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Original article

A cross-sectional study on fetuin-A, high-sensitivity CRP, and cardiovascular risk in female patients with comorbid type 2 diabetes and hypothyroidism

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Abstract

Background: There are growing body of evidence indicating coexistence of hypothyroidism and type 2 diabetes in women despite its prevalence, and it reveals a synergistical adverse consequence of metabolic disturbances.

Objective: Inflammatory biomarkers (fetuin-A and high-sensitivity C-reactive protein [hs-CRP]) were investigated to explore the crosstalk between inflammation and metabolic disturbances in different groups.

Methods: Female participants were divided into four groups: type 2 diabetes (T2D), hypothyroidism, coexistence (T2D and hypothyroidism), and healthy control group. HbA1c and lipid profile were measured, along with various lipid indices including fetuin-A and hs-CRP using an enzyme-linked immunosorbent assay.

Results: In the coexistence group, hs-CRP and fetuin-A were significantly higher (7.1 ± 4.9 mg/L, 712.4 ± 231.5 µg/mL) than in the T2D (5.8 ± 4.1 mg/L, 443.6 ± 198.3 µg/mL) and hypothyroidism groups (3.2 ± 2.8 mg/L, 228.5 ± 112.7 µg/mL; $P < 0.001$). There was a significant association between inflammatory biomarkers and dyslipidemia indices (fetuin-A and HbA1c; $r = 0.44$, $P < 0.01$) and the level of atherogenity (Atherogenic Index of Plasma: $r = 0.47$, $P < 0.001$) in dual pathology groups.

Conclusion: The coexistence of diabetes and hypothyroidism demonstrated a synergistic effect on systemic inflammation and metabolic disturbances in these women. Furthermore, the strong association of inflammation and glycemia and triglyceride-rich lipoprotein levels suggests that relying solely on traditional cholesterol-rich lipoproteins is not sufficient for assessing atherogenicity. This highlights the need for more modifiable lipid index measures.

Keywords: Diabetes and hypothyroidism, fetuin-A, high-sensitivity CRP, lipid profile indices.

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The clinical impacts of type 2 diabetes mellitus (T2D) and hypothyroidism are increasing due to their economic burden and adverse health outcomes. The increasing risk of cardiovascular disease associated with dyslipidemia and low-grade inflammation is a hallmark of both conditions. [1] There are emerging evidences that the coexistence of both conditions is common but the consequences shown to synergistical adverse effects of metabolic disturbances.

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The main metabolic disturbances of T2D are chronic hyperglycemia and insulin resistance; however, dyslipidemia and low-grade inflammation are usually associated with the disease. In addition, inflammation and dyslipidemia are common in patients with hypothyroidism. [2] Despite their frequent co-occurrence, the consequences on lipid profile and atherogenic risk profile, inflammation, and chemokine activity are still poorly understood. Earlier studies observed a potential connection between diabetic metabolic abnormalities and thyroid dysfunction. [3] Furthermore, patients with hypothyroidism often show severe insulin resistance via reduced thermogenesis and abnormal protein synthesis. [4] In addition, hyperglycemia can impair thyroid hormone synthesis and function as well as peripheral T4 to T3 conversion through metabolic dysfunction. [5]

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There is an increased risk of atherosclerosis and incidence of cardiovascular events in both conditions, including high triglycerides (TG), low high-density lipoprotein cholesterol (HDL-C), and elevated low-density lipoprotein (LDL). Furthermore, various lipid profile indices enhance diagnosis and aid early risk detection. For example, the Atherogenic Index of Plasma (AIP), calculated as log(TG/HDL-C), has proven to be a powerful cardiovascular risk marker especially in insulin-resistant conditions and hypothyroidism. [6] In addition, use of high-sensitivity C-reactive protein (hs-CRP) to evaluate low-grade inflammation and use of fetuin-A hepatocyte-derived protein as biomarkers might be associated with metabolic disturbances of these conditions. [7]

