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Research Article

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Assessment of Lipid Profile among Type II Diabetes Mellitus Patients with and Without Hypothyroidism

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ABSTRACT

Background: About 95% of people with diabetes mellitus (DM) have dyslipidaemia. Dyslipidaemia is one of the major risk factors for coronary heart disease (CHD). Due to disturbances in lipoprotein levels, cardiovascular disease contributes to morbidity and mortality in DM patients. To examine the effects of lipid profiles in Type II DM patients with or without hypothyroidism.
Methods: A case-control study was conducted in the Department of Medicine, Santosh Medical College and Hospital, Ghaziabad, for one year from 1st Oct. 2017 to 31st Sept. 2018 on patients attending the Medicine OPD of Santosh Medical College and Hospital. There were three groups of 25 patients compared with the single group of control, which consisted of 25 patients.
Results: Mean value of triglycerides, low-density lipoprotein (LDL), total cholesterol, and lipoprotein A in Group DM, DM+Hypothyroidism, DM+subclinical hypothyroidism (DM+SCH) was significantly higher than the control group ($p<0.00$). Mean high-density lipoprotein (HDL) in Group DM, DM+Hypothyroidism, DM+SCH, was significantly lower than the control group ($p<0.001$). The mean HDL in Group DM+Hypothyroidism was significantly lower than Group DM ($p<0.001$).
Conclusion: Dyslipidaemia in the group of overt hypothyroid diabetics is significantly higher than in the Group of subclinical hypothyroid diabetics. So, it is suggested that diabetic patients with hypothyroidism (both overt and subclinical) should undergo annual lipid status examination and corrective management as and when needed.

Key-words: Cholesterol, Diabetes mellitus, Lipoprotein A, Low-density lipoprotein, Triglycerides

INTRODUCTION

One of the most prevalent types of metabolic diseases is diabetes mellitus (DM), defined by elevated blood glucose levels caused by defects in insulin action, insulin secretion, or both [1].

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Globally, the prevalence of DM is rapidly rising and reaching epidemic proportions. By 2010, 285 million individuals worldwide were expected to be suffering from diabetes (6.4% prevalence), and by 2030, that number is expected to grow to 439 million people (7.7% prevalence) [2]. India now has more than three crore patients who have diabetes, making it the global "diabetes capital" [3]. Globally, coronary artery disease, particularly myocardial infarction, is the primary cause of illness and death [4]. Hyperglycemia and atherosclerosis are associated with type-2 diabetes [5]. The prevalence of dyslipidaemia in DM is 95% [6]. Dyslipidemia is one of the

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foremost risk factors for coronary heart disease (CHD) [7]. The patients with DM are more likely to suffer from cardiovascular disease due to disturbances in lipoprotein levels, namely serum triglycerides (TC) of 69%, serum cholesterol of 56.6%, LDL of 77%, and HDL of 71% [8,9].

Currently, however, this collection of diseases is referred to as "Metabolic Syndrome" by the International Diabetes Federation (IDF) and the World Health Organisation (WHO) [10]. A combination of increased TCs decreased HDL, and an excess of small, dense LDL particles is known as diabetic dyslipidaemia. DM frequently results in lipid abnormalities as insulin resistance or insufficiency affects important lipid metabolism pathways and enzymes [11]. The term "hypothyroidism" refers to a lack of thyroid function, which, independent of the reason, is caused by reduced production of both T3 and T4 [12].

Among endocrine illnesses, it is the most prevalent pathologic hormone deficiency. Hypothyroidism may result from a pituitary thyroid-stimulating hormone (TSH) deficiency or a primary thyroid gland disease [13]. Pituitary TSH hypersecretion and an enhanced increase in serum TSH levels decrease T4 and T3 concentration. This is an important laboratory result, especially for the early diagnosis of thyroid deficiency [14].

Clinical hypothyroidism can cause a wide range of signs and symptoms that affect the body's major systems, including the cardiovascular, musculoskeletal, central nervous, reproductive, gastrointestinal, and endocrine systems [15]. Thyroid hormones majorly impact the production, metabolism, and mobilisation of fats. Coronary artery disease is linked to overt hypothyroidism and a marked rise in total LDL-cholesterol levels in the blood. A hormone deficiency and low lipoprotein lipase activity promote hypercholesterolemia [16,17].

As there is not much data available regarding the effects on lipid profile in patients with hypothyroidism in Type II DM in local population, there is a need to carry out such a study in this population. So, the scope of the present study was to assess the effects of lipid profiles in patients with or without hypothyroidism in Type II Diabetes mellitus so that early screening and prompt treatment of these patients can be done to avoid serious complications.

MATERIALS AND METHODS

It was a case-control study conducted in the Department of Medicine, Santosh Medical College and Hospital, Ghaziabad, India, for one year from 1st Oct. 2017 to 31st Sept. 2018 on patients attending the Medicine OPD of Santosh Medical College and Hospital.

