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Metabolic Syndrome and Cardiovascular Risk Profiles in Female Patients with Hashimoto's Thyroiditis

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ABSTRACT

Background: Hashimoto's thyroiditis (HT) is a common autoimmune disease, especially in females. While hypothyroidism is a recognized complication, emerging evidence links HT to metabolic disturbances and increased cardiovascular risk. However, the broader systemic effects in untreated patients remain insufficiently clarified.
Objectives: To assess the metabolic and cardiovascular risk profiles among women with HT, with a special emphasis on lipid parameters, insulin resistance (IR), and inflammation.
Materials and methods: This case-control study included 100 women: 50 patients with HT (25 newly diagnosed and untreated, and 25 receiving thyroid hormone replacement therapy) and 50 age-matched healthy controls. Anthropometric measures, glycemic status, lipid profile, and inflammatory markers were assessed. The evaluated indices included HOMA-IR, triglyceride/HDL-C ratio, LDL-C/HDL-C ratio, Atherogenic Index of Plasma, Lipid Comprehensive Index, and high-sensitivity C-reactive protein.
Results: Patients with HT had significantly greater body mass index, fasting glucose, fasting triglyceride, LDL-C, IR, and markers of inflammation than controls (all P-values < 0.001). Atherogenic risk, as measured by both AIP and LCI, was significantly elevated in greater than 90% of patients with HT, with the highest burden among untreated patients. While thyroid hormone therapy produced a degree of improvement, a number of cardiometabolic risk factors persisted at elevated levels.
Conclusion: Female patients with HT frequently exhibit adverse metabolic and cardiovascular alterations, likely driven by chronic inflammation and thyroid dysfunction. Early screening and comprehensive management are essential, particularly for newly diagnosed and untreated women with HT.
Keywords: Hashimoto thyroiditis, Cardiovascular disease; Insulin resistance; Lipid indices; Metabolic syndrome.

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INTRODUCTION

Hashimoto thyroiditis (HT) is the most common autoimmune thyroid disorder, affecting mostly the females, and is characterized by lymphocytic infiltration and progressive destruction of the thyroid gland leading to hypothyroidism [1, 2]. Although HT is an endocrine disease, it may lead to cardiovascular and metabolic abnormalities, especially in females [2, 3]. Hypothyroidism impairs the basal metabolic rate, glucose regulation, and lipid metabolism, while the chronic inflammatory status of the HT results in insulin resistance, alterations in adipokine signaling, and endothelial dysfunction [4]. Such alterations could be modulated by female-specific hormonal patterns and fat distribution, rendering the development of cardiometabolic alterations more likely [5].

The metabolic syndrome or MetS, which is associated with central adiposity, dyslipidemia, hypertension, and an impaired resistance state to insulin, is a major predictive factor for CVD. In female patients with HT, cases of MTS are often associated with thyroid autoimmunity. Even if thyroid hormone levels are normal, other values relating to body mass index, waist circumference, blood pressure, glucose, and lipids

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are often altered.

In a recent study, emphasis has been given to other markers related to vasculopathies and metabolism, which are applicable to HT cases [6]. Values including HOMA-IR, as an indicator for impaired resistance to insulin, as well as values with a high-sensitivity C-reactive protein, an indicator related to systemic low-grade inflammation, are elevated [7]. Moreover, cardiovascular risk scoring tools include the atherogenic index of plasma (AIP), Low-Density Lipoprotein-to-High-Density Lipoprotein Cholesterol Ratio (LDL/HDL) and Triglyceride-to-High-Density Lipoprotein Cholesterol Ratio (TG/HDL). Despite the growing recognition of the metabolic and cardiovascular consequences of HT, there is still a noticeable lack of region-specific data, particularly concerning affected women in Middle Eastern populations. Most existing studies have not differentiated between newly diagnosed and treated patients, nor have they incorporated a broad panel of metabolic and inflammatory markers [8, 9]. Therefore, the present study aimed to investigate the association between HT and metabolic as well as cardiovascular risk markers in Iraqi women, by evaluating both treated and newly diagnosed patients. The objective was to determine how treatment status and disease stage influence metabolic parameters, inflammatory markers, and cardiovascular risk indices.

