LIPID PROFILE OF TYPE 2 DIABETES PATIENTS ATTENDING AN URBAN HEALTH CENTRE IN GOA, INDIA

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ABSTRACT

The Global Diabetes Report 2016 states, "Globally, an estimated 422 million adults were living with diabetes in 2014 compared to 108 million in 1980. Diabetes is especially a significant secondary cause for the development of dyslipidaemia. Diabetes patients are prone to have an atherogenic mixture of high Triglycerides (TGs); high, small, dense Low-Density Lipoprotein (LDL) fractions; and low High-Density Lipoprotein (HDL). This study is aimed at finding the prevalence of dyslipidaemia among type 2 diabetes patients residing in a suburban area of Goa.

METHODOLOGY

A record-based cross-sectional study design was employed to find the proportion of dyslipidaemia prevalent among the type 2 diabetes patients attending OPD of the Urban Health and Training Centre (UHTC) at Santa Cruz in Goa. A sample of 100 type 2 DM patient records were randomly selected to capture the information of the type 2 DM patients. Patient records of fasting and postprandial blood sugars and fasting lipid parameters were utilised for this study. SPSS version 23 was utilised for statistical analysis; proportions, and chi-square test were used for analysis.

RESULTS

The overall prevalence of dyslipidaemia was 85% among the type 2 DM cases. Prevalence of hyperlipidaemia among females was high (88.7%) as compared to males (78.9%). The mean level of total cholesterol was 188.78±38.25 mg/dL. Mean HDL-C level was 48.5±14.06 mg/dL. Mean value of LDL-C was 109.93±35.67 mg/dL. Mean level of LDL-C was 137.67±59.77 mg/dL.

CONCLUSION

The type 2 diabetes patients have very high prevalence of dyslipidaemia and should be monitored on regular basis to prevent cardiovascular events.

KEYWORDS

Type 2 Diabetes Mellitus, Dyslipidaemia, Suburban Population.

HOW TO CITE THIS ARTICLE: Dhupdale NY, Quadros N, Shiolkar S, et al. Lipid profile of type 2 diabetes patients attending an urban health centre in Goa, India. J. Evolution Med. Dent. Sci. 2016;5(62):4374-4377, DOI: 10.14260/jemds/2016/998

INTRODUCTION

The World Health Organisation (WHO) defines diabetes as "a serious, chronic disease that occurs either when the pancreas does not produce enough insulin (A hormone that regulates blood sugar or glucose), or when the body cannot effectively use the insulin it produces."¹

The Global Diabetes Report 2016 states "Globally, an estimated 422 million adults were living with diabetes in 2014 compared to 108 million in 1980. The global prevalence (Age-standardised) of diabetes has nearly doubled since 1980 rising from 4.7% to 8.5% in the adult population." This is a reflection of rise in the obesity. In the preceding decade, diabetes prevalence has grown faster in low and middle income countries as compared to high-income countries.¹

Financial or Other, Competing Interest: None. Submission 11-06-2016, Peer Review 21-07-2016, Acceptance 27-07-2016, Published 04-08-2016. Corresponding Author: Dr. Nitin Y. Dhupdale, Lecturer, Department of Preventive and Social Medicine, Goa Medical College, Bambolim-403202, Goa, India. E-mail: nydhupdale@gmail.com DOI: 10.14260/jemds/2016/998 Diabetes is estimated to have caused 1.5 million deaths in the year 2012. Also, more than optimal blood glucose levels have caused an additional 2.2 million deaths by progressively increasing the risks of cardiovascular and other diseases. About, 43% percent of these 3.7 million deaths occur under the age of 70 years. The proportion of deaths attributable to diabetes occurring before the age 70 is more in low and middle-income countries in comparison to high-income countries." ¹

As per Global Diabetes Country Profiles for the year 2016; the overall prevalence of diabetes in India is 7.8% (males 7.9%, females 7.5%). Diabetes has caused 75,900 deaths among males and 51,700 deaths among females in ages 30-69 years; whereas in ages 70 years and above, 46,800 of males and 45,600 of females died.⁽¹⁾ In one study conducted by Vaz NC et al, the overall prevalence of diabetes in rural population of Goa was 10.3%.²

In view of such alarmingly rising magnitude of diabetes pandemic, the WHO has declared "Beat Diabetes" as a theme for the World Health Day 2016 thereby encouraging the entire world to focus attention to combat diabetes and its complications.

