Advanced Medical Journal, Vol.10, No.3, P.176-183,2025 https://doi.org/10.56056/amj.2025.383

Serum Visfatin Level Among Patients with Subclinical Hypothyroidism in Relation to Insulin Resistance

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Abstract

Background and Objectives: Subclinical hypothyroidism is a disorganization of the thyroid gland determined by normal free T4 and T3 with elevated thyroid stimulating hormone. The patients with subclinical hypothyroidism are asymptomatic, it can only be diagnosed through laboratory tests. The prevalence of subclinical hypothyroidism ranged from (4.6 to 16.7%) worldwide. The current study intended to evaluate the level of serum visfatin among patients with subclinical hypothyroidism and it is relation to insulin resistance in opposition to in good health control.

Methods: This study was performed as a cross-sectional study on January 2023 among 160 participants, 80 newly diagnosed subclinical hypothyroidism and 80 healthy personnel as control group. Subclinical hypothyroid individuals were recruited from patients attending Diabetes and Endocrinology Unit in Azadi Teaching Hospital, 80 healthy personnel were picked from relatives and neighbors. Blood samples were collected from both groups for serum (TSH, T4, T3, sugar, insulin, visfatin). Comparison of results were compared.

Results: There was no notable distinction between mean serum visfatin level in subclinical hypothyroidism patients (39.78±11.64) and controls (39.82±10.02).No notable differences were detected in the mean between subclinical hypothyroidism patients with TSH less than (10 microIU/ml) and those with TSH more than (10 microIU/ml) as follows:(p =0.869), insulin(p=0.214), FBS (p=0.811), and HOMA-IR (p=0.511).The mean of visfatin patients with normal HOMA-IR was (37.89) , compared with 40.29 of patients with high HOMA-IR, but the distinction was not of particular note (p=0.454).

Conclusion: There was no particularly notable change of mean serum visfatin level in Subclinical hypothyroid patients, contrary to healthy individuals.

Key words: Cardiovascular disorders,Insulin resistance,Subclinical hypothyroidism,Serum Visfatin

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Introduction

Subclinical hypothyroidism characteristically and biochemically stated by grown up serum thyroid stimulating hormone (TSH) with normal thyroxine (T4) levels.[1] Depending on the different populational studies, the incidence of subclinical hypothyroidism was 3-15%,with higher incidence among women (8%) compared to men (3%)Globally, there was an established correlation between subclinical hypothyroidism and cardiovascular diseases as it considered as a subject to danger for the advancement of coronary artery disease, congestive heart failure and stroke.[2,3,4,5,6] Moreover, 2 to 6% of patients with subclinical hypothyroidism progress to overt hypothyroidism, particularly those patients with serum TSH level more than 10 mIU/mI and positive thyroid peroxidase (TPO) antibody.[5,7] Visfatin is an adipokine secreted from different sources and tissues of the body such as macrophages, chondrocytes, and amniotic epithelial cells, heart, pancreas, liver, skeletal muscle as well as adipose tissue.[8] It has many roles in the body as a regulator of hunger, sensitivity of insulin, immunity, tissue inflammation and homeostasis of vasculature.[9,10] Visfatin has recently been suggested as a possible marker for inflammatory and cardiovascular.[11] Visfatin binds to insulin receptor and exhibits insulin-mimetic actions; therefore, it stimulates glucose uptake in adipocytes and muscle cells and suppresses glucose release from hepatocytes.[8] It is now believed that visfatin action can be endocrine, paracrine, and autocrine as well. These autocrine effects of visfatin may play an important role in regulating insulin sensitivity in the liver.Insulin resistance is defined as a decreased ability of several organs, especially the liver and adipose tissue, to respond to the effects of insulin. This results in reduced glucose utilization and a corresponding rise in beta-cell insulin secretion and hyperinsulinemia.[12,13] Numerous metabolic effects of insulin resistance include diabetes mellitus, hypertension, dyslipidemia, visceral obesity, hyperuricemia, increased generation of inflammatory markers, and endothelial dysfunction.[14] Moreover, research found that those with subclinical hypothyroidism were more probable to have insulin resistance.[15] As there was an inconsistent of the relationship between serum visfatin level and abnormal thyroid function.[16] as well as limited data are available concerning this issue in our locality, therefore; the purpose of our cross-sectional study was to assess serum visfatin level among subclinical hypothyroid patients, as well as to understand the relationship between serum visfatin level and insulin resistance among patients with subclinical hypothyroidism.

