

The Impact of Autoimmune Thyroiditis on Thyroid Function, Lipid Profile, and Glycaemic Control in Patients with Type 2 Diabetes Mellitus

Sarmad Jaafar Mohammed Alrubaye*

College of Medicine, University of Al-Ameed, Kerbala, Iraq

* Corresponding: Sarmad Jaafar Mohammed Alrubaye, Telephone:(+96)47700800811, E-Mail: sarmadalrubaye5@gmail.com

Received: 11 May, 2025

Accepted: 26 June, 2025

Reviewed: 21 June, 2025

Published online: 6 November, 2025

Abstract:

Introduction: Autoimmune thyroiditis (AT) is more frequent in people with type 2 diabetes mellitus (T2DM) than those in general population. Its prevalence usually varies by region and demographic characteristic of patients. **Aim:** to explore the role of AT in thyroid dysfunction, dyslipidaemia and glycaemic control in T2DM patients. Also, to investigate the associations between different clinical and laboratory data with AT in the study groups.

Subjects and Methods: This is a cross-sectional study included 150 T2DM patients attending Al-Hussein Teaching Hospital, Kerbala, Iraq and 50 individuals included as a healthy control group. Full history and physical examination were performed for all subjects, body mass index (BMI) was determined in all individuals. Blood samples were collected to evaluate laboratory parameters including thyroid autoantibodies (Thyroglobulin (Tg) and thyroid peroxidase (TPO)), thyroid function tests, glycaemic control indices and lipid profiles. The thyroid gland size was measured by ultrasonography.

Results: From 150 T2DM patients included in the study, 62 (41.3%) were found to have AT and 88 patients with T2DM alone. The AT+T2DM group had significantly higher BMI ($33.89 \pm 5.59 \text{ kg/m}^2$) and larger thyroid size ($29.22 \pm 8.84 \text{ mL}$) than those of T2DM group (BMI = $31.59 \pm 7.94 \text{ kg/m}^2$, thyroid volume = $21.85 \pm 7.80 \text{ mL}$) and control group (BMI = $29.66 \pm 5.67 \text{ kg/m}^2$, thyroid volume = $9.28 \pm 3.15 \text{ mL}$) ($p < 0.0001$). Thyroid function test showed elevated TSH, decreased FT3, and decreased FT4 levels in AT+T2DM group compared to T2DM and control groups ($p < 0.0001$). The AT+T2DM group had bad glycaemic control, represented by a significant higher HbA1c ($13.27 \pm 5.02\%$) and FBG ($160.72 \pm 19.09 \text{ mg/dL}$) than those in other tested groups ($p < 0.0001$). Lipid profile was significantly higher in the AT+T2DM group than other tested groups ($p < 0.0001$). Thyroid autoantibodies had significant positive correlations with HbA1c, TG, and AI, and negative correlations with FT3, FT4, and HDL-c ($p < 0.01$).

Conclusion: Individuals with T2DM with AT have a higher prevalence of thyroid dysfunction, poorer glycaemic control, and a significantly higher lipid profile than patients without AT and non-diabetic controls.

Keywords: Autoimmune thyroiditis; Type 2 diabetes mellitus (T2DM); Thyroid dysfunction; glycaemic

1. Introduction

Relative insulin insufficiency with type 2 diabetes mellitus (T2DM), is a complicated metabolic disease. It is defined by insulin resistance that results in persistent hyperglycaemia and a number of long-term complications. The International Diabetes Federation claims that obesity, sedentary lifestyle, elderly populations and unhealthy diets are the primary reasons of the fast rise in the global burden of T2DM [1]. About 537 million people worldwide were reported to have diabetes in 2021 (10.5%), and by 2045, nearly 780 million people are expected to be diabetic [2]. Since thyroid dysfunction, in particular autoimmune thyroiditis (AT), has been demonstrated to impact cardiac outcomes in other cardiometabolic diseases, including diabetes, it has been identified as a potentially significant complication of diabetes [3]. Autoimmune thyroiditis is an endocrine disease that is most commonly reported as Hashimoto's thyroiditis. It is characterized by an excess of autoantibodies to thyroid antigens, including Thyroglobulin (Tg) and thyroid peroxidase (TPO) with subsequent damage and inflammation of the thyroid gland [4]. Autoimmune thyroiditis is more frequent in people with T2DM than those in general population. Its prevalence usually varies by region and demographic characteristic of patients (ranging from 5 to 45%) [5].

Although the significance of thyroid hormones in glucose metabolism is widely acknowledged, their role is also vital for lipid metabolism. Triiodothyronine and Thyroxine are involved in the pathophysiology of diabetes because they

alter lipid metabolism, hepatic glucose synthesis, and insulin sensitivity [6]. Evidence illustrated that elevated pancreatic beta-cell dysfunction and insulin resistance are linked with thyroid dysfunction, particularly hypothyroidism, which is more frequent in individuals with T2DM [7]. The mechanism underlying this association may directly impact the mechanism mediating glucose uptake and the feedback that thyroid hormones exert on various metabolic pathways.

