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Assessment of newly onset thyroid dysfunction and dyslipidemia in patients of Type 2 diabetes mellitus: A prospective cross sectional study in Kumaon region of Uttrakhand

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Abstract

Introduction-Type2 Diabetes mellitus and thyroid disorders are common metabolic disorders with a highly significant global burden. The relationship between T2DM with TD is intricate and bidirectional in terms of their pathophysiology, coexistence as well as mutual aggravation.

Aim- There is a lack of sufficient literature regarding the association of TD and dyslipidemias in patients of T2DM in this kumaon area. To address these problems and to assess the magnitude of the problem this study was planned.

Material and methods- After obtaining approval from the Institutional Ethics Committee, this cross-sectional study was conducted in 150 eligible patients. Data were collected on demographic characteristics, clinical presentations, thyroid function tests, glycemic parameters, and lipid profiles. Thyroid dysfunction was categorized into subclinical hypothyroidism, overt hypothyroidism, subclinical hyperthyroidism, and overt hyperthyroidism based on TSH, T3, and T4 levels. Statistical analysis was performed to identify significant associations among variables.

Results: Hypothyroidism was the most prevalent thyroid dysfunction (43.3%), followed by subclinical hypothyroidism (34%) and hyperthyroidism (22.7%). Females constituted 78.67% of the study population. Significant correlations were observed between thyroid dysfunction and glycemic control, with higher fasting blood sugar, postprandial blood glucose, and HbA1C levels in hypothyroid patients. Lipid abnormalities, including elevated total cholesterol, triglycerides, and

LDL levels, were more pronounced in hypothyroidism. Subclinical hypothyroidism showed intermediate values, while hyperthyroidism was associated with lower lipid levels. A significant positive correlation of duration T2DM was observed with TSH and HbA1c levels.

Conclusion: The study highlights the high prevalence of thyroid dysfunction in T2DM patients, emphasizing the need for routine thyroid screening and integrated management of glycemic and lipid parameters for optimal care and to prevent complications. These findings underscore the importance of targeted interventions and awareness campaigns, especially in resource-limited settings like the Kumaon region.

Keywords: T2DM, Thyroid Disorders(TD), Dyslipidemias.

Introduction

Diabetes mellitus (DM) is a common endocrine metabolic disorder worldwide, characterized by raised blood glucose levels due to decreased insulin secretion or resistance or both[1]. T2DM has emerged as a prominent public health challenge worldwide, particularly in developing nations including India. As per the International Diabetic Federation (IDF) 2021 report, there were 74.2 million cases of DM in 2021 with prevalence of 8.7% of adult population (20-79 years) and is expected to rise to 124.5 million[2]. This pandemic rise of Type 2 DM may be due multiple interactions between dietary habits, sedentary life style, ethnicity, obesity, hypertension and genetic predisposition to the disease[3].

Diabetes can affect thyroid function in various ways. In T2DM, the response of thyroid-stimulating hormone (TSH) to thyrotropin-releasing hormone (TRH) can become impaired, leading to hypothyroidism and, subsequently, lower levels of T3. This decrease in T3 levels might also be due to the reduced conversion of T3 from T4, as evidenced by studies showing a reversible decrease in deiodinase activity and hepatic thyroxine concentration induced by hyperglycemia[4,5]. However, there are studies suggesting that a short-term excess of T3 could induce Insulin Resistance (IR), potentially contributing to the development of T2DM.[6,7]

Insulin resistance appears to be a possible connection between thyroid dysfunction and T2DM[2].Thyroid hormones exert a direct influence on insulin secretion[9]. Hypothyroidism results in a decrease in insulin production via beta cells whereas hyperthyroidism lead to an increase in beta-cell responsiveness to catecholamine or glucose due to increased beta-cell mass. Additionally, thyrotoxicosis results in an increase in insulin clearance. Beta-cell dysfunction and insulin resistance both are negatively connected to TSH, which may be explained by thyroid hormone's insulin-antagonistic properties combinedwith a rise in TSH.[10,11]