Patients and methods

The present cross-sectional research was done at Azadi Teaching Hospital/Duhok City/Kurdistan Region/Iraq, over the period of seven months from January 2023 to July 2023. A total of 160 the attendees were sign up in this study, 80 were patients with recently diagnosed subclinical hypothyroidism as they are being present the Endocrine and Diabetic Unit at Azadi Teaching Hospital, as well as 80 were healthy for comparison and were taken from relatives and neighbors. Patients with TSH level more than (4.2 microIU/ml) with normal free T4 regarded as having subclinical hypothyroidism.[16] The patients and healthy subjects were matching age and sex.The questionnaire was well prepared and distributed to all participants as included questions about name, age, sex, as well as height, weight, and waist circumference were measured. Calculation of BMI depends on the division of weight in kilograms by square of the height in meters (kg/m^2). Body mass index below 18.5 kg/m^2 means underweight range, between 18.5 kg/m^2 and 24.9 kg/m^2 it

https://doi.org/10.56056/amj.2025.383

means healthy weight, 25.0-29.9 kg/m^2 means overweight, and BMI 30.0 kg/m^2 and above means obesity.[17] Exclusion criteria include patients with chronic inflammation, Diabetes mellitus, cancer, Hypertension, cardiovascular disease, as they affect visfatin levels. In the morning and after an overnight fasting at Mazi laboratory, a 5ml venous blood sample was taken into a Gel tube and centrifuged for 20 minutes at 3000 revolutions per minute. All parameters were measured by Cobas 6000 (Roche-HITACHI) based upon different principles. Hormones such as (TSH, T4, T3, Insulin, TPO) were depended upon electrochemiluminescence (ECL) immuno assay, whereas the serum glucose was depended upon enzymatic colorimetric method , serum visfatin were measured by ELISA that is done in sandwich enzyme immunoassay form depending on antigen -antibody reaction and enzymatic reaction .The cutoff used for serum glucose (74-109 mg/dl), insulin (less than 25 mIU/L), thyroid stimulating hormone(0.2-4.2 microIU/mL), free T4(12-22 pmol/L), and free T3 (3.1-6.8 pmol/L), TPO (up to 34IU/L). Homeostatic Model Assessment of Insulin Resistance HOMA-IR was measured from fasting glucose and insulin as follows: HAMA-IR = Glucose(mg/dl) × insulin(µU/l) / 405 (HOMA-IR>3.0) was regarded as insulin resistance.[18] Ethical permission was gained from the Research Committee of the Directorate of Health in Duhok and Kurdistan Higher Council of Medical specialties. (On 26 Oct 2022, Reference number:26102022-8-7). Data were analyzed using the Statistical Package for Social Sciences (SPSS, version 26). Chi square test of association was used to compare proportions of two or more groups. Fisher's exact test was employed when the estimated frequency (value) was less than 5 of more than 20% of the cells of the table. Student's t test for two separate samples (unpaired t test) was used to contrast the means of the two groups. A p value of ≤ 0.05 was regarding as statistically notable.

Results

The study included 80 patients with subclinical hypothyroidism (Group I), and 80 apparently healthy individuals (Group II) which served as a control group. The mean (SD) age of patients was 33.4 (8.9) years, and that of the controls was 32.8 (8.6) years. The majority (75.6%) of the whole sample was females (75% of patients and 76.3% of the controls). The mean BMI of the patients (28.3 Kg/m^2) was significantly ($p<0.001$) higher than that of the controls (24 Kg/m^2). More than one third (35%) of the patients were obese, compared with 2% among the controls ($p<0.001$). The majority (85.0%) of the male patients' waist circumference were <102cm, and majority of female patients (66.7%) waist circumference was >88cm was significantly ($p<0.001$) higher than that of controls (51.2%). as presented in Table (1).

Table (1): Basic characteristics of the study groups.