Type 2 diabetes mellitus patients are also frequently having dyslipidaemia, which is defined by an elevated in triglycerides (TG) with low-high-density lipoprotein-cholesterol (LDL-c). Because hypothyroidism is related to elevated triglyceride and LDL-c, this abnormal lipid profile is more apparent in patients with altered thyroid function [8, 9]. It is not a coincidence that T2DM, thyroid dysfunction, and dyslipidaemia interact; these conditions affect one another and raise the already elevated risk of cardiovascular diseases in diabetic patients. Prior researches revealed that compared to people without AT, patients with chronic AT exhibit significant metabolic abnormalities [10, 11]. Empirical research will be required to ascertain the degree to which these observed adverse outcomes can be applied to T2DM patients with AT. Dyslipidaemia and impaired glucose metabolism may result from metabolic disturbances brought on by an autoimmune attack on the thyroid, according to some theories. Furthermore, even though several endocrine societies advocate for routine testing of thyroid

stimulating hormone (TSH) and thyroid autoantibodies in patients with T2DM, thyroid dysfunction in these patients is still not well understood [12]. Thyroid conditions are extremely common in people with diabetes and may be related to how metabolism is regulated, indicating that medical services providers need to be aware of and vigilant in monitoring thyroid function in these patients. Our goal was to explore the role of AT on thyroid dysfunction, dyslipidaemia and glycaemic control in T2DM patients. Also, to investigate the associations between body mass index (BMI), thyroid autoantibody levels, thyroid size, thyroid function indices, glycaemic control measures, lipid profile parameters, and disease duration.

2. Subjects and Methods

2.1 Study design and population:

This study is a cross-sectional involving 150 T2DM patients attending Al-Hussein Teaching Hospital in Kerbala, Iraq. A healthy control group; 50 non-diabetic subjects with matched age; were included also in the study. This study was approved by Al-Ameed University Research and Ethics committee (no. 126/25). All procedures were in accordance with the Helsinki Declaration. Thorough explanation of the study's nature and goal to each participant was conducted and written informed consent was acquired.

Inclusion criteria:

- Ages between 18 and 65 years with T2DM or healthy controls without T2DM agreed to be included in the study and,
- without any exclusion criteria mentioned below.

Exclusion criteria:

- Ages under 18 and over 65 years,
- hypertensive patients,
- people with type 1 diabetes,
- pregnant women,
- people with cardiovascular disease,
- people with complicated glycated haemoglobin (i.e., caused by severe anaemia and hemoglobinopathy),
- people taking thyroid-related systemic medications,
- people taking metformin due to fatty liver disease,
- people with polycystic ovarian syndrome,
- people taking medication for dyslipidaemia,
- people taking folic acid or vitamin B12 systemically, and
- people undergoing any hormonal therapy were all excluded from the patient and control group.

2.2 Clinical investigations

Every patient and participant were subjected to full history and physical examination, and their body mass index (BMI) was determined. Demographic information and the length of the illness were collected from patient's hospital files. A blood sample was aseptically collected from each patient.

The thyroid lobes' parameters were measured using Philips ClearVue 350 Ultrasound Machine (USA) and each lobe's volume (V) was determined as reported previously [13]. The isthmus volume was not included in the computation of total thyroid volume.

2.3 Laboratory investigations

Anti-TPO antibodies and Anti-Tg were measured with the ZEUS AtheNA Multi-

Lyte TPO/Tg Plus Test System as described by the manufacture (Zeus Scientific, New Jersey, United States). Autoimmune thyroiditis was identified by elevated anti-Tg and anti-TPO (≥ 40 International units per millilitre and ≥ 35 International units per millilitre, respectively) and characteristic ultrasound findings of the hypoechoic thyroid gland [14].

Serum levels of thyroxine (FT4), free triiodothyronine (FT3) and TSH were investigated to assess thyroid function using a Minividas compact multiparametric analyser (USA) according to manufacture instruction. Interpretation of results was as follows [15]:

- i. Subclinical hypothyroidism; normal FT3 with normal FT4 and TSH > 4.2 milliunits per litre
- ii. Primary hypothyroidism: FT4 < 12 picomoles per litre plus TSH > 4.2 milliunits per litre and/or FT3 < 2.8 picomoles per litre
- iii. Subclinical hyperthyroidism; normal FT4 and FT3 and TSH < 0.27 milliunits per litre
- iv. Primary hyperthyroidism, defined as TSH below 0.27 milliunits per litre and FT4 above 22 picomoles per litre and/or FT3 > 7 picomoles per litre

Lipid profile (triglycerides (TG), high-density lipoprotein cholesterol (HDL-c), and total cholesterol (TC)) was investigated using the Hitachi 902 Chemistry Analyzer - Hitachi, Japan as described by the manufacture. The atherogenic index (AI) was computed using the Friedewald formula, very low-density lipoprotein cholesterol (VLDL-c), and low-density lipoprotein

cholesterol (LDL-c) (Abbott, Wilson, Kannel, & Castelli, 1988; Friedewald, Levy, & Fredrickson, 1972). A pre-calibrated portable glucometer (CareTouch) and the KT-889 Human Insulin ELISA Kit (Epitope Diagnostics Inc., United States) were used to measure fasting insulin levels and fasting blood glucose (FBG), respectively. After that, The insulin resistance assessment using the homeostatic model (HOMA-IR) was calculated (Abbasi, Okeke, & Reaven, 2014). Chromatographic spectrophotometric ion exchange was used to measure glycated haemoglobin (HbA1c) (Genius Diagnostics, Belgium).

2.4 Statistical analysis

SPSS version 24.0 was used to conduct the statistical analysis. The mean $\pm$ standard deviation was used to display non-categorical data. Study groups were compared using the independent t-test for continuous variables and the Chi-squared test for categorical variables. Pearson's correlation for the associations between BMI, thyroid volume, duration of disease, autoantibody levels, thyroid function, glycaemic indices, and lipid profile. Statistical significance was defined as a p-value of less than 0.05.