MATERIALS AND METHODS

This case-control study was conducted between January and August 2024 at the Endocrinology and Diabetes Center, Al-Sadr Medical City, Najaf, Iraq. Ethical approval was obtained from the Faculty of Science, University of Kufa (Reference number 159; 17-09-2024). Informed consent was obtained from all participants. The required sample size was calculated using G*Power (effect size d = 0.5, $\alpha = 0.05$, power 80%) indicated a minimum of 64 subjects; this was increased to 100 women to allow subgroup analyses. The first group (F-HT) included 50 HT patients, 25 treated (FT-HT), 25 newly diagnosed (FND-HT), and 50 age-matched healthy controls ((F-HC). HT diagnosis was confirmed by elevated TSH, positive anti-TPO antibodies, and supportive ultrasound findings.

Females aged 18-65 years, either newly diagnosed with HT or on levothyroxine, were included. Exclusion criteria were pregnancy, acute or chronic infections, malignancy, hepatic or renal disease, and recent use of immunosuppressive or anti-inflammatory drugs. Anthropometric data (weight, height, waist circumference) were obtained using standard methods. Blood pressure was measured twice at 5-minute intervals, and the average was recorded. Fasting venous blood (3 mL) was collected in plain gel tubes, allowed to clot, centrifuged at 3000 rpm for 10 minutes, and serum samples were stored at -80 °C within one hour. TSH and insulin were measured using Cobas e411, and hs-CRP using Cobas Integra 400 Plus. Fasting glucose, TC, TG, and HDL-C were analyzed with BIOLABO enzymatic kits on a UV-Vis spectrophotometer. Insulin resistance was calculated using the HOMA-IR = (fasting glucose × insulin) / 405. Atherogenic indices included AIP [$\log_{10}$(TG/HDL-C)], TG/HDL-C, LDL/HDL-C, and the Lipid Combination Index [TC × TG × LDL-C / HDL-C]. Body mass index (BMI) was estimated as weight in kilograms divided by height in meters squared. Patients were categorized according to the World Health Organization (WHO) classification into underweight (<18.5 kg/m^2), normal (18.5-24.9 kg/m^2), overweight (25-29.9 kg/m^2), and obesity class I (≥30 kg/m^2). Metabolic syndrome was diagnosed using NCEP-ATP III criteria (NHLBI, 2001), requiring ≥ 3 of the following: Waist ≥ 88 cm, TG ≥150 mg/dL, HDL-C <50 mg/dL, BP ≥ 130/85 mmHg or on antihypertensives, fasting glucose ≥100 mg/dL.

The data were analyzed using GraphPad Prism 9.5. Data normality was tested with the Shapiro-Wilk test. Categorical variables were presented as frequency and percentage in tables or figures. While continuous variables were presented as mean ± SD. Categorical variables were analyzed using a chi-square or Fisher's exact test. Continuous variables were compared using unpaired t-tests or one-way ANOVA with Tukey's post hoc test. A P-value < 0.05 was considered a statistically significant difference.

RESULTS

There is no significant difference (P-value = 0.767) between the mean age of F-HC Group (34.55 ± 2.02 years) and F-HT Group (33.80 ± 1.51 years). In contrast, hypertension was significantly more prevalent in the F-HT group, with 37.25% of patients classified as hypertensive, At the same time none of the F-HC participants had elevated blood pressure (P-value < 0.001). Additionally, the F-HT group showed markedly elevated levels of TSH (2.47 ± 0.10 vs. 10.14 ± 1.83 μIU/mL; P-value < 0.001) and anti-TPO antibodies (150.38 ± 7.39 vs. 439.43 ± 18.69 IU/mL; P-value < 0.001) compared to controls, as shown in Table 1.

