Original Article

Evaluation of Serum Triglyceride and TG/HDL-Cholesterol Ratio in Good and Poor Control Type-2 Diabetes Mellitus

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Abstract

Introduction: *Patients with type-2 diabetes have an increased prevalence of dyslipidemia, which contributes to their high risk of cardiovascular diseases. The mortality and morbidity are increased if there is poor glycemic control. Diabetic dyslipidemia is characterized by elevated TG levels, decreased HDL-C levels and increased levels of LDL-C. The aim of this study was to evaluate serum triglyceride and TG/HDL-cholesterol ratio in good and poor control type-2 diabetes mellitus.* ***Materials and Methods:*** *The present study was carried out in departments of Biochemistry, Medicine and Endocrinology of Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh between the periods of July 2020 to June 2021. By convenient sampling, a total of 50 subjects aged between 30-59 years were enrolled in this study. Study subjects were divided into 2 groups. Group-A included subjects with good glycemic control (n=25) and group-B included subjects with poor glycemic control (n=25).* ***Results:*** *The mean HbA1c level was significantly higher in poor glycemic group than good glycemic group (10.9±12.02% vs. 5.98±0.95%). The mean TG was 173.16±36.9 mg/dl in good glycemic control group and 219.4±70.32 mg/dl in poor glycemic group. There was significant difference in TG/HDL-C ratio in 2 groups. Serum triglyceride and TG/HDL-C ratio had positive correlation with HbA1c but was not significant.* ***Conclusion:*** *Poor glycemic control is associated with dyslipidemia in Type-2 Diabetes Mellitus.*

Keywords: Type-2 Diabetes, HbA1c, Serum Triglyceride, TG/HDL-Cholesterol Ratio.
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Introduction

Diabetes mellitus (DM) is characterized by chronic hyperglycemia and impaired carbohydrates, lipids and proteins metabolism caused by complete or partial insufficiency of insulin secretion and/or insulin action[1]. There are two primary forms of diabetes, insulin-dependent diabetes mellitus (type-1 diabetes mellitus, T1DM) and non-insulin-dependent diabetes mellitus (type-2 diabetes mellitus, T2DM)[1-2]. T2DM is the most common form of DM, which accounts for 90% to 95% of all diabetic patients. The prevalence of T2DM worldwide has increased dramatically in recent decades and is expected to increase to 300 million by the year 2025[3].

Dyslipidemia in individuals with type-2 diabetes is very common, with a prevalence of 72-85%. This phenomenon is associated with a significantly increased risk of coronary artery disease relative to individuals without diabetes[4]. Characteristic abnormalities of lipids in type-2 diabetes include elevated TG levels, decreased atheroprotective HDL-C levels and increased levels of LDL-C[5]. A few factors may contribute to the alterations in lipid metabolism observed in patients with diabetes, including insulin deficiency or resistance, adipocytokines and hyperglycemia[6]. This lipoprotein pattern is present even before the onset of diabetes and is strongly associated with insulin resistance[7].

Hypertriglyceridemia is considered the dominant lipid abnormality in insulin resistance and plays a pivotal role in determining the characteristic lipid profile of diabetic dyslipidemia[8]. Elevated triglyceride levels are the result of increased production and decreased clearance of triglyceride-rich lipoproteins in both fasting and non-fasting states. Increased production of very low-density lipoprotein (VLDL), the main transporter of fasting

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triglycerides, is a prominent feature of insulin resistance[8]. The decrease level of HDL-C concentration in patients with type-2 diabetes is due to increased catabolism of HDL which is demonstrated in different Kinetic studies using radioisotopes and stable isotopes[9]. Hypertriglyceridemia is said to be a major contributing factor to the accelerated HDL catabolism observed in type-2 diabetes mellitus[10].

Glycosylated hemoglobin (HbA1c) is an absolute indicator of long-term blood glucose control and is a gold standard of glycemic control in subjects with type-2 diabetes mellitus[11]. In accordance with its function as an indicator for the mean blood glucose level, HbA1c predicts the risk for the development of diabetic complications in diabetes patients.