3. Results

The present results revealed that, from 150 T2DM patients included in the study, 62 (41.3%) were found to have AT and 88 patients with T2DM alone. Table (1) displays the demographic, clinical, radiological and laboratory data of the populations included in the study. Our results found that, the duration of T2DM,

BMI, size of thyroid gland, presence of thyroid nodules, serum levels of anti-Tg and anti TPO were significantly higher in AT+T2DM group than T2DM and control groups ($p < 0.05$).

All groups were assessed for thyroid function, glycaemic control, and lipid profiles (Table 2- 3). The AT+T2DM group had significantly higher TSH levels and lower FT3 and FT4 levels than the T2DM and control groups ($p < 0.0001$). Furthermore, all glycaemic indices and lipid profile of AT+T2DM group were significantly higher than those of T2DM and

control groups ($p < 0.00$). Figure 1-2 also displays this data. The associations between BMI, thyroid autoantibody levels, thyroid volume, thyroid function indices, glycaemic control measures, lipid profile parameters, and disease duration were investigated using Pearson's correlation analysis. Table 4 provides a summary of the correlations' strength as indicated by the correlation coefficient (r). Weak (r equal to 0.10–0.29), moderate (r equal to 0.30–0.49), and strong (r equal to 0.50–1.0) correlations were distinguished.

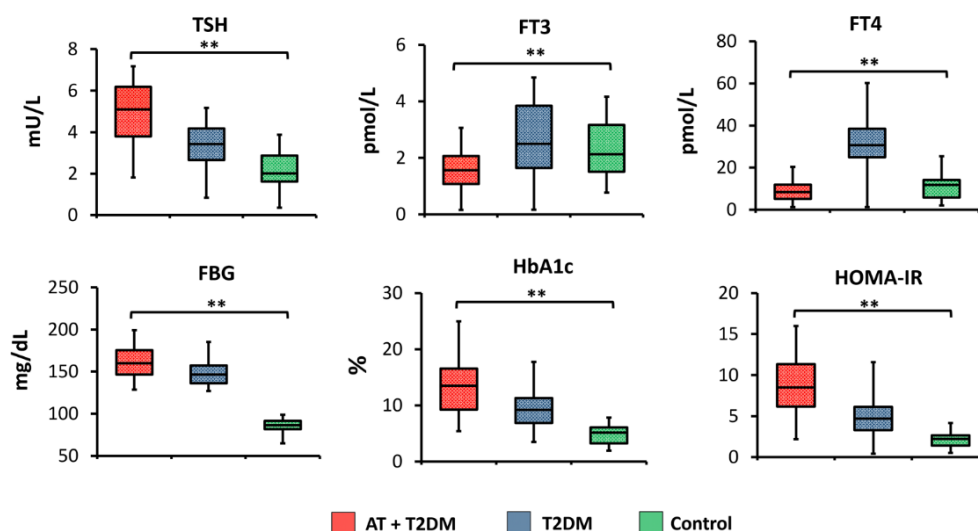


Figure 1: Thyroid Hormonal and Glycaemic Measurements of the Study Subjects

Box plots showing measurements of thyroid hormones (TSH, FT3 and FT4) as well as glycaemic indices (FBG, HbA1c and HOMA-IR) in the three study groups: AT + T2DM (red), T2DM (blue), and Control (green).

TSH; thyroid-stimulating hormone, FBG; fasting blood glucose, FT3; free triiodothyronine, HOMA-IR; homeostasis model assessment of insulin resistance, FT4; free thyroxine, HbA1c; glycated haemoglobin.

** Statistical significance between the groups at $p < 0.0001$

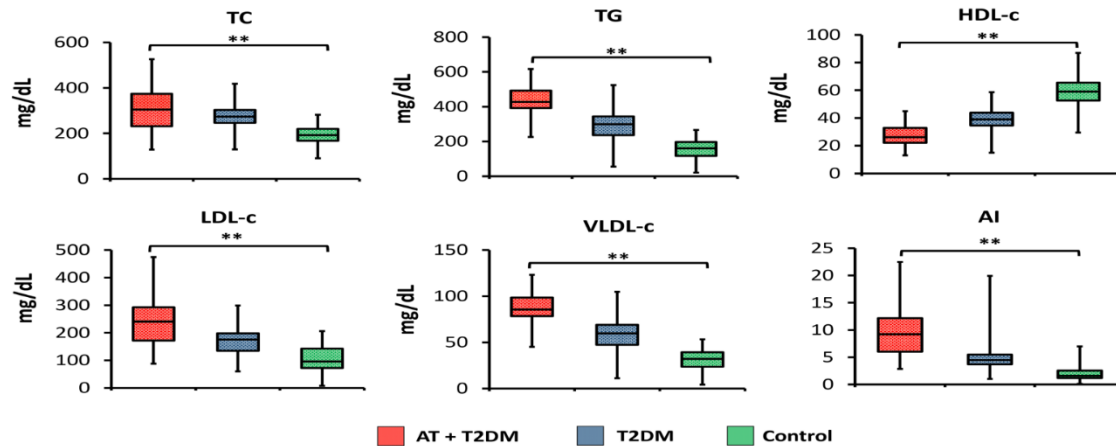


Figure 2: Study Participants' Lipid Profile Measurements

Box plots illustrating measurements of lipid profile parameters (TC, TG, HDL-c, LDL-c, VLDL-c, and AI) in the three groups: AT + T2DM (red), T2DM (blue), and Control (green). TG; triglycerides, TC; total cholesterol, VLDL-c; very low-density lipoprotein cholesterol, LDL-c; low-density lipoprotein cholesterol, HDL-c; high-density lipoprotein cholesterol, AI; atherogenic index.