Moreover, hyperinsulinemia and Insulin resistance promote thyroid tissue proliferation, increase the prevalence of nodular thyroid disease, and result in a goiter. On the other hand, thyroid dysfunction affects glucose metabolism in diabetes.[11-13]

Earlier studies have recognized a significant impact of Thyroid Hormones (TH) along with insulin on the maintenance of glucose levels in the body as well as on the specific metabolic pathways for glucose and lipids. For instance, both triiodothyronine (T3) and insulin seem to modulate the activities of key regulatory proteins responsible for glucose uptake into cells and those crucial for the metabolic cascades of glucose and lipids[13,14].

All of these changes occur as a result of alternations in thyroid hormone which increases the risk of developing T2DM and can lead to diabetic complications or can

worsen diabetic symptoms (14). It is important to screen for TD in T2DM patients as each of these endocrinopathies and their complex interdependent interactions increase the cardiovascular risks[15,16]. Unrecognized TD can worsen glycaemic control and increase the cardiovascular risk in T2DM[13-15].

In hypothyroid patients, the rate of insulin degradation will be reduced, which in turn, may lower exogenous insulin requirement and is often accompanied by abnormalities in the metabolism of plasma lipid, which include elevation in the concentration of triglyceride, cholesterol and low-density lipoprotein (LDL)[16]. Dyslipidemias coexisting with T2DM gets exacerbated by subclinical hypothyroidism (SCH) and further increases the risk of cardiovascular diseases[17]. Hence, it is necessary to diagnose TD in T2DM patients as early as possible for the provision of effective treatment since undiagnosed TD in diabetic people can adversely affect metabolism in such patients, thereby causing an elevation in the risk of diabetic complications.[18]

There is a lack of sufficient literature in the present study area to address these problems or assess the magnitude of the problem. In light of the aforementioned issues, the present study aimed to assess the lipid profile parameters of new onset thyroid dysfunction in patient with type 2 diabetes mellitus.

Material and methods

The study was commenced after obtaining approval from the Institutional Ethics Committee. This cross-sectional study was conducted in the Department of General Medicine and Biochemistry, Government Medical College, Haldwani and associated Dr. Susheela Tiwari Hospital, Haldwani, Nainital. All patients with Type 2 diabetes mellitus attending the department of medicine, presenting in casualty, medicine OPD, and medicine IPD were screened for thyroid profile. 150 eligible patients were identified and invited to participate.Written informed consent was obtained.

Inclusion criteria encompassed known Type 2 DM and newly detected Type 2 DM according to American Diabetic Association guidelines, new onset thyroid dysfunction, dyslipidemias and willingness to participate.

Exclusion criteria included Type 1 DM, known thyroid disease, chronic renal failure, diabetic nephropathy, acute illness, hepatic dysfunction, psychiatric illness, pregnancy, and use of drugs interfering with thyroid function or antihyperlipidemic drugs. The study aimed to investigate the glycemic indices, thyroid and lipid profile of type 2 diabetes mellitus patients with new-onset thyroid dysfunctions.

Biochemical analysis

Taking all the aseptic precautions about 5 ml of blood was collected from the peripheral vein after an overnight fast of 8-10 hours. The blood was divided into two aliquots, one 3ml was divided into a fluoride vial (for FBG), EDTA vial (for Hb, HbA1c), and plain vial for estimation of lipid profile and other routine parameters (urea, creatinine, uric acid). Samples collected from study subjects were processed and analyzed on Roche / Hitachi Cobas 501c fully automated biochemistry analyzer in the Biochemistry central laboratory of STH hospital. All estimations were performed on the closed system using the manufacturer's kit.

Weight and height of all the patients were measured according to standard protocol. BMI was calculated by using the software with the formula: BMI (kg/m2) = Weight (kg)/ Height (m2). Systolic and diastolic blood pressure was measured on the right arm using a sphygmomanometer by the auscultatory method.