The aim of this study was to evaluate serum triglyceride and TG/HDL-cholesterol ratio in good and poor control type-2 diabetes mellitus.

Materials and Methods

The study was conducted from July 2020 to June 2021 at Sir Salimullah Medical College (SSMC) and Mitford Hospital, Dhaka, Bangladesh. Ethical permission was taken from the Institutional Review Board of Sir Salimullah Medical College (SSMC). By convenient sampling, a total of 50 subjects of age between 30-59 years attending in Biochemistry department and department of Medicine and Endocrinology of SSMC, were enrolled in this study. The subjects with type-1 diabetes mellitus, liver disease, gastrointestinal disease, thyroid disease, underweight or morbid obese, taking medications like corticosteroids, antiepileptic, methotrexate, amiodarone, tamoxifen or other hepatotoxic drugs were excluded from this study.

After enrollment, they were grouped based on glycemic control. Among them (group-A), 25 were diabetic with good glycemic control (T2DM subjects having duration of ≤8 years and HbA1c level ≤7%) and (group-B), 25 were diabetic with poor glycemic control (T2DM subjects havingduration of >8 years and HbA1c level >7%). Informed written consents were taken and with allaseptic precaution fasting blood samples were collected from each study subject. Initial evaluation of the study subjects by history and clinical examination was performed and were recorded in the preformed data collection sheet. Demographic profile and pulse, blood pressure, height, weight, WC etc. were measured. HbA1c was measured using immunoflurescence method. Fasting serum TG and HDL were measured by enzymatic method. The statistical analysis was carried out using SPSS version 23. Categorical variables were expressed as frequency and percentage. Continuous variables were expressed as mean and standard deviation. An unpaired t-test was performed to compare good glycemic control and poor glycemic control. To determine the linear relation between continuous variables, Pearson's correlation coefficient was done. A p-value of <0.05 was considered statistically significant.

Results

Distribution of study subjects according to age was shown in table-I which shows that almost half (44%) patients belonged to age 30-39 years in group-A and 16% in group-B. The mean age was 42.04±9.14 years in group-A and 47.8±7.88 years in group-B. Figure-1 shows that more than half (56%) patients were male in group-A and 72% in group-B.

The mean FPG was 5.45±0.88 mmol/l in group-A and 10.8±5.62 mmol/l in group-B. The mean HbA_1C was 5.98±0.95% in group-A and 10.9±12.02% in group-B. The difference was statistically significant (p<0.05) between two groups (Table-II).

Table-III shows the mean TG was 173.16±36.9 mg/dl in group-A and 219.4±70.32 mg/dl in group-B. The mean High-density lipoprotein (HDL-C) was 41.92±2.96 mg/dl in group-A and 39.32±4.23 mg/dl in group-B. The mean TG/HDL-C ratio was 4.19±1.11 in group-A and 5.8±2.4 in group-B. The difference was statistically significant (p=0.001) between the two groups.

Table-I: Distribution of the respondents according to age (n=50)

Age (in years)	Group A (n=25)		Group B (n=25)	
	n	%	n	%
30-39	11	44.0	4	16.0
40-49	7	28.0	7	28.0
50-59	7	28.0	14	56.0
Mean ± SD	42.04±9.14		47.8±7.88	

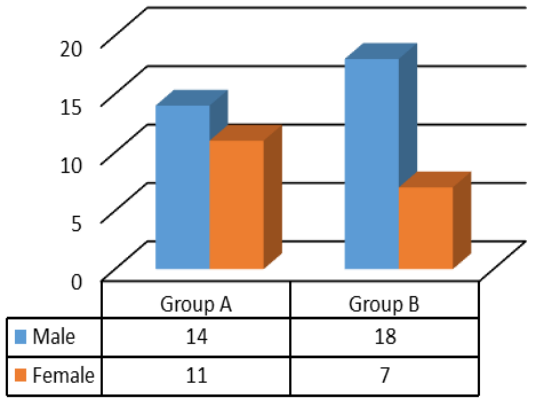

Figure-1: Distribution of the gender in the study cases (n=50)