Female patients with HT (F-HT) showed a mean BMI of 30.91 ± 0.565 kg/m^2. The distribution of BMI categories was as follows: 66% obese (BMI ≥ 30), 2% underweight, and 2% within the normal range. Central obesity was observed in 60% of patients, with a mean waist circumference of 91.7 ± 1.16 cm. The mean SBP was 144.08 ± 3.10 mmHg, and the mean DBP was 95.7 ± 1.86 mmHg. The average HOMA-IR score was 5.64 ± 0.74, with 42% of patients above 5 and 44% below 2. Mean hs-CRP levels were 2.56 ± 0.07 mg/L; 56% of patients were within the average-risk range, and 24% exceeded 3 mg/L. The mean atherogenic index was 0.79 ± 0.06, with 90% of patients above the high-risk cutoff (> 0.21) as shown in Table 2.

Central obesity (waist circumference ≥ 88 cm) was observed in 60.0% of females with HT (F-HT) compared to 25.9% of female healthy controls (F-HC), a difference that was statistically significant (P-value = 0.0089). A markedly higher proportion of F-HT patients also exhibited elevated

Table 1. Demographic and clinical details of 100 participants*.

Parameter	F-HC Group (n=50)	F-HT Group (n=50)	P-value
Age (years)	34.55 ± 2.02	33.80 ± 1.51	0.767#
Hypertension			
Present	0 (0%)	19 (37.25%)	0.001*
Absent	50 (100%)	32 (62.75%)	
TSH (Mean ± SE)	2.47 ± 0.10	10.139 ± 1.83	0.001#
Anti-TPO(Mean±SE)	150.38 ± 7.39	439.43 ± 18.69	0.001#

* BMI: Body mass index, TSH: Thyroid-stimulating hormone, Anti-TPO: Anti-thyroid peroxidase antibody, #: unpaired Student's *t*-test, *: Fisher's exact; (P-value < 0.05). F-HC = Females healthy controls and F-HT = Females Hashimoto's thyroiditis patients.

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Table 2. Descriptive characteristics and cardio-metabolic risk profiles of 50 female patients with Hashimoto's thyroiditis*.

Variable	Descriptive statistics
BMI (kg/m^2) (group mean)	30.91 ± 0.565
<18.5 (n, %)	1 (2.0%)
18.5–24.9 (n, %)	1 (2.0%)
25.0–29.9 (n, %)	15 (30.0%)
>30.0 (n, %)	33 (66.0%)
WC	91.7 ± 1.160
Central obesity (n, %)	30 (60.0%)
Blood Pressure (group mean)	
SBP (mmHg)	144.08 ± 3.10
DBP (mmHg)	95.7 ± 1.86
HOMA-IR (group mean)	5.64 ± 0.74
No insulin resistance (<2) (n, %)	22 (44.0%)
Mild insulin resistance (2–2.9) (n, %)	1 (2.0%)
Moderate insulin resistance (3–5) (n, %)	6 (12.0%)
Severe insulin resistance (>5) (n, %)	21 (42.0%)
Hs-CRP (group mean)	2.56 ± 0.07
Low risk (<1.0 mg/L) (n, %)	10 (20.0%)
Average risk (1.0–3.0 mg/L) (n, %)	28 (56.0%)
High risk (>3.0 mg/L) (n, %)	12 (24.0%)
Atherogenic Index (group mean)	0.79 ± 0.06
Low risk (<0.11 mg/L) (n, %)	2 (4.0%)
Average risk (0.11–0.21 mg/L) (n, %)	3 (6.0%)
High risk (>0.21 mg/L) (n, %)	45 (90.0%)

* BMI: Body mass index; WC: Waist circumference; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; Hs-CRP: High-sensitivity C-Reactive Protein; SBP: Systolic blood pressure; DBP: Diastolic blood pressure.

Table 3. Distribution of metabolic syndrome among female Hashimoto's patients and female healthy controls*.

