

## Clinical Impact of Thyroid Dysfunction on Metabolic Syndrome Components in Obese Patients

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### Abstract:

**Background:** One of the main factors controlling metabolic homeostasis is thyroid dysfunction. Changes in energy expenditure and lipid metabolism, two important aspects of metabolic syndrome, are often linked to hypothyroidism.

**Methodology:** Twenty controls and sixty patients with primary hypothyroidism made up the 80 participants in total. Thyroid hormones (TSH, T3, and T4) and metabolic parameters (FBS, total cholesterol, triglycerides, HDL, LDL, and VLDL) were assessed. To compare the groups and assess the importance of the metabolic alterations, statistical analysis was done.

**Results:** The results demonstrated a highly significant increase in BMI and TSH levels among patients compared to controls ( $p < 0.01$ ). Regarding the lipid profile, a profound and significant elevation was observed in Triglycerides (TGs) and VLDL levels ( $p < 0.0001$ ). However, Total Cholesterol, LDL, HDL, and Fasting Blood Sugar (FBS) showed non-significant (NS) differences between the two groups ( $p > 0.05$ ).

**Conclusion:** These findings highlight that primary hypothyroidism leads to a "lipid-dominant" metabolic disturbance characterized by isolated hypertriglyceridemia and increased BMI. The stability of glycemic markers (FBS) suggests that lipid alterations precede glucose impairment in the early stages of thyroid dysfunction. Routine assessment of TGs is recommended for early identification of metabolic risk in hypothyroid patients.

## التأثير السريري لاختلال وظائف الغدة الدرقية على مكونات متلازمة التمثيل الغذائي لدى المرضى الذين

### يعانون من السمنة.

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#### الخلاصة

**المقدمة:** يُعدّ خلل وظائف الغدة الدرقية أحد العوامل الرئيسية المتحكمة في استتباب التمثيل الغذائي. وغالبًا ما ترتبط التغيرات في استهلاك الطاقة واستقلاب الدهون، وهما جانبان مهمان من متلازمة التمثيل الغذائي، بقصور الغدة الدرقية. **العينات وطرق العمل:** شملت الدراسة 80 مشاركًا، منهم 20 شخصًا سليمًا و60 مريضًا يعانون من قصور الغدة الدرقية الأولي. تم تقييم هرمونات الغدة الدرقية (TSH، T3، وT4) ومؤشرات التمثيل الغذائي (سكر الدم الصائم، الكوليسترول الكلي، الدهون الثلاثية، البروتين الدهني عالي الكثافة، البروتين الدهني منخفض الكثافة، والبروتين الدهني منخفض الكثافة جدًا). وللمقارنة بين المجموعتين وتقييم أهمية التغيرات الأيضية، أُجري تحليل إحصائي.

**النتائج:** أظهرت النتائج زيادة كبيرة في مؤشر كتلة الجسم ومستويات هرمون TSH لدى المرضى مقارنةً بالمجموعة الضابطة ( $p < 0.01$ ). وفيما يتعلق بالدهون، لوحظ ارتفاع ملحوظ وكبير في مستويات الدهون الثلاثية والبروتين الدهني منخفض الكثافة جدًا ( $p < 0.0001$ ). مع ذلك، لم تُظهر مستويات الكوليسترول الكلي، والكوليسترول الضار (LDL)، والكوليسترول النافع (HDL)، وسكر الدم الصائم (FBS) فروقًا ذات دلالة إحصائية بين المجموعتين ( $p > 0.05$ ).

**الاستنتاج:** تُشير هذه النتائج إلى أن قصور الغدة الدرقية الأولي يؤدي إلى اضطراب أيضي "مهيمن عليه اضطراب الدهون"، يتميز بارتفاع مُعزل في مستوى الدهون الثلاثية وزيادة في مؤشر كتلة الجسم. ويُشير استقرار مؤشرات مستوى السكر في الدم (FBS) إلى أن تغيرات الدهون تسبق اضطراب مستوى الجلوكوز في المراحل المبكرة من خلل وظائف الغدة الدرقية. يُوصى بإجراء تقييم روتيني للدهون الثلاثية للكشف المبكر عن المخاطر الأيضية لدى مرضى قصور الغدة الدرقية.

