquenchable thirst and frequent urination. There are probably 100 million people in the world with DM and incidences of diabetes are on the rise [2].

Type 2 DM, obesity, and dyslipidemia are considered as independent risk factors for CHD and cerebrovascular disease. Dyslipidemia (raised triglycerides [TGs], raised cholesterol, and low high-density lipoprotein [HDL]) were common in patients with Type 2 DM with other features of insulin resistance such as hyperinsulinemia, hypertension with central obesity together known as metabolic syndrome or Reaven's syndrome; and is strongly associated with atherosclerosis. Dyslipidemia is one of the major CVD risk factors and plays an important role in the progress of atherosclerosis, the underlying pathology of CVD.

Hence, the present work was undertaken to assess the total cholesterol (TC), TGs, and HDL cholesterol (HDL-C) of diabetic patients and compared them with the controls.

METHODS

After the approval of the Institutional Ethical Committee, a case-control study was carried out on 50 normal individuals taken from the Government Medical College, Miraj, and 50 diabetic patients were taken from the Dr. Patwardhan's Endocrinology and Diabetic Clinic, Miraj (Maharashtra). The diabetic patients taken were undiagnosed cases and were only diagnosed when fasting blood sugar levels were taken. Clinical history was also documented. Patient who were on steroid therapy in past 3 months, any liver, kidney or cardiac failure, neoplasm and patients who were on any antilipidemic therapy were excluded from the study. Informed written consent was obtained from the patients for the study.

Venous blood sample was collected after overnight (12 hrs) fasting, in fluoride Vacutainer and samples were centrifuged to obtain plasma for the biochemical analysis. Serum TC was determined by an enzymatic (cholesterol oxidase/phenol-aminophenazone [PAP]) colorimetric method [3]; TGs were determined by an enzymatic (glycerol phosphate oxidase-PAP) method [4]; HDL-C was estimated by a precipitant method [5].

Statistical analysis

Results were presented as mean±standard deviation. Unpaired *t*-test was used to find the significance of study parameters

Table 1: Comparison of TC, TGs, and HDL-C between control and Type 2 diabetic groups

Parameters	Mean±SD of control (N=50)	Mean±SD of Type II diabetic (N=50)	SE	Z value	Observed difference	p value
TC (mg/dl)	165.5±24.24	179.3±31.69	5.64	2.44	13.8	<0.05*
TG (mg/dl)	118.7±41.58	164.35±27.93	7.08	6.44	45.65	<0.001*
HDL-C (mg/dl)	28.38±7.85	25.4±6.86	1.47	2.02	2.98	<0.05*

*Significant. NS: Not significant, HDL-C: High-density lipoprotein cholesterol, TG: Triglyceride, TC: Total cholesterol, SD: Standard deviation, SE: Standard error

using SPSS 16.0 version. P<0.05 was considered as statistically significant.

OBSERVATIONS AND RESULTS

In Table 1, TC and TGs are more significantly increased in diabetic patient as compared to control, but HDL-C is significantly decreased in the diabetic patient as compared to control.

DISCUSSION

In the present study, the results showed that the TC and TGs of the diabetics were higher than that of the controls, but HDL-C was decreased in diabetic group than controls.

Tables 2-4 show distribution of subject according to TC (mg/dl), TGs (mg/dl), and HDL-C (mg/dl) between normal and diabetic subjects.

The mean value of TC, TGs, and HDL-C in normal subjects is 165.5±24.24, 118.7±41.58, 28.38±7.85 mg/dl, respectively, and the value of TC, TGs, and HDL-C in diabetic patients is 179±31.69, 164.35±27.93, 25.4±6.86 mg/dl, respectively. The observed difference in the means of normal and diabetic patients for TC, TGs, and HDL-C is 13.8, 45.65, and 2.98, respectively, and is statistically significant (p<0.05) (Table 1). The mean value of TC and TGs was increased, but HDL-C was decreased in diabetic patients as compared to control.

The present study is similar to the studies performed by Kreisberg [6] and Kesavulur *et al.* [7], Idogun *et al.* [8], Albrki *et al.* [9], Harder H *et al.* [10], and Gordon *et al.* [11]. Similar studies done by Vikram *et al.* in New Delhi showed a significant increase in TGs and high waist to hip ratio in young adults with family history of Type 2 DM [12].

The probable cause for increased TC, TGs, and a reduction in HDL-C levels in Type 2 DM patients is due to an abnormal pattern of cholesterol transport and transfer. The transfer of lecithin-cholesterol acyltransferase enzyme, which esterifies free cholesterol to very low-density lipoprotein (VLDL) and LDL, is inhibited; with a concomitant increase in their transfer to HDL [13]. Lower HDL-C level is attributed to TG enrichment by cholesterol ester transfer protein and increased hepatic TG lipase activity [14]. Although liver is produced the HDL particles, a significant part of HDL is formed from remnant particles of TG-rich lipoproteins as metabolized. This metabolism is often defective in diabetes, lowering the production of HDL-C from the liver by protein which is called cholesterol ester transport protein transports cholesterol ester away from HDL particles in exchange for TG from the VLDL particles. This transport protein lowers HDL-C in the blood, in addition, it promotes for small, dense LDL particles [15]. Lipid levels affected by glucose levels because metabolism of carbohydrates and lipid is interrelated to each other because any disorder in metabolism of carbohydrate leads to a disorder in metabolism of lipid, so high concentration of cholesterol, TGs, and a reduction in HDL-C levels leads to insulin resistance with or without hyperglycemia which is related to qualitative changes in the lipid profile [16].