Nonetheless, population-specific studies are still infrequent, specifically in Middle East populations with dietary, socioeconomic, and cultural factors that might worsen cardiovascular risk. For instance, Iraqi women are increasingly vulnerable to both conditions. This is compounded by rising obesity rates and inadequate preventive and primary care, further increasing their risk of developing cardiovascular disease. [8, 9] This study investigates the risk of cardiovascular events by analyzing various lipid profile markers and indices in different groups of Iraqi women with metabolic disturbances. These include T2D, hypothyroidism, and the simultaneous presence of both conditions. The study also examines the interaction between fetuin-A and hs-CRP in this population. Ultimately, this research aims to deepen our understanding of metabolic disturbances associated with endocrine conditions, ultimately leading to improved personalized risk assessment and treatment strategies.

Materials and methods

This was a cross-sectional study including four distinct groups of Iraqi women recruited during routine visits to specialized endocrine clinic in of Baquba Teaching Hospital, Dyala, Iraq, from October 2020 to March 2021.

- T2D group (n = 45): known history of diabetes diagnosed according to WHO criteria (FBS ≥ 126 mg/dL, random blood sugar (BS) ≥ 200 mg/dL and hemoglobin A1c (HbA1c) ≥ 6.5%) with 2–10 years of disease
- Hypothyroidism group (n = 45): women newly diagnosed with overt hypothyroidism diagnosed with thyroid-stimulating hormone (TSH) > 4.5 μIU/mL and free- thyroxine4 < 0.8 ng/dL
- T2D + hypothyroidism group (n = 45): women with previously established T2D for 2–10 years diagnosed with new hypothyroidism meeting the above diagnostic criteria
- Control group (n = 45): body mass index (BMI) /age matched healthy women with normal thyroid and diabetic profiles

The study employed a convenience sampling method to accommodate the time constraints and desired outcomes. However, caution is needed in comparing results to the general population. Therefore, the results of the current study are more representative to our cohort and could build a base for future, wide-ranging research.

Exclusion criteria included acute illness, thyroidectomy, pregnancy, or known renal, heart, and chronic inflammatory conditions. The study was approved by the scientific committee of the Department of Chemistry and Biochemistry, College of Medicine, Mustansiriyah University, according to national and/or international rules (UOM-COM-2020-135).

Blood analysis

Blood samples were obtained in a fasting state after taking verbal consent. An anti-coagulated tube was used for whole blood HbA1c measurements (SD A1cCareTM system, Biosensor, South Korea). Serum was collected for the remaining measures (fasting blood sugar (FBG), total cholesterol (TC), triglycerides (TGL), LDL, and HDL) spectrophotometrically by Biolabo, France. Fetuin-A was measured using enzyme-linked immune sorbent assay (ELISA) kits (Abcam company, UK, as ab108855-Fetuin-A). hs-CRP was measured using ELISA kits (Catalog No: MBS2506093-96T, from MyBiosource, San Diego, USA). Thyroid panels from bioMérieux, TSH, and FT4 tests were measured by Enzyme Linked Fluorescent Assay technique. All biochemical variables were measured using a standardized commercial kit, and quality control was strictly performed according to the manufacturer's instructions. BMI was calculated as weight (kg) / square height (m^2).

Lipid profile indices

Four lipid profile indices that had been shown in the scientific literature to more accurately predict the risk of atherogenicity than traditional lipid parameters were selected.

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AIP: Log (Triglycerides / HDL-Cholesterol).
Castelli Risk Index I (CRI-I): Total Cholesterol / HDL-Cholesterol.
Castelli Risk Index II (CRI-II): LDL-Cholesterol / HDL-Cholesterol.
Triglycerides/HDL-Cholesterol Ratio (TG/HDL-C): Triglycerides / HDL-Cholesterol.