The Merck manual defines dyslipidaemia as elevation of plasma cholesterol, triglycerides (TGs), or both, or a low highdensity lipoprotein level that contributes to the development of atherosclerosis;³ the atherosclerosis leads to cardiovascular events. Diabetes is especially a significant secondary cause for the development of dyslipidaemia. Diabetes patients are prone to have an atherogenic mixture of high TGs; high small, dense LDL fractions; and low HDL.

Patients with type 2 diabetes are especially at risk. The atherogenic combination is presumed due to obesity, poor control of diabetes, or both causing circulating Free Fatty Acids (FFAs) leading to increased hepatic Very-Low-Density Lipoprotein (VLDL) production. TG-rich VLDL then transfers TG and cholesterol to LDL and HDL promoting formation of TG-rich, small, dense LDL, and clearance of TG-rich HDL. Increased caloric intake and physical inactivity is a typical characteristic lifestyle of some patients with type 2 diabetes, which is favourable for the development of diabetic dyslipidaemia. Women with diabetes maybe at special risk of cardiac disease from dyslipidaemia.(3) In one study conducted by Vinoth M et al in the rural Goa, the proportion of dyslipidaemia among the diabetes patients was 93.7% as per ATP III criteria.^{4,5}

OBJECTIVES

The objectives of this study are, 1) to study proportion of dyslipidaemia and 2) to study types of lipid abnormalities prevalent in type 2 diabetes patients attending an Urban Health Centre in Goa. The study findings will help the programme managers and policy makers to understand the magnitude of dyslipidaemia among the type 2 diabetes and accordingly formulate strategies to combat lipid abnormalities to prevent early deaths.

METHODOLOGY

A record-based cross-sectional study design was employed to find the proportion of dyslipidaemia prevalent among the type 2 diabetes patients attending OPD of the Urban Health and Training Centre (UHTC) at Santa Cruz in Goa, which is a field practice area under Department of Preventive and Social Medicine of Goa Medical College at Bambolim - Goa. A purposive sample of 100 type 2 DM patient records were randomly selected to capture the information of the type 2 DM patients. A verbal consent was obtained from the patients to access their personal file records maintained at the Health Centre. The study was approved by the Institutional Ethics Committee. The blood of the patient was collected at UHTC Santa Cruz and transported to the Central Laboratory at Goa Medical College Hospital, Bambolim, for estimation of various blood parameters. Following laboratory procedures are used for the estimation of various blood parameters. Fasting lipid profile [serum cholesterol (TC), Triglyceride (TG), Highdensity lipoprotein cholesterol (HDL-C)] using automated method based on principle of chemiluminescence, low density lipoprotein cholesterol (LDL-C) was calculated by using Friedewald's formula [LDL-C (mg/dL) = TC (mg/dL) - HDL-C (mg/dL) - TG (mg/dL)/5].6

Fasting blood sugar level of all patients was measured using glucose oxidase enzymatic method.

Patients having one or more parameters outside the target levels recommended by American Diabetes Association were considered to have dyslipidaemia and uncontrolled diabetes.⁷ Diabetes is diagnosed at Fasting Blood Sugar Level (FBSL) of greater than or equal to 126 mg/dL and 2-hour Postprandial Blood Sugar Level (PPBSL) of greater than or equal to 200 mg/dL. 7

The data was compiled using licensed software IBM SPSS version 23 (Customer ID 227011) and proportions and chisquare test were utilised for analysis, p<0.05 was used as level of significance.

Lipid	Optimal/Near Optimal Concentration	Borderline Serum Concentration	High risk/Very High Risk Serum Concentration
LDL-C	<100 optimal (100-129 near optimal)	130-159	160-189 high ≥190 very high
Total cholesterol	<200	200-239	≥240
HDL-C	≥60	40-59 (men) 50-59 (women)	<40 men <50 women
TG	<150	150-199	200-499 high ≥500 very high
Table 1: ATP III Classification of LDL-C, TC, HDL-C, and TG (mg/dL)(5)			

The normal values of fasting lipids were taken as TC desirable <200 mg/dL, TG <150 mg/dL, HDL-C >40 mg/dL, LDL-C near optimal <130 mg/dL.

OBSERVATIONS

Out of total 100 type 2 diabetes patients studied, 62 were females and 38 were males. The mean age was 59.58 ± 9.7 years. Overall prevalence of dyslipidaemia among the type 2 diabetes patients was 85% (i.e., raised levels of plasma cholesterol, triglycerides (TG), or both, or a low high-density lipoprotein level.) (Table 1).