Table-II: Glycemic status of the study subjects (n=50)

Glycemic profile	Group A (n=25)	Group B (n=25)	p-value
	Mean±SD (Range)	Mean±SD (Range)	
FPG (mmol/l)	5.45±0.88 (4.8-8)	10.8±5.62 (3.8-25.2)	0.001*
HbA_1C (%)	5.98±0.95 (5.5-6.4)	10.9±12.02 (7.6-10.4)	0.035*

* = significant, p-value reached from Unpaired t-test

Table-III: Comparison of serum Triglyceride, HDL-Cholesterol and TG/HDL-Cholesterol ratio in between two study groups (n=50)

Serum TG, HDL-C and TG/HDL-C ratio	Group A (n=25)	Group B (n=25)	p-value
	Mean±SD (Range)	Mean±SD (Range)	
TG (mg/dl)	173.16±36.9 (114-242)	219.4±70.32 (114-383)	0.001[s]
HDL-C (mg/dl)	41.92±2.96 (36-48)	39.32±4.23 (30-46)	0.001[s]
TG/HDL-C ratio	4.19±1.11 (2.5-6.72)	5.8±2.4 (2.48-10.53)	0.001[s]

[s] = significant, p-value reached from Unpaired t-test

Table-IV: Distribution of the respondents according to TG/HDL-C ratio (n=50)

TG/HDL-C ratio	Group A (n=25)		Group B (n=25)	
	n	%	n	%
≤2	0	0.0	0	0.0
>2	25	100.0	25	100.0

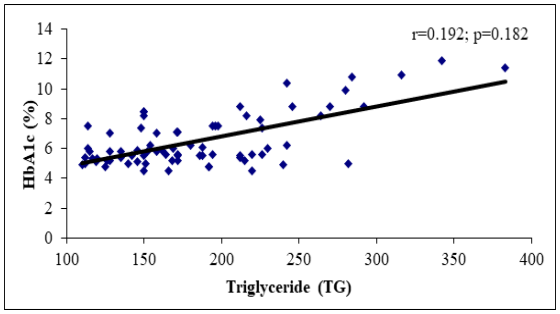

Figure-2: Scatterplot graph showing that the triglyceride had positive correlation with HbA1c which was not significant (r=0.192; p=0.182)

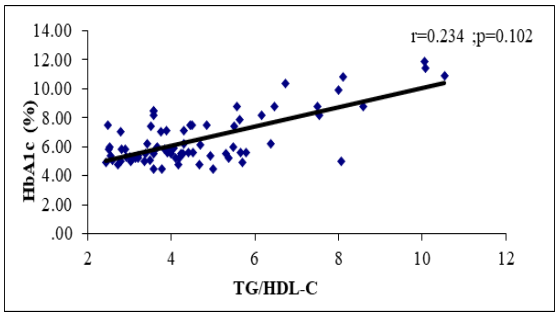

Figure-3: Scatterplot graph showing that the TG/HDL-C had positive correlation with HbA1c which was not significant (r=0.234; p=0.102).

Table-IV shows the distribution of the respondents according to TG/HDL-C ratio. It was observed that all (100%) patients of group-A and group-B belonged to TG/HDL-C ratio >2. Fig-2 and Fig-3 shows that triglyceride and TG/HDL-C ratio had positive correlation with HbA1c but were not statistically significant.

Discussion

Diabetes mellitus is a common secondary cause of dyslilipidemia[12]. Dyslipidemia makes diabetic patients more susceptible to coronary artery disease which is the major cause of increased mortality and morbidity in these[13]. This study showed that almost half (44%) patients were aged 30-39 years in group-A and 16% in group-B. The mean age was 42.04±9.14 years in group-A and 47.8±7.88 years in group-B. In this study there were more males than females with type-2 diabetes mellitus in both groups. The high proportion of females in this study may be due to the nature of population admitting to this hospital. Aamir et al.[14] and Saleh et al.[15] also reported similar age distribution of diabetes mellitus subjects.