Statistical analysis

Statistical data were analyzed using Graph Pad Prism 8.0.2 (263). Data are expressed as a mean ± standard deviation (SD), after testing data normality. One-way analysis of variance (ANOVA) with Tukey's post hoc test (parametric data) or Kruskal–Wallis test with Dunn's correction (non-parametric data) were used for comparisons. Correlation analysis was conducted using Pearson's correlation with a *t*-test. Statistical significance was considered at $P < 0.05$.

Results

An ANOVA test revealed a significant difference in traditional lipid profile measures and calculated lipid indices among the study groups. Furthermore, the inflammatory state assessed via hs-CRP showed significant differences in means between the groups, with appropriate post hoc tests confirming this. Notably, hs-CRP was highly significant in T2D patients with coexisting hypothyroidism compared to those without ($P < 0.01$), compared to the hypothyroid group ($P < 0.05$), and compared to the control healthy group ($P < 0.001$), while in differences observed comparing T2D to hypothyroid groups, however, both of them were higher than the control group. thyroid function profile results were significantly different among groups ($P < 0.001$). Free T4 levels in patients with T2D with coexisting hypothyroidism were significantly lower in those with hypothyroidism alone ($P < 0.001$); however, TSH results showed the opposite trend. Regarding BMI, the coexistence group showed significantly higher BMI than the T2D group ($P < 0.05$) and control group ($P < 0.001$); there were no differences in BMI in comparison to the hypothyroid group (**Table 1)**.

Table 2 shows correlation analysis regarding fetuin-A and hs-CRP. Traditional lipid profile results revealed a significant positive correlation between TG and both fetuin-A and hs-CRP in the coexistence group (T2D and hypothyroidism; $P < 0.001$). A significant negative strong correlation was observed with HDL-cholesterol especially in the coexistence group for hs-CRP ($r = -0.47$; $P < 0.001$) and for fetuin-A ($r = -0.43$; $P < 0.01$). No significant correlation was observed between total cholesterol and LDL.

Figure 1 shows mean ± SD of fetuin-A and hs-CRP in different study groups. Fetuin-A levels were significantly higher in coexistence group (712.4 ± 231.5 µg/mL) than in T2D (443.6 ± 198.3 µg/mL) and control (298.7 ± 97.4 µg/mL; $P < 0.001$) groups, while hypothyroidism group showed the lowest value among groups (228.5 ± 112.7 µg/mL).

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Table 1. Biochemical and BMI results of study groups.

Parameter	Diabetic	Hypothyroid	Diabetic + Hypothyroid	Control	ANOVA *P*-value	Post-hoc significance
TG (mg/dL)	261.9 ± 94.3	239.6 ± 108.2	289.1 ± 117.5	161.2 ± 22.1	<0.001	D+H > D, *H* > C***
TC (mg/dL)	258.6 ± 76.2	276.8 ± 62.4	292.5 ± 59.8	176.8 ± 24.7	<0.001	D+H, H > D* > C***
LDL (mg/dL)	152.4 ± 65.3	142.7 ± 53.1	129.8 ± 47.6	91.2 ± 23.4	<0.001	D, H > C***
HDL (mg/dL)	30.7 ± 9.8	35.2 ± 13.6	30.1 ± 10.3	43.5 ± 10.9	<0.001	C > H* > D/D+H***
AIP	1.0 ± 0.2	0.8 ± 0.3	1.0 ± 0.2	0.6 ± 0.1	<0.001	D+H > D** > H* > C***
CRI-I	9.8 ± 3.7	8.1 ± 3.9	11.2 ± 4.5	4.3 ± 1.2	<0.001	D+H > D** > H* > C***
CRI-II	6.4 ± 3.2	4.9 ± 3.1	5.9 ± 3.8	2.3 ± 0.9	<0.001	D, D+H > H* > C***
TG/HDL-C	10.2 ± 4.9	8.5 ± 6.2	12.7 ± 7.1	3.9 ± 1.1	<0.001	D+H > D* > H* > C***
hs-CRP (mg/L)	5.8 ± 4.1	3.2 ± 2.8	7.1 ± 4.9	1.2 ± 0.8	<0.001	D+H > D** > H* > C***
Fetuin-A (µg/mL)	443.6 ± 198.3	228.5 ± 112.7	712.4 ± 231.5	298.7 ± 97.4	<0.001	D+H > D*** > C* > H***
Free-T4 (ng/dL)	1.3 ± 0.4	0.6 ± 0.3	0.4 ± 0.2	1.6 ± 0.5	<0.001	C > D*** > H* > D+H***
TSH (µIU/mL)	5.9 ± 3.2	14.7 ± 12.5	18.3 ± 15.2	3.1 ± 1.8	<0.001	D+H, H > D* > C***
BMI (kg/m²)	27.6 ± 2.3	29.1 ± 3.1	29.8 ± 3.4	26.3 ± 2.5	<0.001	D+H, H > D* > C***
HbA1C (%)	8.4 ± 2.6	5.2 ± 1.1	9.1 ± 2.9	4.7 ± 0.7	<0.001	D+H > D*** > C* > H***
FBS (mg/dL)	221.7 ± 72.5	112.4 ± 32.8	234.6 ± 85.3	89.5 ± 16.2	<0.001	D+H > D** > C* > H***