Gender	Dyslipidaemia		Total	
	Yes (%)*	No (%)*	(%)*	
Female	55 (88.7)	7 (11.3)	62 (100.0)	
Male	30 (78.9)	8 (21.1)	38 (100.0)	
Total	85 (85.0)	15 (15.0)	100 (100.0)	
Table 2: Gender-Wise Distribution of Dyslipidaemia Among Study Subjects				

*figures in parenthesis are percentages. Pearson chi-square = 1.761, df = 1, p = 0.184.

Prevalence of hyperlipidaemia among females was high (88.7%) as compared to males (78.9%). (Table 2). The gender was not found to be statistically associated with the dyslipidaemia (P=0.184). The higher prevalence of dyslipidaemia among the female gender may be due to higher representation by the female patients in the study.

Type of Lipid	Mean±S.D. (mg/dL)	
Total Cholesterol	188.78±38.25	
HDL-C	48.5±14.06	
LDL-C	109.93±35.67	
TG	137.67±59.77	
Table 3: Mean Values of Different Types Lipids Among Study Participants		

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The mean values of each type of cholesterol are mentioned in Table 3. The prevalence of very low HDL-C (that is <40 mg/dL in men and <50 mg/dL in women) was 51% as seen from the Table 4. Very high levels of LDL-C were observed among 9% of the study participants. (Table 4).

	Plasr			
Type of Lipid	Optimal (%)*	Borderline (%)*	High Risk (%)*	Total (%)*
Total	65	25 (25.0)	10	100
cholesterol	(65.0)	23 (23.0)	(10.0)	(100.0)
HDL-C	17	32 (32.0)	51	100
IIDL-C	(17.0)		(51.0)	(100.0)
LDL-C	69 (69.0)	22 (22.0)	High-7; very high-2 (9.0)	100 (100.0)
TG	71 (71.0)	15 (15.0)	14 (14.0)	100 (100.0)
Table 4: Distribution of Study Subjects According to				
Level of Lipid Profile				

*figures in parenthesis are percentages.

High Risk	Male	Female	Total
Dyslipidaemia	(%)*	(%)*	(%)*
High cholesterol	5 (50.0)	5 (50.0)	10 (100.0)
Low HDL	14 (27.5)	37 (72.5)	51 (100.0)
High LDL	6 (66.7)	3 (33.3)	9 (100.0)
High TG	5 (35.7)	9 (64.3)	14 (100.0)
Table 5: Gender-Wise Distribution of High Risk			
Dyslipidaemia			

*figures in parenthesis are percentages.

The prevalence of very low levels of HDL-C was observed more among women than men. (Table 5).

FBSL	Dyslipidaemia		Total	
гдэг	Yes (%)	No (%)	(%)	
Controlled	44 (88.0)	6 (12.0)	50 (100.0)	
Uncontrolled	41 (82.0)	9 (18.0)	50 (100.0)	
Total	85 (85.0)	15 (15.0)	100 (100.0)	
Table 6: Fastina Sugar (FBSL) and Dyslipidaemia				

Pearson chi-square = 0.706; df = 1; p = 0.401.

PPBSL	Dyslipidaemia		Total (%)	
FFDSL	Yes (%)	No (%)	10tal (70)	
Normal	10 (66.7)	5 (33.3)	15 (100.0)	
Controlled	21 (84.0)	4 (16.0)	25 (100.0)	
Uncontrolled	54 (90.0)	6 (10.0)	60 (100.0)	
Total	85 (85.0)	15 (15.0)	100 (100.0)	
Table 7: Postprandial Sugar Level (PPBSL) and				
Dyslipidaemia				

Pearson Chi-square = 5.156; df = 2; p = 0.076.

The prevalence of hyperlipidaemia was almost similar among the controlled and uncontrolled type 2 DM cases (Table 6 and 7). Total of 59 subjects had high TG/HDL (>2.4) ratio.

DISCUSSION

Out of the total 100 type 2 DM patients studied, the prevalence of dyslipidaemia was 85% as compared to 8% reported by the Agarwal Y et al⁸ and 92.4% reported by Jayarama N et al⁹ whereas Vinoth M⁴ et al reported 97.3%. In our study, the prevalence of hyperlipidaemia among females was 88.7% and males was 78.9% as compared to 95.4% (males) and 86.75% (females) reported by Jayarama N et al.⁹ There is no statistically significant association between gender and dyslipidaemia. The high level of dyslipidaemia maybe attributed to the urbanisation and the westernisation of diet.