The Diabetes complications and control trial (DCCT) established HbA1c as the gold standard of glycemic control[16]. Increased HbA1c is now considered as an independent risk factor for cardiovascular disease[17]. The level of HbA1c value <7.0% was said to be appropriate for reducing the risk of cardiovascular complications[16]. The current study showed mean HbA1c were significantly higher ($p<0.05$) in T2DM with poor glycemic control when compared to good glycemic control group. This observation was consistent with the findings of Tabazzum R, et al[18].

Hypertriglyceridemia is considered the dominant lipid abnormality in insulin resistance and plays a pivotal role in determining the characteristic lipid profile of diabetic dyslipidemia[8]. In this study serum TG level was significantly higher in poor glycemic control group than good glycemic control group (219.4±70.32 vs 173.16±36.9, p=0.001). The significantly lower value of the ratio TG/HDL-cholesterol was found in the group of patients with good glycemic control. (p=0.001) The mean value of the ratio TG/HDL-cholesterol in a group of patients with good glycemic control was 4.19±1.11 and in the group of patients with poor glycemic control was 25.8±2.4. Similar findings were observed in other studies[19,20].

In the current study, we studied the correlations among HbA1c with serum triglyceride and TG/HDL-C ratio and found a positive correlation but were not statistically significant. In a study by Panjeta E, et al[19] showed the positive significant correlation between HbA1c and serum TG. Khan HA, et al[21] in their study showed the impact of glycaemic control on various lipid parameters in which severity of dyslipidemia increases in patients with higher HbA1c value.

Conclusion

This study showed a relationship between HbA1c and TG, as well as HbA1c and TAG/HDL-cholesterol ratio, indicating that HbA1c is associated with dyslipidemia in patients with type-2 diabetes mellitus in addition to as glycemic control parameter. So, proper monitoring with good glycemic control of diabetes mellitus improves the serum lipid profile and thus might play an important role in reducing the risk of cardiovascular diseases in these patients.

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Conflict of interest

The authors declared that they have no conflict of interests.

References

1. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2013; 36 Suppl 1(Suppl 1): S67-74. doi: 10.2337/dc13-S067.
2. Tripathi BK, Srivastava AK. Diabetes mellitus: complications and therapeutics. Med Sci Monit. 2006; 12 (7): RA130-47.
3. Wu Y, Ding Y, Tanaka Y, Zhang W. Risk factors contributing to type-2 diabetes and recent advances in treatment and prevention. Int J Med Sci. 2014 Sep 6;11 (11): 1185-200. doi: 10.7150/ijms.10001.
4. Turner RC, Millns H, Neil HA, Stratton IM, Manley SE, Matthews DR, et al. Risk factors for coronary artery disease in non-insulin dependent diabetes mellitus: United Kingdom Prospective Diabetes Study (UKPDS: 23). BMJ. 1998; 316 (7134): 823-8. doi: 10.1136 /bmj.316.7134.823.
5. Beckman JA, Creager M, Libby P. Diabetes and atherosclerosis, Epid, Pathophysiology and management: Review article. JAMA. 2002; 287 (19): 2570-81. doi: 10.1001/jama.287.19. 2570.
6. Taskinen MR. Diabetic dyslipidaemia: from basic research to clinical practice. Diabetologia. 2003; 46 (6): 733-49. doi: 10.1007/s00125-003-1111-y.
7. Haffner SM, Stern MP, Hazuda HP, Mitchell BD, Patterson JK. Cardiovascular risk factors in confirmed prediabetic individuals. Does the clock for coronary heart disease start ticking before the onset of clinical diabetes? JAMA. 1990; 263 (21): 2893-8. doi: 10.1001/jama.263. 21.2893.
8. Adiels M, Olofsson SO, Taskinen MR, Borén J. Over production of very low-density lipoproteins is the hallmark of the dyslipidemia in the metabolic syndrome. Arterioscler Thromb Vasc Biol. 2008; 28 (7): 1225-36. doi: 10.1161/ATVBAHA.107.160192.
9. Verges B. Pathophysiology of diabetic dyslipidaemia: where are we? Diabetologia. 2015; 58 (5): 886-99. doi: 10.1007/s00125-015-3525-8.
10. Vergès B. Abnormalities in lipoprotein kinetics in Type 2 Diabetes. Clin Lipidol. 2010; 5 (2): 277-89. doi: https://doi.org/10.2217/clp.10.2.
11. Awadalla H, Noor SK, Elmadhoun WM, Bushara SO, Almobarak AO, Sulaiman AA, et al. Comparison of serum lipid profile in type 2 diabetes with and without adequate diabetes control in Sudanese population in north of Sudan. Diabetes Metab Syndr. 2018; 12 (6): 961-4. doi: 10.1016/j.dsx.2018.06.004.
12. Sirsikar M, Supriya, Mohanty S, Pinnelli VBK. Role of glycated hemoglobin (HBA1c) as a dual marker to predict glycemic status and dyslipidemia in type II diabetes mellitus. Int J Res Med Sci. 2016; 4 (10): 4524-49. doi: https://doi.org/10.18203/2320-6012.ijrms2016 3322.
13. Toto RD. Heart disease in diabetic patients. Semin Nephrol. 2005; 25 (6): 372-8. doi: 10. 1016/j.semnephrol.2005.05.005.
14. Aamir AH, Ul-Haq Z, Mahar SA, Qureshi FM, Ahmad I, JawaA, et al. Diabetes Prevalence Survey of Pakistan (DPS-PAK): prevalence of type 2 diabetes mellitus and prediabetes using HbA1c: a population-based survey from