D+H represents T2D & hypothyroid; D signifies T2D group; H for hypothyroid group and C represents control group; * means ($P < 0.05$);** ($P < 0.01$); *** ($P < 0.001$). AIP, Atherogenic Index of Plasma; BMI, body mass index; CRI, Castelli Risk Index; FBS, fasting blood sugar; HbA1C, hemoglobin A1c; HDL, high-density lipoprotein; hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein; TC, total cholesterol; TG, triglycerides; TSH, thyroid-stimulating hormone.

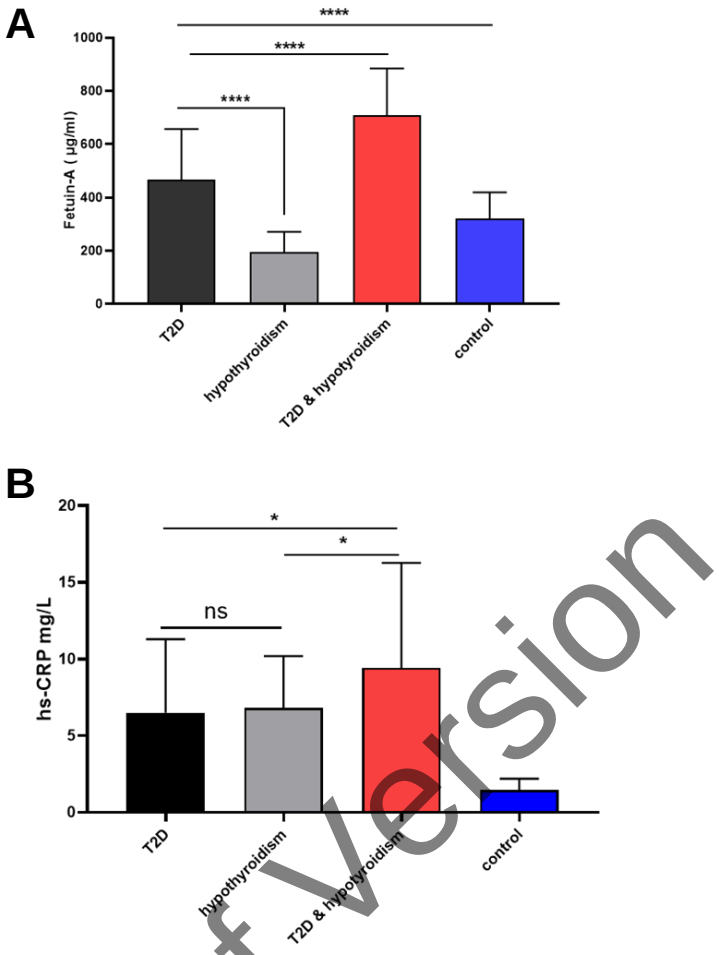

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Figure 1. Graphs representative of the means measures for groups of the study (black; type 2 diabetes group, gray; bars for the hypothyroidism, red for coexistence T2D and hypothyroidism, while blue bars representative for control group. **(A)** represents the results of fetuin-A, **(B)** represents serum hs-CRP. Differences between means calculated by unpaired student t test; * means $P < 0.05$; **** means $P < 0.0001$. hs-CRP, high-sensitivity C-reactive protein; T2D, type 2 diabetes.

Table 2. Correlation analysis results of fetuin-A and hs-CRP with regards to study variable measured and calculated.

Parameter	Diabetic hs-CRP	Diabetic fetuin-A	Hypothyroid hs-CRP	Hypothyroid fetuin-A	Diabetic+ Hypothyroid hs-CRP	Diabetic+ Hypothyroid Fetuin-A	Control hs-CRP	Control fetuin-A
TG	0.42**	0.38*	0.37*	0.12	0.51***	0.62***	0.22	0.15
TC	0.19	0.21	0.23	0.14	0.18	0.22	0.17	0.26
LDL	0.18	0.21	0.09	0.08	0.19	0.17	0.21	0.18
HDL	-0.35*	-0.41**	-0.28	-0.33*	-0.47***	-0.43**	-0.18	-0.24
AIP	0.47***	0.39*	0.38*	0.34*	0.58***	0.54***	0.11	0.15
CRI-I	0.45**	0.4*	0.31*	0.21	0.53***	0.6***	0.12	0.1
CRI-II	0.42**	0.38*	0.28*	0.19	0.57***	0.63***	0.22	0.18
TG/HDL-C	-0.38**	-0.31*	-0.32*	-0.19	-0.47***	-0.43**	-0.18	-0.18
HbA1C	0.52***	0.44**	0.23	0.25	0.49***	0.67***	0.17	-0.20
BMI	0.31*	0.27	0.34*	0.22	0.38*	0.26	0.26	0.12
TSH	0.18	0.19	0.45**	0.19	0.32*	0.41**	0.16	0.11