Sample Size- There were three group of 25 patients compared to the single group of control, which consisted of 25 patients.

Cases- All diabetic patients attending the Medicine OPD and IPD aged 25-65 years were subjected to physical examination, history, anthropometry, thyroid profile, and routine investigations. After assessment, they were divided into three sex- and age-matched groups as follows:

Group 1 comprises age- and sex-matched confirmed 25 cases of Type II DM.

Group 2- It comprises age- and sex-matched confirmed 25 cases of Type II DM with SCH.

Group 3- It comprises age- and sex-matched confirmed 25 cases of Type II DM with overt hypothyroidism.

Control Group- The present study included 25 healthy subjects matched with age and sex as controls in the age group of 25-75 years.

Inclusion Criteria

The American Diabetic Association defines the following criteria for diagnosis of DM:

- HbA1C>6.5%, fasting plasma glucose≥126 mg/dl (7.0 mmol/L), 2-hour plasma glucose≥ 200 mg/dl (11.1 mmol/L) during an oral glucose tolerance test, or random plasma ≥ 200 mg/dl (11.1 mmol/L).
- SCH, that is, without usual symptoms of hypothyroidism, normal circulating thyroid hormone (T3 and T4), and elevated TSH (>5-10 µU/ml).
- Overt hypothyroidism is accompanied by a clinical hypothyroidism syndrome and an increased TSH (>10 mU/L).

Exclusion Criteria

- Patients with hepatic dysfunction-hepatitis and cholestasis, disease-nephrotic syndrome, chronic renal insufficiency, and acute illness (sepsis, severe heart failure, acute myocardial infarction, recent admission in intensive care unit).

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- Patients with gestational diabetes.
- Individuals receive medication that interferes with thyroid function (amiorone, corticosteroids, propranolol, and oral contraceptives).
- Patients having a history of total/subtotal thyroidectomy.
- Patients on I131 treatment, lithium, antithyroid drugs.
- Diagnosed cases of Grave's disease, toxic multinodular goitre, toxic adenoma, gestational hyperthyroidism patients, and carcinoma patients.
- Patients with history of chronic renal failure or radiation exposure.
- Patients with known liver, kidney or other acute and chronic diseases such as tuberculosis, etc.

Data Collection- All control and patients underwent anthropometric measurements routine and special investigations. Special investigation includes glycosylated haemoglobin, T3, T4, TSH and lipid profile (TG, HDLC, LDLC, VLDLC, Lipoprotein A).

Ethical Consideration- The Institutional Ethical & Review Committee of Santosh Medical College and Hospital, Ghaziabad, Uttar Pradesh, granted ethical approval. The current investigation complied with the Declaration of Helsinki of the World Medical Association (WMA). The participant's anonymity has been preserved for the duration of the investigation.

Statistical Analysis- SPSS version 23 was used as the statistical tool for tabulation and analysis of collected data. Kolmogorov Smirnov & Shapiro Wilk test of normality and descriptive statistics were employed to understand the data distribution. Continuous data were reported as mean (SD) for normally distributed continuous variables or median (Interquartile Range - 25th–75th percentile) for skewed distributions. Continuous data in four groups were compared using one-way ANOVA, depending on data distribution. Post hoc test was also performed to observe the inter-relationship between the groups. p-value less than 0.05 was considered statistically significant at 95% confidence level.

RESULTS

Tables 1–3 show that the mean value of LDL, triglycerides, and Lipoprotein A in Group DM, DM+Hypothyroidism, DM+SCH was significantly higher than in the control group (p<0.001). The mean value of triglycerides, LDL, and lipoprotein A in Group DM+Hypothroidism was significantly higher than in Group DM. The mean value of triglycerides, LDL, and lipoprotein A in Group DM+SCH was non-significantly higher than in Group DM (p=0.55). The mean value of triglycerides, LDL, and lipoprotein A in Group DM+SCH was significantly lower than in Group DM+Hypothyroidism (p<0.001).

Table 1: Mean difference of LDL among groups and Post hoc test for mean difference in each group.

Group	Mean	SD	Median	Minimum	Maximum	ANOVA test
Control (n=25)	98.12	9.69	99.00	80.00	114.00	p=<0.01 Effect size (Omega - Squared) of this One-way ANOVA test=0.939
DM (n=25)	250.08	18.39	252.60	199.23	280.38	
DM+SCH (n=25)	257.06	15.82	256.83	229.11	287.50	
DM+Hypothyroid (n=25)	278.11	26.60	272.75	229.96	334.51	
Post-Hoc Test						
Comparison	**Mean Difference (95% CI)**			**Unpaired t-test (p-value)**		
DM–Control	151.95 (138.17-165.74)			<0.001		
DM+Hypothyroid–Control	179.98 (166.3-193.77)			<0.001		
DM+SCH–Control	158.94 (145.17-172.73)			<0.001		
DM+Hypothyroid–DM	28.04 (14.27-41.85)			<0.001		
DM+SCH–DM	6.99 (-6.7-20.78)			0.55		
DM+SCH–DM+Hypothyroid	-21.05 (-34.82-7.25)			<0.001		

DM: diabetes mellitus; SCH: subclinical hypothyroidism

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Table 2: Mean difference of triglyceride among groups and Post-hoc test for mean difference in each group.