** Statistical significance among the groups at p value < 0.0001

Table 1: The demographic, clinical, radiological and laboratory data of the study populations

	AT+T2DM (n = 62) Mean ± SD or n (%)	T2DM (n = 88) Mean ± SD or n (%)	Control (n = 50) Mean ± SD or n (%)	p1-value	p2-value
Age, years	49.06±9.41	47.36±8.76	48.08±10.73	0.1305	0.5632
Sex					
Female	38 (61.3)	49 (55.7)	27 (54.0)	0.4931	0.7011
Male	24 (38.7)	39 (44.3)	23 (46.0)		
BMI, Kg/m ²	33.89±5.59	31.59±7.94	29.66±5.67	0.0226*	0.0047*
Duration of T2DM, years	6.80±3.19	4.17±1.97	-	< 0.0001*	
Thyroid Volume, mL	29.22±8.84	21.85±7.80	9.28±3.15	< 0.0001*	< 0.0001*
Thyroid Nodules					
Yes	49 (79.0)	50 (56.8)	13 (26.0)	0.0046*	< 0.0001*
No	13 (21.0)	38 (43.2)	37 (74.0)		
Anti-Tg, IU/mL	99.50±42.95	23.26±10.28	8.71±5.27	< 0.0001*	< 0.0001*
Anti-TPO, IU/mL	102.28±61.56	20.35±10.80	15.14±9.02	< 0.0001*	< 0.0001*

T2DM; type 2 diabetes mellitus, BMI; body mass index, AT; autoimmune thyroiditis, SD; standard deviation, Anti-Tg; anti-thyroglobulin antibody, Anti-TPO; anti-thyroid peroxidase antibody, p1; implies comparison between AT+T2DM and T2DM, p2.; implies comparison between the 3 study groups.

* Signifies statistical significance at p value < 0.05

Table 2: The impact of AT on thyroid function, glycaemic control and lipid profile

	AT+T2DM (n = 62) mean±SD	T2DM (n = 88) mean±SD	Control (n = 50) mean±SD	p1-value	p2-value
Thyroid Hormones					
TSH, mU/L	5.07±1.76	3.42±1.24	2.27±0.99	< 0.0001	< 0.0001
FT3, pmol/L	1.68±0.88	2.71±1.33	2.31±0.96	< 0.0001	< 0.0001
FT4, pmol/L	8.83±4.94	30.70±10.91	10.96±5.60	< 0.0001	< 0.0001
Glycaemic Indices					
HbA1c, %	13.27±5.02	9.07±3.14	4.76±1.61	< 0.0001	< 0.0001
FBG, mg/dL	160.72±19.09	148.45±14.80	85.91±8.79	< 0.0001	< 0.0001
HOMA-IR	8.54±3.56	4.88±2.18	2.07±0.89	< 0.0001	< 0.0001
Lipid Profile					
TC, mg/dL	302.34±95.68	272.93±46.11	193.53±57.47	0.0071	< 0.0001
TG, mg/dL	431.06±88.68	299.05±87.93	155.40±57.47	< 0.0001	< 0.0001
HDL-c, mg/dL	27.36±6.87	38.90±7.17	58.76±11.82	< 0.0001	< 0.0001
LDL-c, mg/dL	243.48±89.96	174.22±42.77	103.69±49.86	< 0.0001	< 0.0001
VLDL-c, mg/dL	86.21±17.74	59.81±17.59	31.08±11.49	< 0.0001	< 0.0001
AI	9.68±4.80	4.90±2.55	1.93±1.26	< 0.0001	< 0.0001

SD stands for standard deviation. TSH: thyroid-stimulating hormone; FT3: free triiodothyronine; FT4: free thyroxine; T2DM: type 2 diabetes mellitus; AT: autoimmune thyroiditis; Fasting blood glucose, or FBG HOMA-IR: Homeostatic model evaluation of insulin resistance; HbA1c: Glycated haemoglobin; TG: Triglycerides TC: Total cholesterol LDL-c: Low-density lipoprotein cholesterol; VLDL-c: Very low-density lipoprotein cholesterol; HDL-c: High-density lipoprotein cholesterol; Atherogenic index, or AI. p1; implies a comparison between AT+T2DM and T2DM, p2.; implies a comparison between the 3 study groups.

Table 3: Thyroid Dysfunction Frequency in the study groups

	AT+T2DM (n = 62) n (%)	T2DM (n = 88) n (%)	Control (n = 50) n (%)
Subclinical hypothyroidism	16 (25.8)	8 (9.1)	0 (0.0)
Primary hypothyroidism	25 (40.3)	3 (3.4)	4 (8.0)
Subclinical hyperthyroidism	4 (6.5)	0 (0.0)	0 (0.0)
Primary hyperthyroidism	1 (1.6)	0 (0.0)	1 (2.0)

T2DM; type 2 diabetes mellitus, AT; autoimmune thyroiditis

Table 4: Correlation between clinical and laboratory data of the study populations

	r-value				
	BMI	Disease Duration	Anti-Tg	Anti-TPO	Thyroid volume
Anti-Tg	0.12	0.496**			
Anti-TPO	0.171	0.394**	0.600**		
Thyroid Volume	0.150	0.588**	0.424**	0.346**	
TSH	0.136	0.267**	0.338**	0.348**	0.214**
FT3	-0.147	-0.236**	-0.318**	-0.248**	-0.178*
FT4	-0.154	-0.332**	-0.600**	-0.511**	-0.305**
FBG	0.347**	0.207*	0.126	0.153	0.172*
HbA1c	0.462**	0.388*	0.426**	0.390**	0.189*
HOMA-IR	0.251*	0.547**	0.387**	0.421**	0.226**
HDL-c	-0.429**	-0.285**	-0.467**	-0.436**	-0.245**
LDL-c	0.309**	0.176*	0.390**	0.428**	0.261**
VLDL-c	0.402**	0.231**	0.474**	0.480**	0.174*
TC	0.345**	0.047	0.286*	0.269**	0.136
TG	0.402**	0.231*	0.474**	0.479**	0.174*
AI	0.279**	0.179*	0.410**	0.432**	0.217*