Parameter	F-HC, n (%)	F-HT, n (%)	P-value
WC ≥ 88 cm	7 (25.9%)	30 (60.0%)	0.0089
TG ≥ 150 mg/dL	0 (0.0%)	50 (100.0%)	0.001
HDL < 50 mg/dL	15 (55.6%)	39 (78.0%)	0.0731
FBG ≥ 100 mg/dL	1 (3.7%)	26 (52.0%)	0.0001
BP ≥ 130/85 mmHg	0 (0.0%)	45 (90.0%)	0.001
≥ 3 criteria (MetS diagnosed)	1 (3.7%)	46 (92.0%)	0.001

* WC: Waist circumference; TG: Triglycerides; HDL: High-density lipoprotein; FBG: Fasting blood glucose; BP: Blood pressure; MetS: Metabolic syndrome. Chi-square or Fisher's exact test, as appropriate based on expected cell counts. (P-value < 0.05). F-HC = Females healthy controls and F-HT = Females Hashimoto's thyroiditis patients.

Table 4. Cardiovascular risk indicator in 50 female healthy control compared to 50 female Hashimoto's patients*.

Parameter	F-HC (n=50)	F-HT (n=50)	P-value
Triglycerides/HDL	2.04 ± 0.12	9.26 ± 1.14	0.001
LDL/HDL	2.74 ± 1.13	9.51 ± 1.10	0.001
hs-CRP (mg/L)	2.10 ± 0.00	2.56 ± 0.07	0.001
AIP	0.29 ± 0.03	0.79 ± 0.06	0.001
LCI	78047.11 ± 5940.84	1785384.50 ± 339271.19	0.001

* Female healthy control: F-HC, Female Hashimoto's patients: F-HT, Values are expressed as Mean ± Standard error (SE). Triglycerides/HDL: Triglycerides to high-density lipoprotein ratio. LDL/HDL: the ratio of low-density lipoprotein to High-density lipoprotein. hs-CRP: High-sensitivity C-reactive protein. AIP: Atherogenic index. LCI: Lipoprotein combined index.

triglyceride levels (100.0% vs. 0%, P-value < 0.001) and hypertension (90.0% vs. 0%, P-value < 0.001) compared with controls. Impaired fasting glucose (FBG ≥ 100 mg/dL) was significantly more frequent among F-HT cases (52.0%) than in F-HC (3.7%, P-value = 0.0001). Although low HDL-C (<50 mg/dL) was more prevalent in the F-HT group (78.0% vs. 55.6%), this difference did not reach statistical significance (P-value = 0.0731). Notably, the diagnosis of metabolic syndrome (defined as ≥ 3 positive criteria) was confirmed in 92.0% of F-HT patients, compared to only 3.7% of controls, a highly significant difference (P-value < 0.001), as shown in Table 3.

The Triglycerides/HDL-C ratio was 2.04 ± 0.12 in F-HC and 9.26 ± 1.14 in F-HT (P-value =0.001). The LDL/HDL-C ratio was 2.74 ± 1.13 in F-HC and 9.51 ± 1.10 in F-HT (P-value =0.001). The hs-CRP level was 2.10 ± 0.00 mg/L in F-HC and 2.56 ± 0.07 mg/L in F-HT (P<0.001). The AIP was 0.29 ± 0.03 in F-HC and 0.79 ± 0.06 in F-HT (P-value =0.001). The LCI was 78,047.11 ± 5,940.84 in F-HC and 1,785,384.50 ± 339,271.19 in F-HT (P-value =0.001) as illustrated in Table 4.

On average, BMI values were significantly elevated in the F-HT group (31.0 ± 0.66 kg/m^2) compared to the control group (27.3 ± 0.58 kg/m^2), indicating a higher prevalence of overweight or obesity in the affected individuals. Similarly, total cholesterol was markedly increased in F-HT patients (365.7 ± 7.76 mg/dL) relative to the control group (197.2 ± 4.19 mg/dL), accompanied by a pronounced rise in fasting blood glucose levels (135.2 ± 2.87 mg/dL vs. 77.6 ± 1.64 mg/dL). Lipid fraction analysis revealed further dysregulation, with HDL levels significantly reduced in the F-HT group (37.6 ± 0.80 mg/dL) compared to controls (48.3 ± 1.02 mg/dL), in comparison both LDL (275.1 ± 5.84 mg/dL vs. 129.5 ± 2.74 mg/dL) and triglycerides (265.9 ± 5.64 mg/dL vs. 97.2 ± 2.07 mg/dL) were substantially elevated (Figure 1).