Group	Mean	SD	Median	Minimum	Maximum	ANOVA test
Control (n=25)	142.08	11.068	143	118	166	p=<0.01 Effect size (Omega - Squared) of this One-way ANOVA test=0.866
DM (n=25)	208.96	11.145	208	187	239	
DM+SCH (n=25)	213.24	12.296	216	194	235	
DM+Hypothyroid (n=25)	234.48	19.380	232	204	269	
Post-Hoc Test						
Comparison	**Mean Difference (95% CI)**			**Unpaired t-test (p-value)**		
DM–Control	66.87 (56.7-77.15)			<0.001		
DM+Hypothyroid–Control	92.41 (82.13-102.66)			<0.001		
DM+SCH–Control	71.17 (60.89-81.45)			<0.001		
DM+Hypothyroid–DM	25.53 (15.23-35.7)			<0.001		
DM+SCH–DM	4.29 (-6-14.55)			0.7		
DM+SCH–DM+Hypothyroid	-21.25 (-31.53-10.95)			<0.001		

DM: diabetes mellitus; SCH: subclinical hypothyroidism

Table 3: Mean difference of Lipoprotein A among groups and post hoc test for mean difference in each group.

Group	Mean	SD	Median	Minimum	Maximum	ANOVA test
Control (n=25)	25	15.299	4.907	14.91	6.167	p=<0.01 Effect size (Omega - Squared) of this One-way ANOVA test=0.415
DM (n=25)	25	18.800	5.781	20.00	5.000	
DM+SCH (n=25)	25	24.600	8.201	24.00	8.000	
DM+Hypothyroid (n=25)	25	34.640	8.597	35.00	14.000	
Post-Hoc Test						
Comparison	**Mean Difference (95% CI)**			**Unpaired t-test (p-value)**		
DM–Control	3.51 (-1.72 to 8.73)			0.3		
DM+Hypothyroid–Control	19.35 (14.14 to 24.56)			<0.001		
DM+SCH–Control	9.31 (4.08 to 14.52)			<0.001		
DM+Hypothyroidism–DM	15.85 (10.62 to 21.04)			<0.001		
DM+SCH–DM	5.81 (0.58 to 11.02)			0.02		
DM+SCH–DM+Hypothyroid	-10.05 (-15.26 to -4.84)			<0.001		

DM: diabetes mellitus; SCH: subclinical hypothyroidism

In Table 4, the mean HDL in Group DM, DM+Hypothyroidism, DM+SCH, was significantly lower than control Group (p<0.001). The mean HDL in Group DM+Hypothyroidism was significantly lower than in Group DM (p<0.001). The mean HDL in Group DM+SCH was non-significantly lower than in Group DM (p=0.43). The mean HDL in Group DM+SCH was significantly lower than in Group DM+Hypothyroidism (p<0.001).

Table 4: Mean difference of HDL among groups and Post-hoc test for mean difference in each group.

Group	Mean	SD	Median	Minimum	Maximum	ANOVA test
Control (n=25)	142.08	11.068	143	118	166	p=<0.01 Effect size (Omega - Squared) of this One-way ANOVA test= 0.866
DM (n=25)	208.96	11.145	208	187	239	
DM+SCH (n=25)	213.24	12.296	216	194	235	
DM+Hypothyroidism (n=25)	234.48	19.380	232	204	269	
Post-Hoc Test						

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Comparison	Mean Difference (95% CI)	Unpaired t-test (p-value)
DM–Control	-7.61 (-11.45 to -3.76)	<0.001
DM+Hypothyroid–Control	-18.81 (-22.65 to -14.93)	<0.001
DM+SCH–Control	-9.85 (-13.6 to -5.96)	<0.001
DM+Hypothyroidism–DM	-11.21 (-15.05 to -7.33)	<0.001
DM+SCH–DM	-2.26 (-6.1 to 1.63)	0.43
DM+SCH–	-7.61 (5.2 to 12.83)	<0.001

DM: diabetes mellitus; SCH: subclinical hypothyroidism

DISCUSSION

The mean LDL level in the present study was significantly enhanced in all the subgroups compared to control. Similar results were documented by Ghosh *et al*. in their study in which serum LDL was significantly increased among diabetic hypothyroid patients as compared to normal diabetics [18]. Because of a decrease in receptor-mediated degradation of LDL and intermediate-density lipoprotein (IDL), LDL levels rise as hypothyroidism progresses. It has been demonstrated that thyroid hormones (fT3 and fT4), particularly fT3, control sterol-regulatory element-binding protein and bind to thyroid hormone-responsive elements (TREs) to modulate LDL receptors. Additionally, thyroid hormone expresses hydroxymethyl glutaryl coenzyme A reductase in the liver, increasing cholesterol synthesis.