Results and Discussion

Table 1: Demographic characteristics of the study population (n=150)

Parameters n=150	Females n=118 (78.67%)	Males n=32 (21.33%)
Age (years) ≤ 30 31-50 ≥50	 13 (8 .66%) 81 (54 %) 24 (16%)	 4 (2.6%) 19 (12.66 %) * 9 (6%)
BMI(kg/m2)	24.57 ± 4.03	27.42 ± 7.52*
WHR	0.92±0.22	1.34 ± 0. 49*
WC	79.63 ± 1.68	82.67 ± 3.96*
BP Systolic Diastolic	 137.15 ± 8.35 84.37 ± 4.95	 139.35± 6.81 86.40± 4.72

Table 2: Age and sex wise distribution of the Type 2 Diabetes mellitus patients in thyroid dysfunction groups

Age groups	Total no. (n=150)	Hyperthyroidism n=24 (16%) Group I	Hypothyroidism n=75 (50%) Group II	Subclinical hypothyroidism n=51 ,(34%) Group III
≤30 Female(n=11) Male(n=5)	 16 (10.66 %)	 2(1.34%) 0(0%)	 7 (4.6%) 1 (0.6%)	 2 (2.66 %) 4 (%)
31-50 Female(n=85) Male(n=18)	103 (68.67 %)	 11(7.3 %) 6(4%)	 47 (31.33%) 7 (4.66 %)	 27 (18%) 5 (3.3 %)
≥50 Female (n=22) Male(9)	 31 (20.67 %)	 4(2.6%) 1(0.6%)	 10(6.6 %) 4(2.6%)	 8 (5.3%) 4 (2.6%)

Table 3: Duration of diabetes mellitus (in years) in different thyroid dysfunction groups

Thyroid dysfunction	no (150)	Maximum	Minimum	Mean	Std. Deviation
Hyperthyroidism	24	11.00	2.00	6.84	2.44
Hypothyroidism	65	13.00	4.00	7.52	2.52
Subclinical-Hypothyroidism	51	11.00	2.00	5.88	2.56
Total	150	12.00	2.66	6.12	2.50

					P value
Subclinical hypothyroidism vs Hypothyroidism			0.0142*		
Hypothyroidism vs Hyperthyroidism			0.2352		
Hyperthyroidism vs subclinical Hypothyroidism			0.1531		
*P value < 0.05 – Significant , P < 0.01 Very Significant P value < 0.001HighlySignificant					

Table 4: Correlation of T3, T4, TSH and HbA1c with the duration of diabetes mellitus (in years)

Parameters	Duration of diabetes(years)
TSH	p < 0.001 r + 0.476
T4	p < 0.048 r -0.198
T3	P < 0.043 r -0.617
HbA1C	P < 0.001 r + 0.642