Free-T4	0.20	0.19	-0.41**	-0.25	-0.37*	-0.47**	0.11	0.14

The results in cells of the table represents the value of r calculated using Pearson's correlation with its *t*-test, negative value means negative correlation, positive value means positive correlation; *, $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$. AIP, Atherogenic Index of Plasma; BMI, body mass index; CRI, Castelli Risk Index; FBS, fasting blood sugar; HbA1C, hemoglobin A1c; HDL, high-density lipoprotein; hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein; TC, total cholesterol; TG, triglycerides; TSH, thyroid-stimulating hormone.

Discussion

Here we analyzed inflammatory, metabolic, and thyroid function markers among different groups of Iraqi women (T2D, hypothyroid, diabetic with hypothyroidism, and healthy controls). The most striking results emerged in the diabetic–hypothyroid group, which exhibited the most severe metabolic instability. This was evident from significantly elevated atherogenic indices like AIP, fetuin-A, and inflammation markers hs-CRP. Furthermore, glycemic parameters including HbA1c and FBG were also notably elevated. These findings align with previous research indicating a synergistic decline in metabolic and cardiovascular risk in patients with coexisting T2D and hypothyroidism. [1, 6, 10] In addition, the worsening dyslipidemia observed in this group evidenced by the TG/HDL-C ratio, AIP, CRI-I, and TG could result from the combined effects of dysglycemia and thyroid hormone deficiency on lipid metabolism. [11, 12] These results in combined pathology were shown to be higher than each disease alone. Furthermore, lipid profile indices like AIP, CRI-I, CRI-II, and TG/HDL-C were higher and more pronounced than traditional lipid measures in combined disease rather than in T2D or hypothyroidism groups alone. These could be considered signs of an increased risk of cardiovascular disease. These results differ from previous studies. [13-15]