In our study, the mean TC value was 188.78±38.25 mg/dL as compared to 178±5.27 mg/dL reported by Agarwal Y. et al⁸ Smith S et al¹⁰ reported as 299.361±13.461 mg/dL and Singh G et al¹¹ reported 203.9±15.8 mg/dL. The mean value of serum TG in our study was 137.67±59.77 mg/dL as compared to the 186.97±9.28 mg/dL reported by Agarwal Y. et al⁸ 199.4± 157.21 mg/dL reported by Jayarama N et al⁹ 178.32±10.12 mg/dL reported by Smith S et al¹⁰ and Sigh G et al¹¹ reported 151.1±17.7 mg/dL.

The mean LDL-C levels observed in our study are 109.93 \pm 35.67 mg/L as compared to 102.78 \pm 3.94 mg/dL reported by Agarwal Y et al⁸ 113.3 \pm 57.89 mg/dL reported by Jayarama N et al⁹ 164.15 \pm 5.98 mg/dL reported by Smith S et al¹⁰ and Singh G et al¹¹ reported 124.4 \pm 11.9 mg/dL. The mean HDL-C levels observed in our study are 48.5 \pm 14.06 mg/dL as compared to 41.34 \pm 3.10 mg/dL reported by Agarwal Y et al⁸ and 46.8 \pm 72.88 mg/dL as reported by Jayarama N et al⁹ Smith S et al¹⁰ reported 31.12 \pm 1.10 mg/dL and Singh G et al¹¹ reported 47.7 \pm 6.2 mg/dL.

In our study, total of 38 individuals had TC levels more than 200 mg/dL. These patients are in need of medical diet therapy, which is recommended by the ATP-III including weight management and increased physical activity after cardiac evaluation: Saturated fat <7% of calories, cholesterol <200 mg/day, consider increased viscous (soluble) fibre (10-25 g/day) and plant stanols/sterols (2 g/day) as therapeutic options to enhance LDL lowering.⁵

In our study, 31 type 2 diabetes patients had increased levels of TG >200 mg/dL. These patients are to be managed as per the guidelines of American Diabetes Association. Treatment of elevated triglycerides (\geq 150 mg/dL): Primary aim of therapy is to reach LDL goal of <130 mg/dL, intensify weight management, increase physical activity. If triglycerides are >200 mg/dL after LDL goal is reached, set secondary goal for non-HDL cholesterol (total-HDL) 30 mg/dL higher than LDL goal.⁵

If TG levels are 200-499 mg/dL after LDL goal of <130 mg/dL is reached, consider adding drug if needed to reach non-HDL goal of <160 mg/dL (i.e. TC-HDL): Intensify therapy with LDL-lowering drug, or add nicotinic acid or fibrate to further lower VLDL. If TG >500 mg/dL, first lower triglycerides to prevent pancreatitis: Very low-fat diet (<15% of calories from fat), weight management and physical activity, fibrate or nicotinic acid. If TG <500 mg/dL then switch to LDL-lowering therapy.⁵

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CONCLUSIONS

The prevalence of dyslipidaemia among type II diabetes is very high. The female patients were having higher prevalence compared to males. The prevalence of high risk dyslipidaemia was very high, which increases their risk of developing cardiovascular events in future. The proportion of type 2 DM patients having elevated levels of specific atherogenic cholesterol carrying lipoproteins was observed.

STRENGTH

This study is the only kind of study performed on the suburban population of type 2 diabetes population and it will be helpful to document the baseline levels of dyslipidaemia prevalent among the type 2 DM patients residing in the suburbanised areas of Goa.

LIMITATIONS

The small sample size of the study population may have resulted in many findings being reported as statistically insignificant. The patients sampled are the ones who are utilising this healthcare facility and those who are not utilising are missed out. This limitation will affect the generalisability of the findings. The study findings could not be correlated to the HBA1C levels due to unavailability of these records at the time of study. The current ongoing treatment details were also could not be correlated due to paucity of records. The BMI, hypertension, and many other variables of the patients could not be correlated to the prevalence of dyslipidaemia.

RECOMMENDATIONS

All the type 2 patients should be strictly monitored for the associated dyslipidaemia by subjecting the patients for a regular blood testing. The patients with elevated levels of lipids should be initiated on the medical nutrition therapy followed by medical line of treatment if the target goals are not achieved. These patients should be advised regarding weight management by increasing the level of physical activity. These patients are also recommended a thorough clinical correlation to assess any coexisting IHD changes by subjecting patients to cardiac evaluations.

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