BMI was significantly higher in both FT-HT (P-value = 0.0013) and FND-HT (P-value = 0.0029) compared to F-HC, with no significant difference between FT-HT and FND-HT (P-value = 0.9938). Cholesterol levels were significantly increased in FT-HT (P-value = 0.0142) and FND-HT (P-value < 0.0001) relative to F-HC, and also significantly higher in FND-HT than in FT-HT (P-value = 0.0003). Fasting blood glucose although significantly elevated in FT-HT (P-value = 0.0202) and FND-HT (P-value < 0.0001) versus F-HC, while the difference between FT-HT and FND-HT was not significant (P-value = 0.0872). HDL was significantly lower in FT-HT (P-value = 0.0040) and FND-HT (P-value = 0.0022) compared to F-HC, with no significant difference between FT-HT and FND-HT (P-value = 0.9966). LDL levels were significantly increased in FT-HT (P-value = 0.0041) and FND-HT (P-value < 0.0001) compared to FHC, with a significant difference between FT-HT and FND-HT (P-value = 0.0083).

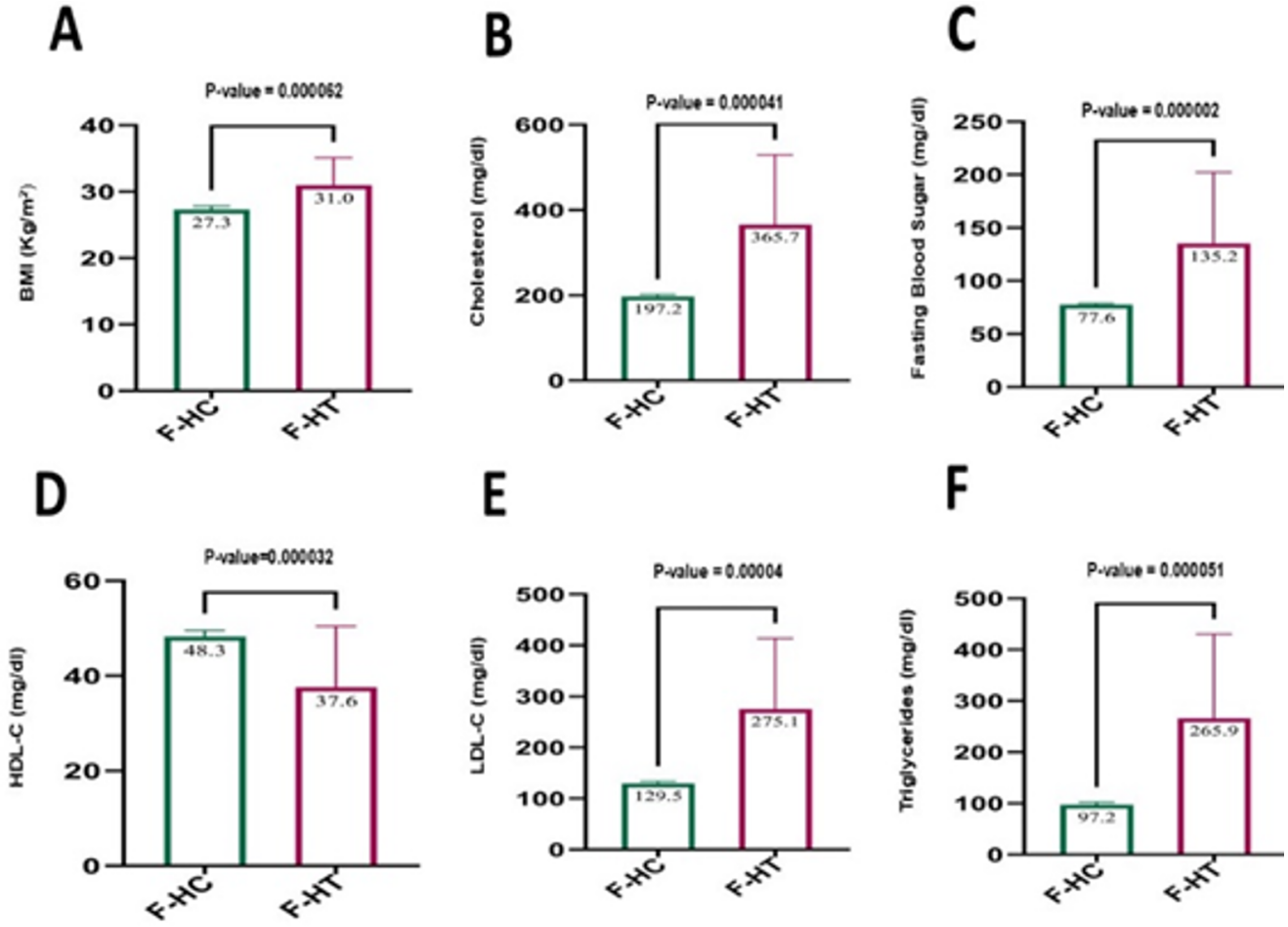

Figure 1. Comparison of metabolic parameters in 50 female healthy controls (F-HC) compared to 50 female Hashimoto's patients (F-HT). Values are expressed as Mean ± Standard error (SE), unpaired Student's *t*-test, LDL: Low-density lipoprotein, HDL: High-density lipoprotein ratio, BMI: Body mass index.

Triglyceride levels were significantly higher in FT-HT (P-value = 0.0068) and FND-HT (P-value < 0.0001) compared to FHC, and also significantly elevated in FND-HT relative to FT-HT (P-value = 0.0050) as indicated in Figure 2.

DISCUSSION

HT has implications beyond thyroid disease, as recent studies have indicated the metabolic and vascular impacts of HT. The current study identified a remarkable change in the values of metabolic and cardiovascular risk factors among Iraqi female patients with Hashimoto's thyroiditis, making early evaluation of metabolism a strength and a crucial point for their prevention and management.