Moreover, our correlation analysis supports the exploration of mechanisms of aggravation of metabolic disorders. In the combined T2D and hypothyroidism group, both fetuin-A and hs-CRP displayed a strong association with TG and the glycemic marker HbA1c, which suggests that metabolic dysregulation was aggravated by inflammation being higher than those with T2D or hypothyroidism alone. [16-18] Particularly, fetuin-A was linked to the metabolic syndrome and established as a potential marker in combined disease. Fetuin-A levels were observed to be significantly higher in the coexistence group than in the T2D group and hypothyroidism group, similar to the previous studies examining the relationship between the fetuin-A level and insulin resistance [19] or with hypothyroidism. [20]

In contrast, the hypothyroidism group showed an inflammatory response related to thyroid function. A strong positive correlation was observed between TSH and hs-CRP but not fetuin-A. This suggests that low-grade systemic inflammation could be mediated by inflammatory cytokines such as interleukin-6 upstream of hs-CRP. [21] The lack of association between fetuin-A and TSH might be due to TSH's independent metabolic effects beyond its role as a hepatic chemokine. Furthermore, a negative correlation between free T4 and hs-CRP suggests a clinical implication for treating hypothyroidism with thyroxine replacement. This could potentially reverse the inflammation and dyslipidemia often associated with the condition. [22]

The T2D group represents an intermediate state, when the fetuin-A level is elevated significantly compared to the hypothyroid and control groups. The hepatokine level correlated positively and significantly with the glycemic marker HbA1c and the level of AIP. T2D represents a low-grade inflammatory state associated with elevated inflammatory markers. [23] In the current study, hs-CRP was related to HbA1c level but surprisingly not with LDL-cholesterol. This suggests that rising inflammatory biomarkers are more linked to TGL dysfunction, HDL dysfunction, and glycemic levels than cholesterol-rich lipoproteins. However, patients remain at evolving risk of atherosclerosis, cardiovascular accidents, and other complications associated with diabetes. [24] The lack of association in the control group suggests that elevated inflammatory biomarkers stem from metabolic derangement across various diseases. Furthermore, in the control group, BMI—a measure of adiposity—correlated with the inflammation levels observed in the disease state. This indicates that obesity complicates the metabolic–inflammatory crosstalk. [25] These observations suggest that a strict treatment regimen for managing blood sugar and thyroxin levels might reverse or slow the development of atherogenic events.

Conclusion

The combination of diabetes and hypothyroidism showed a synergistic effect on systemic inflammation, glycemia, and dyslipidemia compared to each disease alone and healthy controls. The strong association of inflammation and levels of glycemia and TGL-rich lipoproteins raise the concern that traditional measurements of cholesterol-rich lipoproteins are not sufficient to assess the risk of atherogenity and highlight the need for modifiable lipid indices. Finally, a tight therapeutic measure to all groups of patients could adjust the risk in those patients. The synergistic effects of both diseases and rising risk suggest that dual screening for thyroid function and level of glycemia in both conditions presented individually. Further studies are needed to monitor patients with close glycemic control and a thyroxin replacement regimen for inflammatory state and dyslipidemia.

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Author contributions

Both authors contributed substantially to the concept and design of this study, acquiring the data, reviewing the literature, and its analysis and interpretation. Both authors contributed substantially to acquiring the data. Both authors contributed to drafting the manuscript. Both authors edited the manuscript critically for important intellectual content. All authors approved the final version submitted for publication and take responsibility for statements made in the published article.

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Conflicts of interest statement

We have no potential conflicts of interest relevant to this article.

Data sharing statement

All data generated or analyzed during the present study are included in this published article. Further details are available for non-commercial purposes from the corresponding author on reasonable request.

Declaration of AI use

No AI tools were used in any part of the preparation of this manuscript.

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