Table 5: Laboratory parameters studied in the thyroid dysfunction groups

Parameters	Hyperthyroidism Group I (GI) n=24	Hypothyroidism Group II (GII) n=75	Subclinical hypothyroidism Group (GIII) n=51	G1 vs GII	GI vs GIII	GII vs GIII
Hb	11.04 ± 1.46	9.86 ± 1.56	10.89± 1.88	0.021	0.053	0.049
Type of anaemia 1-microcytic						
hypochromic	10 (6.67%)	20 (13.33%)	20 (13.34%)	0.014	0.032	0.002
2-normocytic	12 (8.0%)	45 (30.0%)	26 (17.33%)	0.001	0.022	0.001
normochromic						
3-macrocytic	2 (1.33%)	10 (6.67%)	5 (3.33%)	0.012	0.035	0.012
FBS (mg/dl)	155.38 ± 11.04	162. 71 ± 14.54	160.16 ± 12.90	0.050	0.070	0.060
PP BS(mg/dl)	245.35 ±21.01	254.66 ± 28.10	247.90±25.52	0.020	0.091	0.072
HbA1c (%)	7.96 ± 0.86	8.15±0.85	8.02± 0.79	0.035	0.080	0.099
TSH (0.27-4.20 IU/L)	0.04 ± 0.05	9.85 ± 2.07	7.92 ± 1.20	0.02	0.04	0.05
T3(0.85-2.02ng/ml)	2.76 ± 0.33	0.59± 0.17	1.50 ± 0.36	0.02	0.01	0.02
T4 (5.13-14.06 (mg/dl)	17.07 ± 1.13	3.72 ± 1.42	9.98 ± 2.50	0.03	0.04	0.01
TC (mg/dl)	203.54 ± 26.23	214.20 ± 29.37	209.18 ± 27.05	0.03	0.04	0.02
TG(mg/dl)	178.26 ± 24.91	186.52 ± 26.58	183.55 ± 25.24	0.02	0.04	0.054
LDLc (mg/dl)	161.62 ± 31.81	167.62 ± 41.81	165.14 ± 38.05	0.04	0.04	0.07
HDLc (mg/dl)	32.09 ± 8.77	36.38 ± 11.79	34.65 ± 9.90	0.03	0.07	0.12
Urea (mg/dl)	19.23 ± 4.56	26.89 ± 3.76	22.76 ± 6.98	0.06	0.74	0.64
creatinine(mg/dl)	1.12 ± 0.06	2.22 ± 0.65	1.65 ± 0.43	0.12	0.18	0.06

Discussion

Endocrine disorders, thyroid dysfunctions and T2DM play complex interdependent interactions. As per table 1, out of 150 participants, with a notable gender disparity: 78.67% were female, and 21.33% were male was observed. This gender imbalance is consistent with other studies of **Demitrost et al** which found that among diabetic patients with thyroid disorders, 76.6% were females.[19] The higher prevalence of thyroid dysfunction in females with type 2 diabetes, as seen in this study and others, can be attributed to multiple factors including increased susceptibility to autoimmune disorders of thyroid, hormonal influences, different stressors affecting the (HPTA) hypothalamic-pituitary-thyroid axis[20]. As shown in table 2, the majority (68.67%) of participants were within the 31-50 age group, with a mean age of 42.64 ± 9.56 years for hyperthyroidism and 42.42 ± 9.9 years for both hypothyroidism and subclinical hypothyroidism. This aligns with findings from other previous studies including **VikheVB et al**, who reported a mean age of 43.16 ± 5.06 years for diabetic patients with thyroid disorders[21]. In the present study hypothyroidism is the most prevalent thyroid disorder among type 2 diabetes mellitus patients, affecting 75 patients (50%), followed by subclinical hypothyroidism in 51 patients (34%), and hyperthyroidism in 24 patients (16%). These findings indicate a high prevalence of thyroid dysfunction in diabetic patients, with a predominance of hypothyroid conditions. Compared to other studies, these results align with the general trend of increased thyroid disorders in diabetic populations[20,21]. Forinstance, a study by **Mehalingam V et al**. reported a prevalence of 31.4% for thyroid dysfunction in type 2 diabetics, with hypothyroidism being the most common disorder.[22]

In the present study, as shown in Table 3 the mean duration of diabetes observed was of 6.12 years. The p-value of 0.236 suggests that the differences in mean duration of diabetes among the three thyroid dysfunction groups are not statistically significant. The findings of our study are consistent with the study done by **Pramanik et al.** which stated that the risk of thyroid dysfunction increased with the duration of diabetes, while they didn't find a significant difference in the type of thyroid dysfunction based on diabetes duration, consistent with our non-significant p-value (= 0.235)[23]

Table 4 of this study shows a significant positive correlation ($p < 0.001$) of duration of T2DM with TSH and HbA1c. Our finding further supports those of **Vikhe et al** reporting a higher prevalence of thyroid dysfunction in diabetic patients with a longer duration of diabetes (>5 years).