Hypertension is significantly higher among F-HT patients than controls, consistent with past observations showing high blood pressure among HT patients irrespective of age distribution, largely attributed to the hemodynamic effects resulting from thyroid hormone depletion and the autoimmune process [10, 11]. Also, the results seen today, including the elevation in levels of both TSH and anti-TPO among F-HT patients, sufficiently show active autoimmune thyroid dysfunction, consistent with past findings [1, 12]. High levels of TSH point towards a condition of hypothyroidism that is well-known to reduce endothelial functions as well as enhance cardiac workload [13]. The high levels of anti-TPO, as mentioned, strengthen the mechanism involving immunoregulatory disturbances as seen among F-HT patients, relevant to its metabolic manifestations [14].

A total of 66% among the HT patients were obese, while 60% presented with central obesity, indicating a lower BMR and an elevated amount of VAT. VAT makes a substantial contribution to the imbalance between the immune system and the vasculature through the secretion of various adipokines, including leptin and resistin, as mentioned by various authors [15]. Higher systolic as well as diastolic blood pressure was a characteristic among the patients, likely due to the intolerance of nitric oxide levels, as well as the resultant chronic inflammation as proposed by other authors [10]. Central obesity could further worsen this condition, as indicated by the elevated BMI, particularly among the population with IRS, as was reported by other authors, referring to various studies published elsewhere [13, 16]. The prevalence among the F-HT was observed to be greater than 40%, while HOMA-IR values were greater than 5, as mentioned by various authors, indicating a defect in the signaling pathways involving the insulin receptors, alongside a reduced number of GLUT4