## 1. Introduction

Obesity is a chronic disease with multiple contributing factors, arising from intricate molecular and genetic, environmental, in addition to behavioral interactions. Its prevalence is increasing globally, impacting on metabolic, cardiovascular oncological, hepatic, respiratory, in addition to skeletal health. Furthermore Caloric excess, inherited genetic susceptibility, epigenetic alterations, dysbiosis of gut microbiota, endocrine disruptors, circadian misalignment, and intergenerational and prenatal factors are essential determinants of obesity risk. (Młynarska et al., 2025) Excess weight gain is usually unintentional and progressive, and reversing it often requires an individual's commitment and determination (Nojilana et al., 2014) Obesity is recognized as an important public health issue and currently poses a challenge to medical science. It is a chronic non-communicable disease (NCD) and counts as the fifth leading cause of global mortality. (Mahdavi et al., 2021) The condition known as the “New World Syndrome” reduces life expectancy (Nammi et al., 2004) About 25% of people around the world are considered overweight or obese. By 2050, above than half of the anticipated adult population, or 3.8 billion people, will be overweight or obese. The growth in obesity is linked to an alarming rise in non-communicable diseases, such as T2D, heart disease, also many malignancies. These illnesses kill over 5 million people each year. (Ahmed and Mohammed, 2025) Obesity is closely associated with insulin resistance due to excess adipose tissue, which impairs insulin activity and contributes to metabolic disorders. A flaw in the target tissues' biochemical reaction to insulin stimulation is referred to as insulin resistance. (Seong et al., 2019) (Brown, Harhay and Harhay, 2017) (Nolan and Prentki, 2019) (Deacon, 2019) (Thomas et al., 2019) Insulin resistance is a key pathogenic factor contributing to numerous metabolic diseases, including type 2 diabetes mellitus, It is marked by decreased insulin response, Target tissues exhibit typical physiological levels of insulin, Although the precise mechanisms responsible for insulin resistance remain incompletely understood, several hypotheses have been proposed. The mechanisms involve ectopic lipid deposition in the liver and skeletal muscle, endoplasmic reticulum stress, and chronic inflammatory responses. Understanding these mechanisms may contribute to the development of potential therapeutic approaches aimed at improving insulin sensitivity. (Lee, Park and Choi, 2022) Patients with obesity commonly exhibit elevated fasting plasma triglycerides, increased LDL cholesterol levels, reduced HDL cholesterol levels, for example insulin concentrations and higher blood glucose, as well to increased blood pressure (Klop, Elte and Cabezas, 2013) An essential connection among metabolic syndrome, obesity in addition to dyslipidemia is the onset of insulin resistance in peripheral tissues, resulting in a heightened hepatic influx from dietary sources of fatty acids. Intravascular lipolysis transpires and adipose tissue develops resistance to the antilipolytic effects of insulin. (Klop, Elte and Cabezas, 2013) Elevated TRL (triglyceride-rich lipoproteins) enhances HDL (high-density lipoprotein) clearance and promotes the development of tiny, dense LDL (low-density lipoprotein) particles. (Klop, Elte and Cabezas, 2013) (Franssen et al., 2011) Losing weight reduces the severity of dyslipidaemia in individuals with insulin resistance with lifestyle modifications, therapy, and bariatric surgery have demonstrated efficacy in decreasing plasma triglycerides and elevating plasma HDL levels. (Chen and Wang, 2024). (Dash, Xiao and Lewis, 2016) However, there is no conclusive evidence that it inhibits atherosclerosis. (Klop, Elte and Cabezas, 2013) Type 2 diabetes (T2D) is defined by insulin resistance (IR) and constitutes one element of metabolic syndrome (MS), The pathogenesis of Type 2 Diabetes (T2D) involves insulin resistance (IR) and  $\beta$ -cell dysfunction, which play essential roles in its pathophysiology. hyperglycemia and Dyslipidemia, beside with added to metabolic diseases, lead to insulin resistance as well as islet  $\beta$ -cell dysfunction through common mechanisms, involving inflammation and oxidative stress. (Lu et al., 2024) Type 2 diabetes often leads to other components of metabolic syndrome, such as