Pakistan. BMJ Open. 2019; 9 (2): e025300. doi: 10.1136/bmjopen-2018-025300.
15. Saleh F, Mumu SJ, Ara F, Begum HA, Ali L. Knowledge and self-care practices regarding diabetes among newly diagnosed type 2 diabetic in Bangladesh: a cross-sectional study. BMC public health. 2012; 12 (1): 1-8. doi: https://doi.org/10.1186/1471-2458-12-1112.
16. Rohlfing CL, Wiedmeyer HM, Little RR, England JD, Tennill A, Goldstein DE. Defining the relationship between plasma glucose and HbA1c: analysis of glucose profiles and HbA1c in the Diabetes Control and Com-plications trial. Diabetes Care. 2002; 25 (2): 275-8. doi: 10.2337/diacare.25.2.275.
17. Selvin E, Marinopoulos S, Berkenblit G, Rami T, Brancati FL, Powe NR, et al. Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. Ann Intern Med. 2004; 141 (6): 421-31. doi: 10.7326/0003-4819-141-6-200409210-00007.
18. Tabazzum R, Mia AR, Haq RU, Epsi EZ. Study on Glycaemic Status and Lipid Profile in Type 2 Diabetic Patients Attending Mymensingh Medical College Hospital, Mymensingh. Mymensingh Med J. 2016; 25 (3): 438-44.
19. Panjeta E, Jadrić R, Panjeta M, Ćorić J, Dervišević A. Correlation of serum lipid profile and glycemic control parameters in patients with type 2 diabetes mellitus. J Health Sci. 2018: 8 (2): 110-6. doi: https://doi.org/10.17532/jhsci.2018.488.
20. Hasan MA, Chowdhury MFK, Yasmin S, Paul S, Ahmed T, Monira S, et al. Association between Glycemic Control and Serum Lipid Profile in type 2 Diabetic Patients: Experience in a Medical College Hospital. Bangabandhu Sheikh Mujib Med Univ J. 2021; 14 (4): 138-43. doi: https://doi.org/10.3329/ bsmmuj.v 14i4.56613.
21. Khan HA. Clinical significance of HbA1c as a marker of circulating lipids in male and female type 2 diabetic patients. Acta Diabetol. 2007; 44 (4): 193-200. doi: 10.1007/s00592-007-0003-x.

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