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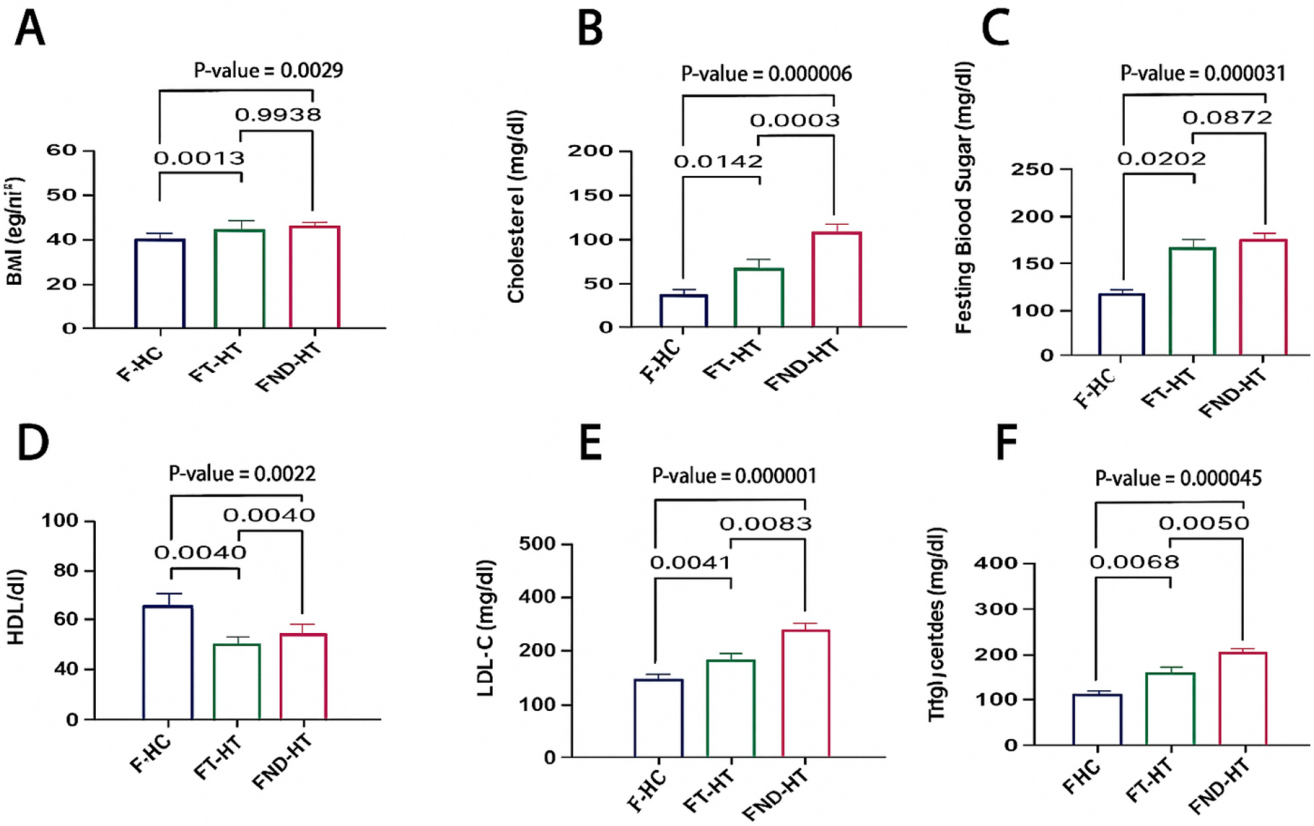

Figure 2. Comparative analysis of metabolic parameters among 50 healthy female controls (F-HC), 25 treated patients with Hashimoto's thyroiditis (FT-HT), and 25 newly diagnosed untreated patients (FND-HT). ANOVA test, LDL: Low-density lipoprotein, HDL: High-density lipoprotein ratio, BMI: Body mass index. P: P-value.

receptors, as well as a reduced amount of thyroid hormone as discussed by other authors [17]. Additionally, a level greater than 3 mg/L among patients was observed, as mentioned by other authors, reinforcing a condition among patients characterized by low-grade chronic inflammation as highlighted by the elevated levels of other inflammatory markers, as proposed by other authors [18]. A total number, around 25% among the patients, indicated an elevated level greater than 3 mg/L, as mentioned by other authors, implying a condition among patients characterized by chronic low-grade inflammation as proposed by other authors due to alterations presented by the secretory functions among adipokines, as indicated by other authors due to the activities involving the number among the immune cells present among the adipose tissues as mentioned by other authors [19].