dyslipidemia. Patients with obesity frequently elevated fasting plasma triglycerides, high LDL cholesterol, low HDL cholesterol, increased blood glucose and insulin levels and high blood pressure. Dyslipidaemia (Semple et al., 2009) Hepatic lipogenesis and synthesis of triglyceride-rich lipoprotein particles (TRL) from the liver have risen. TRL contribute to increased HDL clearance and the development of tiny, dense LDL particles. Both TRL remnants and tiny, dense LDL likely possess atherogenic characteristics. (Ikramuddin et al., 2013) Weight deficiency improves dyslipidaemia in individuals with insulin resistance. Pharmacological treatment, Lifestyle modifications and bariatric surgery have demonstrated efficacy in reducing plasma triglycerides and elevating plasma high-density lipoprotein levels. (Dash and Leiter, 2019) (Pi-Sunyer et al., 2015) (Johnston, Moreno and Foreyt, 2014) Thyroid hormones are well-known for their role in controlling metabolic rate, growth, in addition to many other physical functions. The hypothalamic-pituitary-thyroid axis, a self-regulating system, includes the thyroid gland, the anterior pituitary gland, in addition to the hypothalamus. The principal hormones produced by the thyroid gland are triiodothyronine and thyroxine, or tetraiodothyronine, Thyrotropin-releasing hormone from the hypothalamus, thyroid-stimulating hormone from the anterior pituitary gland, and T4 work synergistically on the way to regulate input pathways and maintain homeostasis. Hypothyroidism, caused by a thyroid gland that does not function properly, typically manifests as weight gain, bradycardia, cold intolerance, constipation, fatigue. Obesity has been associated with an increased risk of overt hypothyroidism (Mahdavi et al., 2021) Moderately elevated TSH values have been known as in patients with obesity (Reinehr, 2010). This rise is proposed as an adaptive mechanism in obese individuals, reflecting a response of the hypothalamus-pituitary-thyroid axis to weight increase (Santini et al., 2014) TSH raise in obese patients is mainly assigned to leptin, the adipose derived hormone (Iacobellis et al., 2005) Circulating leptin levels are related with the level of general obesity. (Sims et al., 2020) The elevated conversion rate from T4 to T3 in obese patients appears as a protective mechanism that reduces fat accumulation by enhancing energy expenditure. (Sims et al., 2020)

## 2. Materials and Methods

### 2.1. Study Subjects

This research was executed as a cross-sectional case-control study using a cohort of obese persons aged 15 to 65 years. Participants were sourced from Ramadi Teaching Hospital, Fallujah Teaching Hospital, and designated private medical facilities. The study population comprised two groups: obese patients with thyroid dysfunction (n=60) and a control group of overweight adults with normal thyroid function (n=20). All participants lacked a previous history of chronic inflammatory or autoimmune disorders.

### 2.2. Anthropometric Measurements

Body weight and height were measured for all participants using standard procedures. Body mass index was evaluated according to the following method:  $BMI = \text{Body weight (kg)} / \text{Height}^2 (\text{m}^2)$  Obesity was defined as a  $BMI \geq 30 \text{ kg/m}^2$  according to World Health Organization criteria.

### 2.3. Biochemical and Hormonal Measurements

Thyroid function was calculated by means of evaluating serum levels of thyroid-stimulating hormone TSH; reference range: 0.4–4  $\mu\text{IU/ml}$ ), triiodothyronine (T3; 1.2–2.9  $\text{nmol/L}$ ), and thyroxine (T4; 3.4–8.4  $\mu\text{g/dl}$  for males and 3.7–9  $\mu\text{g/dl}$  for females). Lipid profile analysis included total cholesterol ( $<200 \text{ mg/dL}$ ), triglycerides (35–150  $\text{mg/dL}$ ), and high-density lipoprotein cholesterol (HDL-C), where levels  $<40 \text{ mg/dL}$  were considered a cardiovascular risk factor and levels  $>60 \text{ mg/dL}$  were considered protective. Glycemic status was assessed by measuring fasting blood glucose (74–106  $\text{mg/dL}$ ). All laboratory analyses were performed according to standard laboratory protocols

### 3. Statistical Analysis

Statistical analysis was performed using standard statistical methods and Microsoft Office programs. Data organization and preliminary arrangement were carried out using Microsoft Excel and Word on Windows 10 Professional. Statistical analyses were performed utilizing GraphPad Prism software version 9.5.1 (GraphPad Software, LLC, CA, USA). comparisons between patients and control groups were performed in alignment with the study objectives. Continuous variables are expressed as mean  $\pm$  standard deviation. (Mean  $\pm$  SD), although categorical variables were presented as frequencies and percentages. Group comparisons for continuous variables were analyzed using the nonparametric Mann–Whitney U test, whereas categorical variables were compared using the Chi-square ( $\chi^2$ ) test. The level of statistical significance was set at  $p < 0.05$ .