* indicates statistical significance when p is less than 0.05

** indicates statistical significance when p is less than 0.01.

BMI; body mass index, Anti-Tg; anti-thyroglobulin antibody, FT3; free triiodothyronine, TSH; thyroid stimulating hormone, Anti-TPO; anti-thyroid peroxidase antibody, FT4; thyroxine, FBG; fasting blood glucose, HbA1c; glycated haemoglobin, HOMA-IR; homeostatic model assessment of insulin resistance, HDL-c; high-density lipoprotein cholesterol, TG; triglycerides, TC; total cholesterol, VLDL-c; very low-density lipoprotein cholesterol, LDL-c; low-density lipoprotein cholesterol, AI; atherogenic index. r-value; correlation coefficient.

4. Discussion

Our goal was to assess how AT may affect the lipid profile, glycaemic control, and thyroid function in individuals with type 2 diabetes. Our findings implied that, a possible disruption in the pathophysiology of AT may impact the diabetic status and correlate it with a poor disease outcome. In contrast to some earlier studies (10%, and 20%), we detected a high prevalence of AT among T2DM patients (41.3%) [16, 17]. Our result is in agreement with Fregenal et al., as they obtained a prevalence of 48% [18]. In comparison to the T2DM group, we

also found that the AT+T2DM group had a higher BMI and a longer history of diabetes. Given that obesity may serve as a common underlying factor that may worsen inflammatory responses that impact thyroid pathology and metabolic health, the elevated BMI seen here suggests that obesity is a risk factor for AT in T2DM [19]. Additionally, the possibility of developing AT is higher in individuals with prolonged exposure to diabetic hyperglycaemia [7], suggesting a bidirectional relationship between AT and diabetes. This is supported by the fact that

thyroid nodules were more common in the AT+T2DM group because people with AT are more likely to develop nodular disease if they also have diabetes than if they only have AT [20]. Previous studies showing thyroid gland expansion in the context of AT support the significant increase in thyroid size observed in the AT+T2DM group [21]. Additionally, the characteristic lymphocytic infiltration and follicular hyperplasia of AT may be the cause of the increase in thyroid volume [22]. Due to the group's AT condition, Anti-Tg and anti-TPO thyroid autoantibody levels were considerably greater in the AT + T2DM group than in the T2DM group, as expected. It was interesting to us that T2DM had significantly higher levels of these autoantibodies than the control group. This suggests that diabetes may increase a person's vulnerability to thyroid autoimmunity. Furthermore, patients with both type 1 and type 2 diabetes frequently have these autoantibodies, indicating an autoimmune phenomenon that makes managing diabetes more difficult [23]. These findings emphasize how crucial it is to regularly examine diabetic patients for thyroid autoantibodies, especially if they are showing clinical symptoms of thyroid dysfunction. Furthermore, AT's effects on type 2 diabetes go beyond obesity and thyroid enlargement; they may also have an impact on lipid profiles, glycaemic control, and thyroid hormone levels.

Comparing AT+T2DM patients to those with T2DM alone and healthy controls, we found that they show notable changes in hormonal and metabolic parameters. First of all, the AT+T2DM

group has markedly elevated TSH levels that point to an underactive thyroid, which is consistent with the pathophysiology of AT, in which the destruction of the thyroid gland results in insufficient hormone production [24]. FT3 and FT4 levels were significantly lower in this group, which supports the idea of hypothyroidism and suggests compromised thyroid endocrine function. Similar results have been documented in earlier research, emphasizing that thyroid dysfunction is common in people with autoimmune thyroid disease, particularly when diabetes is present [7]. We found that the AT+T2DM group had a significantly higher prevalence of thyroid dysfunction, specifically subclinical and primary hypothyroidism, as a result of the thyroid hormone imbalance than did the control group and their T2DM-only counterparts. Thyroid dysfunction and autoimmune thyroiditis are significantly correlated in this diabetic population, as evidenced by the rates of primary hypothyroidism (40.3% vs. 3.4% in T2DM and 8.0% in controls) and subclinical hypothyroidism (25.8% vs. 9.1% in T2DM and 0.0% in controls). When compared to the general population, these findings are comparable to the prevalence found in patients with T2DM and latent autoimmune diabetes in adults (LADA) [15]. Although hypothyroidism is the most prevalent thyroid dysfunction linked to AT, particularly in diabetics, research has also shown that subclinical hypothyroidism occurs in people with type 2 diabetes and has been linked to an increased risk of cardiovascular disease and death [25]. These results are consistent with the prevalence of subclinical hyperthyroidism (6.5%) in the

AT + T2DM group. It is yet unknown, though, if the AT condition would reverse the hyperthyroidism if it were to persist. Additionally, we found that this cohort had a low prevalence of primary hyperthyroidism (1.6%), which is consistent with reports of low rates of hyperthyroidism in T2DM and LADA [15], indicating that hypothyroid states are the predominant form of primary thyroid dysfunction in this study population.