CONCLUSION

From the present study, it is concluded that TC, TGs, and HDL-C levels for all persons with Type 2 DM should be done as a routine test. Furthermore, early diagnosis and treatment of dyslipidemia can be

Table 2: Distribution of TC (mg/dl) between normal and diabetic subjects

TC (mg/dl)	Normal subjects (N=50)	Diabetic subjects (N=50)
90-110	1	0
110-130	4	2
130-150	6	7
150-170	15	9
170-190	17	15
190-210	6	10
210-230	1	2
230-250	0	3
250-270	0	2

TC: Total cholesterol

Table 3: Distribution of TG (mg/dl) between normal and diabetic subjects

TG (mg/dl)	Normal subjects (N=50)	Diabetic subjects (N=50)
45-75	8	0
75-105	15	0
105-135	8	8
135-165	11	18
165-195	7	17
195-225	0	5
225-255	1	2
45-75	8	0

TG: Triglyceride

Table 4: Distribution of subject according to HDL-C (mg/dl) between normal and diabetic subjects

HDL-C (mg/dl)	Normal subjects (N=50)	Diabetic subjects (N=50)
15-20	7	11
20-25	10	17
25-30	15	11
30-35	6	4
35-40	10	4
40-45	0	2

HDL-C: High-density lipoprotein cholesterol

used as a preventive measure for the development of CVD in Type 2 diabetes. All persons with Type 2 diabetes must be started on primary prevention by encouraging healthy lifestyle diets so as to reduce the risk of CHD and atherosclerosis.

REFERENCES

1. Proces S, Delgrange E, Vander Borgh TV, Jamart J, Donckier JE. Minor alterations in thyroid-function tests associated with diabetes mellitus and obesity in outpatients without known thyroid illness. *Acta Clin Belg* 2001;56(2):86-90.
2. Unwin N, Sobngwi E, Alberti KG. Type 2 diabetes: The challenge of preventing a global epidemic. *Diabetes Int* 2001;11:4-8.
3. Allain CC, Poon IS, Chan CH, Richmond W. Enzymatic determination of serum total cholesterol. *Clin Chem* 1974;20(4):470-1.
4. Jacobs NJ, Van Denmark PJ. Enzymatic determination of serum

- triglycerides. *Biochem Biophys* 1960;88:250-5.
5. Gordon T, Gordon M. An enzymatic method for the determination of the serum HDL-cholesterol. *Am J Med* 1977;62:707-8.
 6. Kreisberg RA. Diabetic dyslipidemia. *Am J Cardiol* 1998;82(12A):67U-73.
 7. Kesavulur MM, Giri R, Rao BK, Apparao C. Lipid peroxidation and antioxidant enzyme levels in Type II diabetics with micro vascular complications. *Diabetes Metab* 2000;26(5):387-92.
 8. Idogun ES, Unuigbo EI, Ogunro PS, Akinola OT, Famodu AA. Assessment of the serum lipids in Nigerians with Type 2 diabetes mellitus complications. *Pak J Med Sci (Part 1)* 2007;23(5):708-12.
 9. Albrki WM, Elzouki AN, EL-Mansoury ZM, Tashani OA. Lipid profiles in Libian Type 2 diabetes. *J Sci Appl* 2007;1(1):18-23.
 10. Harder H, Dinesen B, Astrup A. The effect of a rapid weight loss on lipid profile and glycemic control in obese Type 2 diabetic patients. *Int J Obes Relat Metab Disord* 2004;28(1):180-2.
 11. Gordon L, Ragoobirsingh D, Morrison EY, Choo-Kang E, McGrowder D, Martorell E. Lipid profile of Type 2 diabetic and hypertensive patients in the Jamaican population. *J Lab Physicians* 2010;2(1):25-30.
 12. Vikram NK, Tandon N, Misra A, Srivastava MC, Pandey RM, Mithal A, et al. Correlates of Type II diabetes mellitus in children, adolescents and young adults in North India: A multisite collaborative case control study. *Diabet Med* 2006;23(3):293-8.
 13. Fielding CJ, Reaven GM, Fielding PE. Human noninsulin-dependent diabetes: Identification of a defect in plasma cholesterol transport normalized *in vivo* by insulin and *in vitro* by selective immunoadsorption of apolipoprotein E. *Proc Natl Acad Sci U S A* 1982;79:6365-9.
 14. DeFronzo RA. Lilly lecture 1987. The triumvirate: Beta-cell, muscle, liver. A collusion responsible for NIDDM. *Diabetes* 1988;37(6):667-87.
 15. Chatterjee MN, Shinde R. *Metabolism of carbohydrates*. 6th ed. New Delhi, India: Jaypee Brothers Medical Publisher; 2005. p. 266-330.
 16. Del Prato S, Bonadonna RC, Bosom E, Gulli G, Solini A, Shank M, et al. Characterization cellular defects of insulin action in Type2 (noninsulin dependent) diabetes mellitus. *J Clin Invest* 1993;91(2):484-94.