Additionally, thyroid hormone expresses hydroxymethyl glutaryl coenzyme A reductase in the liver, increasing cholesterol synthesis. Thus, lower thyroid hormones result in lower expression of LDL receptors and lower hepatic cholesterol synthesis. These reductions may also lower catabolism and cellular absorption of circulating LDL-C, ultimately leading to higher levels of LDL in the blood.

Similar to the findings of our study, the mean HDL level was significantly lower than that of the other four subgroups. According to Shashi *et al*. there was a substantial (p<0.001) drop in the mean serum levels of HDL between the non-diabetic controls and the SCH and clinical hypothyroid diabetes patients [19]. When comparing SCHI and clinical hypothyroid diabetics, a substantial (p<0.05) decline in HDL (mean difference 11.87-20.62) was found using post hoc Tukey's multiple comparison test following a one-way ANOVA. In clinical versus SCH, HDL concentration showed a significant decrease (95%CI -11.27 to -6.23) with a mean difference of -8.747. In the study by Ghosh *et al*. serum HDL was significantly decreased (p<0.001) among the diabetic hypothyroid patients as compared to normal diabetics [18]. In a study by Nirmala *et al*., 46.6% of individuals with thyroid dysfunction had poor HDL cholesterol. Only 13.6% of the patients in the group without thyroid disease had poor HDL [20]. Type 2 diabetes patients have lower HDL cholesterol because of an enhanced HDL catabolism. The observed increase in HDL catabolism is probably due to increased hepatic lipase activity—the enzyme that controls HDL catabolism—in insulin-resistant conditions.

In a study by Ghosh *et al.* serum TCs were significantly higher (p-0.03) among diabetic hypothyroid patients as compared to normal diabetics [18]. Comparable results were observed in the present work.

In a study by Shashi *et al*. the mean serum level of total cholesterol was considerably (p<0.0001) higher in individuals with hypothyroidism who were clinically and sub-clinically diabetic than in non-diabetic controls [19]. Following a one-way ANOVA, a post hoc Tukey's multiple comparison test revealed a substantial (p<0.05) rise in the serum total cholesterol in diabetes individuals. The overall cholesterol level significantly increased in clinical and SCH patients compared to controls (95% CI -50.83 to -48.00 mean difference -43.88 to -54.84). Additionally, there was a significant mean difference of 10.96 (p<0.05, q=4.748, 95% CI 3.218 to 18.71) between SCH and clinical hypothyroid diabetes patients.

In a study by Shashi *et al*. patients with SCH and clinical hypothyroidism had considerably (p<0.002) higher serum VLDL concentrations than those without diabetes [19].

Following a one-way ANOVA, a post hoc Tukey's multiple comparison test revealed a substantial (p<0.05) rise in serum VLDL. Comparing the VLDL levels to the control, there was a substantial rise in both SCH and clinical hypothyroidism (mean difference -11.810 to -14.280). There was a significant (95% CI 1.344 to 3.605, mean difference of 2.474) elevation in SCH versus clinical hypothyroid diabetics. Hypothyroidism also affects lipoprotein transit and composition. Modifications in

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thyroid function cause modifications in lipoprotein transport and composition. Thus, despite decreased HMG CoA reductase enzyme activity brought on by elevated LDL and IDL levels, there is a frequent increase in serum total cholesterol concentration.

CONCLUSIONS

In the present study, we concluded that dyslipidaemia in a group of overt hypothyroid diabetics is significantly higher than in the group of sub-clinically hypothyroid diabetics, Group of only diabetics, and control.

So, it is suggested that diabetic patients with hypothyroidism (both overt and subclinical) should undergo annual lipid status examination and corrective management as needed to prevent themselves from severe atherosclerotic diseases such as hypothyroidism that acts as a synergistic risk factor for dyslipidemia in diabetics.

CONTRIBUTION OF AUTHORS

Research concept- Aditya Sharma, Ashok Kumar
Research design- Aditya Sharma, Ashok Kumar.
Supervision-Ashok Kumar, Jagmohan Singh Dhakar
Materials and Data collection- Aditya Sharma, Ashok Kumar
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Final approval- Aditya Sharma, Aditya Thakur, Jagmohan Singh Dhakar, Tej Pratap Singh

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