Table 5 shows the laboratory parameters performed in study subjects. For FBS and PPBG, statistically significant differences were observed between the hypothyroid and subclinical hypothyroid groups ($p < 0.05$). However, no significant differences were found between the hyperthyroid group and the other two groups ($p > 0.05$). Similarly, for HbA1C levels, the hypothyroid group had the highest mean, with a statistically significant difference between the hypothyroid and subclinical hypothyroid groups ($p = 0.035$). However, the differences between the hyperthyroid group and the other two were not statistically significant ($p > 0.05$). These results emphasize the importance of monitoring glucose control in patients with thyroid disorders.

Our study shows a trend towards higher FBS levels in hypothyroidism and subclinical hypothyroidism compared to hyperthyroidism. This aligns with findings from a study by Demitrost and Ranabir which reported

higher Fasting Blood Sugar levels in hypothyroid diabetics compared to euthyroid diabetics.[20]

In our study the HbA1C levels across groups are relatively similar with hypothyroidism showing a slightly higher mean. A study by **Ogbonna SU** et al. found that HbA1C levelswere significantly higher in diabetics with thyroid dysfunction compared to those without, but they did not differentiate between types of thyroid dysfunction(24) .In our study type 2 diabetes mellitus patients , the mean HbA1C levels in all groups (ranging from 7.95% to 8.15%) indicate suboptimal glycemic control according to standard guidelines(2). This aligns with findings from a large pan Indian study by **Unnikrishnan et al.** which reported poor glycemic control in a significant proportion of Indian diabetics.[25]

Uncontrolled diabetes and thyroid dysfunction have a complex bidirectional relationship, each potentially exacerbating the other (**Singh & Gupta**). Chronic hyperglycemia in uncontrolled diabetes can impair thyroid function by reducing T3 levels and increasing reverse T3, potentially leading to a state of "low T3 syndrome".[25]

It may also affect the hypothalamic-pituitary-thyroid axis, altering TSH release. Conversely, thyroid dysfunction can significantly impact diabetes management. Hypothyroidism can increase insulin resistance and impair glucose metabolism, potentially worsening glycemic control in diabetics. It may also slow gastric emptying, affecting the timing of postprandial glucose peaks (**Sinha et al**).[26] Hyperthyroidism, on the other hand, can increase and promote rapid gastric emptying, leading to higher postprandial glucose levels (**Stumvoll M et al**) .It can also enhance the degradation of insulin, potentially increasing insulin requirements in diabetics.[12]

In this study we found the highest PPBG levels in the hypothyroidism group, and the differences were statistically significant. This is similar with some Indian studies, which reported higher postprandial glucose levels in hypothyroid diabetics.[15,18,19]

The findings of this study reveal significant differences in thyroid function tests across the three groups. Hyperthyroidism shows the lowest TSH (0.04 IU/ml) and highest T3 (2.76 ng/ml) and T4 (17.07 mg/dL) levels, while Hypothyroidism has the highest TSH (9.85 IU/ml) and lowest T3 (0.59 ng/ml) and T4 (3.72 mg/dL) levels. Subclinical Hypothyroidism falls between the two, with intermediate values for TSH (7.92 IU/ml), T3 (1.50 ng/ml), and T4 (9.98 mg/dL). All group comparisons yielded p- values below 0.05, indicating statistically significant differences in TSH, T3, and T4 levels among the groups.

Furthermore, both hypothyroidism and hyperthyroidism can alter lipid metabolism, indirectly affecting diabetes control .In terms of statistical significance, the p-values for the comparisons between the hypothyroidism group (G_1) and the subclinical hypothyroidism group (G_2), as well as between the hypothyroidism group (G_1) and the hyperthyroidism group (G_3), are all below 0.05 across the total cholesterol, triglyceride, and LDL levels. This indicates that the differences between these groups are statistically significant at a 5% level of significance, confirming that thyroid status has a measurable impact on lipid profiles. However, the associations between the subclinical hypothyroidism group (G_2) and the hyperthyroidism group (G_3) yield p-values greater than 0.05 (ranging from 0.054 to 0.07), suggesting no statistically significant differences between these two groups in terms of lipid levels. This implies that subclinical hypothyroidism and hyperthyroidism may