### 4. Results

#### 4.1. Demographic and Anthropometric Characteristics

As shown in Table1 the basic characteristics of the study participants. Analysis revealed no statistically significant difference in age distribution between the patient group and the control group ( $p > 0.05$ ), confirming that both groups were well-matched. However, a statistically significant rise in body mass index (BMI) was observed in the patient group compared to the control group  $33.86 \pm 30.95$  vs  $28.90 \pm 1.25$ ,  $p = 0.0040$ .

**Table1: Demographic and Anthropometric Characteristics of the Study Groups**

| Parameters                                                                                                                         | patients(n=60)    | control(n=20)    | P-value |
|------------------------------------------------------------------------------------------------------------------------------------|-------------------|------------------|---------|
| Age(years)                                                                                                                         | 24(40%)           | 7(35%)           | 0.8     |
| Bmi (kg/ m <sup>2</sup> )                                                                                                          | $33.86 \pm 30.95$ | $28.90 \pm 1.25$ | 0.0040  |
| Data are presented as Mean $\pm$ SD for BMI and as Count (Percentage) for Age. $p < 0.05$ is considered statistically significant. |                   |                  |         |

#### 4.2. Thyroid Function Tests

As shown in Table2, thyroid hormone analyses revealed a significant increase in TSH levels in the patient group ( $12.61 \pm 9.26$ ) compared to the control group ( $3.99 \pm 0.55$ ), with a  $p$ -value  $< 0.0001$ . T4 and T3 levels were lower in the patient group ( $5.65 \pm 3.82$  and  $1.09 \pm 0.78$ ) compared to the control group ( $7.65 \pm 0.92$  and  $1.10 \pm 0.14$ ), with a  $p$ -value  $= 0.0001$  for T4 and  $0.0216$  for T3.

**Table2: Comparison of Thyroid Hormone Levels Between Patient and Control Groups**

| Parameters                                                                                               | Patients(n=60)   | Control(n=20)   | P value    |
|----------------------------------------------------------------------------------------------------------|------------------|-----------------|------------|
| Thyroid-stimulation hormone (Tsh )<br>(mIU/L)                                                            | $12.61 \pm 9.26$ | $3.99 \pm 0.55$ | $< 0.0001$ |
| Triiodothyronine (T3)<br>(nmol/L)                                                                        | $1.09 \pm 0.78$  | $1.10 \pm 0.14$ | 0.0216     |
| Thyroxin T4)<br>(nmol/L)                                                                                 | $5.65 \pm 3.82$  | $7.65 \pm 0.92$ | 0.0001     |
| Data Are Presented as Mean $\pm$ SD for TSH, T4, T3, $P < 0.05$ Is Considered Statistically Significant. |                  |                 |            |

#### 4.3. Lipid Profile Characteristics

The blood lipid levels showed in Table3 a difference between the two groups. A statistically significant increase in triglyceride and very low-density lipoprotein (VLDL) cholesterol was observed in the patient group compared to the control group ( $p < 0.0001$ ). However, not at all statistically significant differences were observed between the two groups regarding total cholesterol, high-density lipoprotein, and low-density lipoprotein cholesterol ( $p > 0.05$ ).

**Table3: Comparison of Lipid Profile Parameters Between Patient and Control Groups**

| Parameters                                                                                          | Patients Group (Mean±Sd) | Control Group (Mean ± Sd) | P value |
|-----------------------------------------------------------------------------------------------------|--------------------------|---------------------------|---------|
| Total Cholesterol (CHOL)(mg/dL)                                                                     | 164.6±51.2               | 157.5±18.6                | 0.5240  |
| Triglycerides (TGs) (mg/dL)                                                                         | 149.9±60.7               | 98.3±27.4                 | 0.0001  |
| HDL-Cholesterol (HDL) (mg/dL)                                                                       | 35.2±7.2                 | 37.0±7.0                  | 0.5495  |
| LDL-Cholesterol (LDL) (mg/dL)                                                                       | 99.3±48.2                | 100.7±17.1                | 0.4250  |
| VLDL-Cholesterol (VLDL) (mg/dL)                                                                     | 29.9±12.1                | 19.6±5.4                  | 0.0001  |
| Ratio (LDL/ HDL) Cholesterol                                                                        | 2.9±1.5                  | 2.8±0.7                   | 0.8795  |
| Data Are Presented as Mean ± SD For Lipid Profile, P < 0.05 Is Considered Statistically Significant |                          |                           |         |

#### 4.4.Glycemic Status and Insulin Resistance Assessment

Table4 shows that while the fasting mean blood glucose levels were slightly higher in the patient group ( $106.9 \pm 53.83$ ) compared to the control group ( $95.4 \pm 50.5$ ), this difference was not statistically significant ( $p = 0.05$ ). Furthermore, the HOMA-I index, which reflects insulin resistance, was not statistically significant between the two groups ( $1.02 \pm 0.59$ ) for the patient group vs.  $0.99 \pm 0.23$  for controls;  $p = 0.0747$ ).