The AT+T2DM group had significantly higher levels of the glycaemic control metrics, especially HbA1c and FBG. In comparison to the T2DM and control groups, which had HbA1cs of 9.07% and 4.76%, respectively, the comorbid group's glycaemic control is significantly worse, with a HbA1c of 13.27%. According to earlier research, thyroid autoimmunity may worsen metabolic control in a vicious cycle where thyroid dysfunction is made worse by poor glycaemic control [26]. Therefore, glycaemic control in diabetics would probably benefit from effectively regulating thyroid autoimmunity. Additionally, the significant increase in HOMA-IR observed in the AT+T2DM group emphasizes the significance of the hyperinsulinemia state that defines T2DM. The metabolism of glucose depends on thyroid hormones, and hypothyroidism can exacerbate insulin resistance [27]. Our results highlight the necessity of regularly assessing thyroid function in diabetic patients in order to reduce insulin resistance and its negative effects.

VLDL-c TC, TG and LDL-c, were significantly higher in the AT+T2DM group, but HDL-c exhibited the opposite pattern. These modifications also draw

attention to the metabolic abnormalities linked to AT in type 2 diabetes. A higher AI of 9.68, which indicates increased cardiovascular susceptibility, is the consequence of elevated TG levels (431.06 mg/dL) and decreased HDL-c levels (27.36 mg/dL) [28]. These results are supported by earlier research that links thyroid dysfunction to dyslipidaemia, which has been demonstrated to be common in both diabetic and non-diabetic people with AT [29]. Thyroid-related complications are much more common in the AT+T2DM group, which suggests that diabetic patients, particularly those who exhibit obesity or metabolic syndrome characteristics, should undergo thorough screening for thyroid dysfunction. Given the connection between lipid profiles, glucose metabolism, and thyroid hormones, patients may benefit from an integrative management strategy that addresses both diabetes and AT.

The relation between the indices of AT-mediated thyroid dysfunction, poor glycaemic control, and dyslipidemia in type 2 diabetes and the known determinants of these conditions, such as BMI, disease duration, and thyroid autoantibodies, was investigated using Pearson's correlation analysis. Multiple pathways regarding AT's involvement in the development of T2DM were suggested by correlation analysis, which revealed a complex interaction between AT and metabolic dysfunction.

Long-term exposure to diabetes is linked to thyroid growth and insulin resistance, as evidenced by the strong positive correlation between disease duration and thyroid volume ($r = 0.588$) and HOMA-IR ($r = 0.547$). These patterns are

consistent with earlier findings that indicated a prolonged duration of type 2 diabetes is associated with abnormal thyroid function and a worsening of insulin resistance [30]. Furthermore, the somewhat positive associations between thyroid autoantibodies and the length of the disease suggest that chronic diabetes may exacerbate thyroid dysfunction by raising autoimmune activity [3]. On the other hand, FT4 levels and disease duration have a moderately negative correlation ($r = -0.332$), suggesting that thyroid hormone levels fall with longer diabetes duration. Thyroid hormone imbalances like this can worsen metabolic dysfunction, which makes managing diabetes more difficult and raises the risk of cardiovascular disease [31].

In line with an earlier research that found high BMI to be a crucial predictor of glycaemic control in diabetic populations, BMI showed significant correlations with a number of metabolic parameters, including moderately positive correlations with HbA1c and FBG [32]. This study's strong negative correlation between BMI and HDL-c supports reports that lower HDL-c levels, a sign of an increased risk of cardiovascular disease, are frequently associated with higher BMI [33]. Furthermore, the strong associations between BMI and TG, LDL-c, and consequently VLDL-c confirm the connection between dyslipidaemia and obesity. It has been noted that a higher body mass index (BMI) is related to a more dysregulated lipid profile, potentially increasing the risk of cardiovascular comorbidities in individuals with diabetes [34].

As anticipated in autoimmune thyroiditis, the strong correlation between anti-Tg and anti-TPO ($r = 0.600$) shows a close relationship between the two autoantibodies. Additionally, the impact of autoimmune processes on glycaemic control is highlighted by the moderately positive associations of anti-Tg and anti-TPO with HbA1c ($r = 0.426$ and $r = 0.390$, respectively). This interaction adds another level of complexity to the management of diabetes by indicating that patients with elevated thyroid autoantibodies may be more vulnerable to inadequate glycaemic control [3]. Anti-Tg and FT4 have a strong negative correlation ($r = -0.600$), which suggests a possible mechanism by which autoimmune activity causes thyroid dysfunction and contributes to metabolic abnormalities in T2DM patients [26].

5. Conclusion

In conclusion, AT has a major impact on T2DM patients, it affects BMI, thyroid volume, and thyroid autoantibody levels. There are significant correlations between AT and metabolic parameters in T2DM patients. Therefore, the correlations found between lipid profiles, glycaemic control, BMI, and thyroid function support the idea that treating thyroid disease is essential to enhancing metabolic health in diabetic patients.

Future multi-center studies could help validation of these findings across different settings and geographical regions.

6. Acknowledgments

The Al-Hussein Teaching Hospital and Al Kafeel Specialized Hospital in Kerbala,

Iraq, are acknowledged by the author for their assistance, specially all laboratory technicians who performed all the laboratory tests as a part of their routine work.

Source of Funding: University of Al-Ameed.