The condition seen in this study, dyslipidemia manifesting as high levels of triglycerides, low HDL-C, and high LDL-C, in female members of the F-HT group, could be related to reduced thyroid hormone function, which affects lipids metabolism [20]. Hypothyroid conditions are characterized by reduced thyroid functions, low expression of hepatic LDL receptors, and reduced activities of lipoprotein lipase, leading to reduced removal rates of low-density lipoproteins and an accumulation of lipids present as triglycerides-rich lipoproteins, respectively. Additionally, low activities associated with hepatic lipase and cholesteryl ester transfer protein are indicated as potential causes leading to reduced HDL-C levels seen in hypothyroidism conditions [21]. Hypothyroid condition has been indicated to significantly increase total cholesterol, LDL-C, and levels of triglycerides in various populations [21], while HDL-C is lower among hypothyroids than people with normal thyroid functions [22].

Comparison between patients with treatment and those newly diagnosed showed that treatment status may have an effect on the degree of the thyroid condition impacting metabolism. Both groups had a significantly increased BMI when compared with normal participants, implying that the condition of being overweight remained unaffected by treatment. This is supported by other studies showing that the high prevalence of obesity among HT patients may be attributed to hormone deficiency as well as other factors, including energy expenditure, which may remain unaffected even with levothyroxine replacement therapy [23].

Fasting blood sugar was significantly increased in both subgroups of HT patients compared with controls, although the difference between the two subgroups was nonsignificant. The results are consistent with the hypothesis that impaired sugar metabolism continues in HT, likely as a result of the presence of IR and inflammatory cytokines. The values of HDL-C were significantly lower in both groups, and there was no difference between the two subgroups, indicating an impaired RCT system, which is independent of HT treatment [24].

In our research, the indices of cardiovascular risk were significantly higher among female patients with HT than among healthy controls. The ratio of triglycerides to HDL-C, a recognized indicator of IR and atherogenic dyslipidemia, was

significantly elevated among F-HT patients. Such an increase may imply an impairment in lipoprotein metabolism due to the consequent deficiency of thyroid hormones and chronic immuno-inflammatory processes, both known to influence liver handling of lipids and HDL turnover identically; the ratio of LDL to HDL-C, a prognostic indicator of atherogenesis, was greater among F-HT patients. It suggests an elevated production or an impaired clearance of this atherogenic lipoprotein subclass, likely due to the reduced thyroid hormone stimulation of liver LDL receptors [25]. The atherogenic index of plasma, a logarithmical summation index including both levels of triglycerides and HDL-C, was significantly greater among F-HT patients, implying an atherogenic lipoprotein profile [26]. AIP is regarded as a reliable indicator of small, dense LDL, which has a greater susceptibility to oxidative modifications as well as to Arterial deposition [27].

The limitations to the study included single-center setting, enrollment of only female participants which might restrict the generalizability of the results, relatively small sample size which might limit the sensitivity of the statistical tests, and certain parameters such as treatment duration and disease variability, were not taken into consideration. However, the current investigation offers critical information amidst limited regional publications.

CONCLUSION

Women with HT, even in early phases (especially newly diagnosed/untreated), showed metabolic and cardiovascular alterations (IR, dyslipidemia, inflammation). Treatment partially improved parameters but did not normalize all risk factors, underscoring the need for early monitoring and intervention.

ETHICAL DECLARATIONS

Acknowledgments

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Ethics Approval and Consent to Participate

This study was reviewed and approved by the Research Ethics Committee at the University of Kufa, College of Science. All participants were informed about the study objectives and procedures, and informed consent was obtained prior to enrollment.

Consent for Publication

Not applicable.

Availability of Data and Material

Data generated during this study are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that there is no conflict of interest.

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Use of Artificial Intelligence

The authors used artificial intelligence for minor language editing; all scientific content and interpretation were done by the author.

Authors' Contributions

Aziz DZ structured the concept and provided guidance in study design and methodology. Alkhazrajy NS selected the study materials, performed the laboratory work, collected the data, carried out the analysis and interpretation of data, conducted the literature search, and prepared the initial draft of the manuscript. Aziz DZ conducted the critical review of the manuscript. Both authors read and approved the final version of the manuscript.

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