**Table4: Comparison of Glycemic Parameters and Insulin Resistance Between Study Groups**

| Parameters                                                                                                   | Patients Group (Mean± SD) | Control Group (Mean ± SD) | P value |
|--------------------------------------------------------------------------------------------------------------|---------------------------|---------------------------|---------|
| <b>FBG (mg/dL)</b>                                                                                           | 106.9±53.83               | 95.4±5.05                 | 0.0588  |
| <b>HOMA-IR</b>                                                                                               | 1.02±0.59                 | 0.99±0.23                 | 0.0747  |
| Data Are Presented As Mean ± Sd For (Fbs), Also (Homa-Ir ), P < 0.05 Is Considered Statistically Significant |                           |                           |         |

## 5. Discussion

These results of Table1 confirm the prevalence of obesity in the patient group, which is a key component of the metabolic syndrome investigated in this study. This study demonstrated a statistically significant increase in body mass index (BMI) among thyroid patients compared to the control group. This weight gain is a hallmark of hypothyroidism, leading to decreased energy expenditure and a lower basal metabolic rate. These findings are consistent with the recent study by Sun et al. (2024) (Sun, He and Yang, 2024) which demonstrated a significant positive association between elevated TSH levels and increased body fat accumulation, particularly in females. Weight gain is a clinical indicator of hypothyroidism, as both T3 and T4 are essential for maintaining the body's basal metabolic rate. In hypothyroidism, decreased levels of these hormones lead to reduced thermogenesis and oxygen consumption, which in turn promotes energy storage as fat, contributing to obesity. This observation aligns beside the findings of Shams Haider Ali et al. (2024) (Ali et al., no date) who reported a significant correlation between thyroid hormone levels and increased body weight in the studied population. These results of Table2 are indicative of hypothyroidism and demonstrate a statistically significant difference between the two group, this hormonal analysis confirms the diagnosis of primary hypothyroidism. The elevated TSH level is a compensatory response from the pituitary gland to the thyroid gland's inability to produce sufficient T4 in the body. The marked decrease in T3, although less than the P-value of 0.021, indicates that the body is attempting to maintain T3 levels by increasing the conversion of T4 to T3 in peripheral tissues. These findings are in agreement with Stat Pearls (2025) (Eghtedari and Correa, 2026), which describes the physiological mechanisms of TSH as a primary marker for thyroid dysfunction and its compensatory role in primary hypothyroidism. While The result of Table3 indicate that the effect of thyroid dysfunction was more pronounced in this particular study Group on triglyceride-related markers, According to these results, which show a significant increase in triglycerides and VLDL cholesterol with no significant change in total cholesterol and LDL levels, this indicates that the group of patients in this study suffers from early stages of thyroid dysfunction. Because the regulation of triglycerides depends directly on hormonal balance and metabolism, in these early stages of thyroid dysfunction, triglycerides are affected more quickly compared to cholesterol, while changes in LDL and total cholesterol require more time to become evident, as reported by Kebamo et al. (2025) (Kebamo et al., 2025).

## Conclusion

These findings highlight that primary hypothyroidism leads to a "lipid-dominant" metabolic disturbance characterized by isolated hypertriglyceridemia and increased BMI. The stability of glycemic markers (FBS) suggests that lipid alterations precede glucose impairment in the early stages of thyroid dysfunction. Routine assessment of TGs is recommended for early identification of metabolic risk in hypothyroid patients.

## Conflicts of Interest

The authors declare that they have no competing interests.

## Ethical Considerations

The authors declare no conflicts of interest.

The ethics committee of the University of Fallujah, College of Applied Science approved the research protocols on December 28, 2025, under ID number (AS-EC/0008)

## Author Contributions

Doha Chali Fhad: designed the research, collected and analyzed the data, and drafted the manuscript.

Dr.Maysam Najai Ahmed(Supervision) ,Writing \_Review ,Editing.

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