Conflict of Interest: None

AI declaration statement: Not applicable

References

1. Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, Ostolaza H, Martín C. Pathophysiology of Type 2 Diabetes Mellitus. *Int J Mol Sci.* 2020 Aug 30;21(17):6275. doi: 10.3390/ijms21176275. PMID: 32872570; PMCID: PMC7503727.
2. Sun H, Saeedi P, Karuranga S, Pinkepank M, Ogurtsova K, Duncan BB, Stein C, Basit A, Chan JCN, Mbanya JC, Pavkov ME, Ramachandran A, Wild SH, James S, Herman WH, Zhang P, Bommer C, Kuo S, Boyko EJ, Magliano DJ. IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. *Diabetes Res Clin Pract.* 2022 Jan;183:109119. doi: 10.1016/j.diabres.2021.109119. Epub 2021 Dec 6. Erratum in: *Diabetes Res Clin Pract.* 2023 Oct;204:110945. doi: 10.1016/j.diabres.2023.110945. PMID: 34879977; PMCID: PMC11057359.
3. Biondi B, Kahaly GJ, Robertson RP. Thyroid Dysfunction and Diabetes Mellitus: Two Closely Associated Disorders. *Endocr Rev.* 2019 Jun 1;40(3):789-824. doi: 10.1210/er.2018-00163. PMID: 30649221; PMCID: PMC6507635.
4. Tywanek E, Michalak A, Świrski J, Zwolak A. Autoimmunity, New Potential Biomarkers and the Thyroid Gland-The Perspective of Hashimoto's Thyroiditis and Its Treatment. *Int J Mol Sci.* 2024 Apr 26;25(9):4703. doi: 10.3390/ijms25094703. PMID: 38731922; PMCID: PMC11083198.
5. Hadgu R, Worede A, Ambachew S. Prevalence of thyroid dysfunction and associated factors among adult type 2 diabetes mellitus patients, 2000-2022: a systematic review and meta-analysis. *Syst Rev.* 2024 Apr 30;13(1):119. doi: 10.1186/s13643-024-02527-y. PMID: 38689302; PMCID: PMC11061916.
6. Mendez DA, Ortiz RM. Thyroid hormones and the potential for regulating glucose metabolism in cardiomyocytes during insulin resistance and T2DM. *Physiol Rep.* 2021 Aug;9(16):e14858. doi: 10.14814/phy2.14858. PMID: 34405550; PMCID: PMC8371345.
7. Rong F, Dai H, Wu Y, Li J, Liu G, Chen H, Zhang X. Association between thyroid dysfunction and type 2 diabetes: a meta-analysis of prospective observational studies. *BMC Med.* 2021 Oct 21;19(1):257. doi: 10.1186/s12916-021-02121-2. PMID: 34670571; PMCID: PMC8529738.
8. Berberich AJ, Hegele RA. A Modern Approach to Dyslipidemia. *Endocr Rev.* 2022 Jul 13;43(4):611-653. doi: 10.1210/endrev/bnab037. PMID: 34676866; PMCID: PMC9277652.
9. Hmood A, Bdair B, Al-Graittee S. Evaluation of Thyroid Volume and Thyroid Function in Newly Diagnosed Type 2 Diabetes Mellitus Patients. *Systematic Reviews in Pharmacy.* 2020; 11(5): 451-457.
10. Uddin M J, Tran DK, Hannan M A, Rahman MA, Moni A, Lam NT, et al. Autoimmune diseases and metabolic disorders: molecular connections and potential therapeutic targets Translational Autoimmunity. 2022. (pp. 169-183); Elsevier.
11. Jing M, Shao S, Ma S, Gao L, Wang Q, Zhou M. Exploring the link between obesity and hypothyroidism in autoimmune thyroid diseases: a metabolic perspective. *Front Mol Biosci.* 2024 Apr 22;11:1379124. doi:

- 10.3389/fmolb.2024.1379124. PMID: 38712344; PMCID: PMC11070466.
12. Kalra S, Aggarwal S, Khandelwal D. Thyroid Dysfunction and Dysmetabolic Syndrome: The Need for Enhanced Thyrovigilance Strategies. *Int J Endocrinol*. 2021 Mar 29;2021:9641846. doi: 10.1155/2021/9641846. PMID: 33859689; PMCID: PMC8024090.
13. C Chang CY, Lei YF, Tseng CH, Shih SR. Thyroid segmentation and volume estimation in ultrasound images. *IEEE Trans Biomed Eng*. 2010 Jun;57(6):1348-57. doi: 10.1109/TBME.2010.2041003. Epub 2010 Feb 17. PMID: 20172782.
14. Gonçalves F T, Feibelmann T C M, Ranza R, Daud M S, Taliberti BH B, Pinto R d M C, Jorge PT. Autoimmune thyroiditis and rheumatoid arthritis: is there really an association? *The Endocrinologist*. 2009; 19(1): 31-34.
15. Akbar DH, Ahmed MM, Al-Mughales J. Thyroid dysfunction and thyroid autoimmunity in Saudi type 2 diabetics. *Acta Diabetol*. 2006 May;43(1):14-8. doi: 10.1007/s00592-006-0204-8. PMID: 16710644.
16. Sah N K, Deo S K, Walia H K, Singh A, Prasad S, Kaur K. Thyroid autoimmunity among type 2 diabetes mellitus: assessing anti-thyroid peroxidase (anti-TPO) antibodies. *Biomedicine*. (2021; 41(4): 720-723.
17. Subekti I, Pramono LA, Dewiasty E, Harbuwono DS. Thyroid Dysfunction in Type 2 Diabetes Mellitus Patients. *Acta Med Indones*. 2017 Oct;49(4):314-323. PMID: 29348381.
18. Centeno Maxzud M, Gómez Rasjido L, Fregenal M, Arias Calafiore F, Córdoba Lanus M, D'Urso M, Luciardi H. Prevalencia de disfunción tiroidea en pacientes con diabetes mellitus tipo 2 [Prevalence of thyroid dysfunction in patients with type 2 diabetes mellitus]. *Medicina (B Aires)*. 2016;76(6):355-358. Spanish. PMID: 27959843.
19. Walczak K, Sieminska L. Obesity and Thyroid Axis. *Int J Environ Res Public Health*. 2021 Sep 7;18(18):9434. doi: 10.3390/ijerph18189434. PMID: 34574358; PMCID: PMC8467528.
20. Ji B, Shi S, Zhao H, Ma X, Sheng J, Ban B, Gao G. Pituitary-Thyroid Hormones and Related Indices in Euthyroid Type 2 Diabetes: Association With Thyroid Nodules. *Diabetes Metab Syndr Obes*. 2025 Feb 27;18:627-636. doi: 10.2147/DMSO.S503444. PMID: 40034479; PMCID: PMC11875124.
21. Pankiv V, Yuzvenko T Y, Pankiv I. Thyroid volume and nodules in patients with impaired fasting glucose and type 2 diabetes mellitus. *International Journal of Endocrinology (Ukraine)*. 2022; 18(5), 273–277. <https://doi.org/10.22141/2224-0721.18.5.2022.1185>
22. Brancatella A, Torregrossa L, Viola N, Sgrò D, Casula M, Basolo F, Materazzi G, Marinò M, Marcocci C, Santini F, Latrofa F. In Graves' disease, thyroid autoantibodies and ultrasound features correlate with distinctive histological features. *J Endocrinol Invest*. 2023 Aug;46(8):1695-1703. doi: 10.1007/s40618-023-02044-0. Epub 2023 Feb 25. PMID: 36840841; PMCID: PMC10348939.
23. Hu Y, Zheng J, Ye X, Song Y, Wu X. Association Between Elevated Thyroid Peroxidase Antibody and Abdominal Fat Distribution in Patients with Type 2 Diabetes Mellitus. *Diabetes Metab Syndr Obes*. 2022 Mar 17;15:863-871. doi: 10.2147/DMSO.S345507. PMID: 35321353; PMCID: PMC8938158.
24. Orgiazzi J. Thyroid autoimmunity. *La presse médicale*. 2012; 41(12): e611-e625.
25. Geng, J., Lu, W., Hu, T., Tao, S., Zhang, H., Chen, J., . . . Wang, B. (2015). Subclinical hyperthyroidism increases risk of coronary heart disease events in type 2 diabetes mellitus. *Endocrine*, 49, 557-559.

26. Eom YS, Wilson JR, Bernet VJ. Links between Thyroid Disorders and Glucose Homeostasis. *Diabetes Metab J*. 2022 Mar;46(2):239-256. doi: 10.4093/dmj.2022.0013. Epub 2022 Mar 24. PMID: 35385635; PMCID: PMC8987680.
27. Răcățaian N, Leach N V, Bolboacă S D, Soran M L, Flonta M, Valea A, et al. The crosstalk between insulin resistance, systemic inflammation, redox imbalance and the thyroid in subjects with obesity. *Journal of Mind and Medical Sciences*. 2021; 8(1): 139-148.
28. Lawal H, Ado A M, Usman M, Garba U S. Antihyperlipidemic Activity of *Plectranthus neochilus* Leaf Ethanolic Extract on Fat-Fed Male Wistar Rats. *International Journal of Science for Global Sustainability (IJS GS)*. 2024; 10(2).
29. K VB, Cm P, V KV, Ss Y. Prevalence and Association of Thyroid Dysfunction With Diabetes Mellitus in a Tertiary Care Hospital: A Retrospective Study. *Cureus*. 2025 Feb 28;17(2):e79855. doi: 10.7759/cureus.79855. PMID: 40166517; PMCID: PMC11955718.
30. Sarabhai T, Kostev K. Thyroid disorders and the incidence of type 2 diabetes: insights from a 10-year cohort study in Germany. *Endocr Connect*. 2025 Jan 31;14(3):e240554. doi: 10.1530/EC-24-0554. PMID: 39831884; PMCID: PMC11799829.
31. Anwar U, Arshad J, Naeem UH, Zahid A, Jehan AS, Ramzan S, Awan MA. The Impact of Thyroid Hormone Imbalance on Cardiovascular Health: A Population-Based Study. *Cureus*. 2024 Dec 27;16(12):e76457. doi: 10.7759/cureus.76457. PMID: 39867074; PMCID: PMC11769698.
32. Oumer A, Ale A, Tariku Z, Hamza A, Abera L, Seifu A. Waist-to-hip circumference and waist-to-height ratio could strongly predict glycemic control than body mass index among adult patients with diabetes in Ethiopia: ROC analysis. *PLoS One*. 2022 Nov 9;17(11):e0273786. doi: 10.1371/journal.pone.0273786. PMID: 36350840; PMCID: PMC9645629.33. Kosmas, C. E., Rodriguez Polanco, S., Bousvarou, M. D., Papakonstantinou, E. J., Peña Genao, E., Guzman, E., & Kostara, C. E. (2023). The triglyceride/high-density lipoprotein cholesterol (TG/HDL-C) ratio as a risk marker for metabolic syndrome and cardiovascular disease. *Diagnostics*, 13(5), 929.
34. La Sala L, Pontiroli AE. Prevention of Diabetes and Cardiovascular Disease in Obesity. *Int J Mol Sci*. 2020 Oct 31;21(21):8178. doi: 10.3390/ijms21218178. PMID: 33142938; PMCID: PMC7663329.