have less distinct effects on lipid profiles compared to overt hypothyroidism. For HDL levels, the pattern is less consistent, with only the comparison between the hypothyroidism and subclinical hypothyroidism groups showing a significant difference ($p = 0.03$). The comparisons between the hypothyroidism and hyperthyroidism groups, as well as between subclinical hypothyroidism and hyperthyroidism, result in p-values greater than 0.05 ($p = 0.07$ and $p = 0.09$, respectively), indicating no statistically significant relationship. These findings underline the importance of monitoring lipid levels in patients with thyroid dysfunction, particularly those with hypothyroidism. These findings are similar to several other studies on diabetes mellitus with thyroid dysfunction.[15-19]

The LDL results in our study, showing the highest levels in the hypothyroidism group, similar to the findings by others where higher LDL levels were observed in hypothyroid diabetics. Our HDL findings partially align with a study by **Nair et al**. which reported lower HDL levels in hypothyroid diabetics, consistent with our observation of lowest HDL in the hypothyroid group.[15] **Maheshwari et al.,** found that the mean total cholesterol level and VLDLc was significantly higher in hypothyroid subjects among diabetics when compared with euthyroid diabetic group.[26] We had also observed that levels of triglyceride were also higher in subjects of Group I (60.42%) as compared with Group II (45.67%). The results are similar with **Singh *et al*** who found significant associations between subclinical hypothyroidism and higher total cholesterol and LDL levels.[28]

Hypothyroidism and hyperthyroidism significantly affect lipid metabolism, leading to distinct alterations in lipid profiles.[26-29] In hypothyroidism, reduced thyroid hormone levels slow metabolic processes, resulting in elevated total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglyceride levels. This occurs due to decreased hepatic clearance of lipoproteins and reduced expression of LDL receptors, impairing cholesterol uptake by the liver and contributing to dyslipidemia and increased cardiovascular risk.[30] Additionally, hypothyroidism lowers high density lipoprotein (HDL) cholesterol, further exacerbating the lipid imbalance. Conversely, hyperthyroidism, characterized by excess thyroid hormones, increases the rate of lipid metabolism, often leading to reduced total cholesterol, LDL cholesterol, and triglycerides.[28-29] However, hyperthyroidism can also elevate HDL levels due to enhanced lipolysis and fatty acid oxidation.[26, 31] Despite these seemingly beneficial effects, hyperthyroidism can promote hepatic gluconeogenesis and increase the synthesis of cholesterol precursors, potentially altering lipid homeostasis and posing long-term cardiovascular risks. Thus, both thyroid dysfunctions— hypothyroidism and hyperthyroidism—can significantly disrupt lipid metabolism, though their impacts areopposing, requiring careful management to prevent adverse cardiovascular outcomes (**Goglia & Silvestri**,)[32]

Limitations

1. The relatively small sample size of 150 participants may have limited the statistical power to detect significant differences between groups.
2. A notable gender imbalance, with 78.67% female participants, could affect the generalizability of findings, particularly for male patients.
3. The absence of a control group of diabetic patients without thyroid dysfunction limits comparative analysis.

4. The cross-sectional design prevents the establishment of causal relationships or tracking changes over time.
5. The study's limited geographical scope and potential lack of control for confounding factors such as diet, physical activity, and concurrent medications may affect the broader applicability of the results

Conclusions

The present study concludes that the patients of type 2 diabetes mellitus have higher prevalence of thyroid dysfunctions as well as dyslipidemia. Thus this suggest the early management of these patients with dietary intervention, structured exercise, optimize thyroid hormone replacement therapy may be helpful in early management and prevention of cardiovascular complications associated with type 2 diabetes mellitus patients .Further longitudinal study in this kumaon region of Uttrakhand should be conducted with focus on elicidating the mechanisms behind the relationship regarding glycemic control, dyslipidemias and thyroid dysfunction in diabetes mellitus patients.

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