Parameters	Subclinical hypothyroidism		Control group		Total		P
Age(years)	No.	%	No.	%	No.	%	
<40	61	(76.3)	63	(78.8)	124	(77.5)	0.705*
>40	19	(23.8)	17	(21.3)	36	(22.5)	
Mean (SD)	33.4	(8.9)	32.8	(8.6)			0.705*
Sex							
Male	20	(25.0)	19	(23.8)	39	(24.4)	
female	60	(75.0)	61	(76.3)	121	(75.6)	0.854*
BMI(Kg/m$^{2)}$							

https://doi.org/10.56056/amj.2025.383

<25	22	(27.5)	48	(60.0)	70	(43.7)	
25-29	30	(37.5)	30	(39)	62	(38.8)	
>30 Mean (SD)	28	(35.0)	2	(2.6)	28	(17.5)	<0.001
	28.3	(5.0)	24.0	(1.9)			>0.001
Waist circumference(cm)							
Male<102cm	17	(85.0)	19	(100.0)	36	(92.3)	
male>102cm	3	(15.0)	0	(0.0)	3	(7.7)	0.231*
total	20	(100.0)	19	(100.0)	39	(100.0)	
Female<88cm	20	(33.3)	39	(63.9)	59	(48.8)	
female>88cm	40	(66.7)	22	(36.1)	62	(51.2)	0.001*4
Total	60	(100.0)	61	(100.0)	121	(100.0)	

The mean TSH of patients (7.31 micro-IU/ml) was significantly (p<0.001) higher than that of the controls (2.42 micro-IU/ml). The mean insulin level of patients (19.15 mIU/l) was significantly (p<0.001) higher than that of the controls (6.31 mIU/l). The mean serum glucose of patients (96.11 mg/dl) was significantly (p<0.001) higher than that of the controls (89.74 mg/dl). The mean HOMA-IR of patients (4.66) was significantly (p<0.001) higher than that of the controls (1.40).and those whose HOMA-IR >3 (63.0) was significantly (<0.001) higher than control (7.0). More than one third (35%) of patients had high Anti TPO level, compared with 6.3% of the controls (p<0.001). No significant difference was detected between cases and controls regarding the mean visfatin (p=0.981) as presented in Table (2).

Table (2): Biochemical parameters of the study groups.

Parameters	Subclinical hypothyroidism No.	%	Control group No.	%	p
TSH (micro-IU/ml), mean (SD)	(7.31)	(2.90)	2.42	(0.98)	< 0.001†
freeT4 (pmol/l), mean (SD)	(15.05)	(1.97)	(16.70)	(1.84)	< 0.001†
freeT3 (pmol/l), mean (SD)	(3.72)	(0.55)	(3.63)	(0.32)	0.230†
Insulin (mIU/l), mean (SD)	(19.15)	(8.19)	(6.31)	(3.68)	< 0.001†
FBS (mg/dl), mean (SD)	(96.11)	(6.73)	(89.74)	(5.91)	< 0.001†
HOMA IR, mean (SD)	(4.66)	(2.12)	(1.40)	(0.83)	< 0.001†
HOMA-IR, No. (%)					
< 3.0	(17.0)	(21.3)	(73.0)	(91.3)	
≥ 3.0	(63.0)	(78.8)	(7.0)	(8.8)	< 0.001*
Anti TPO, mean (SD)	(54.37)	(82.93)	(16.61)	(14.67)	< 0.001†
ANTI TPO, No. (%)					
< 34 IU/L	(52.0)	(65.0)	(75.0)	(93.8)	
≥ 34 IU/L	(28.0)	(35.0)	(5.0)	(6.3)	< 0.001*
Visfatin	(39.78)	(11.64)	(39.82)	(10.02)	0.981†

*By Chi square test. †By unpaired t test.

https://doi.org/10.56056/amj.2025.383

Considering only those with subclinical hypothyroidism (Group I), no significant differences were detected in the following means between those with TSH less than 10 microIU/ml and those with TSH more than 10 microIU/ml as follows: visfatin (p=0.869), insulin (p=0.214), FBS (p=0.811), and HOMA-IR (p=0.511) as presented in Table (3).

Table (3): Biochemical parameters by TSH levels among patients with subclinical hypothyroidism.

parameters	TSH < 10 (n = 58)		TSH ≥ 10 (n = 22)		
	Mean	(SD)	Mean	(SD)	P*
Visfatin	(39.43)	(12.56)	(39.91)	(9.03)	0.869
Insulin (mIU/l)	(18.44)	(8.39)	(21.00)	(7.50)	0.214
FBS (mg/dl)	(95.82)	(7.06)	(96.22)	(5.92)	0.811
HOMA-IR	(4.56)	(2.25)	(4.92)	(1.76)	0.511

*By unpaired t test.

Only patients with subclinical hypothyroidism were included in Table 4. The mean of visfatin of patients with normal HOMA-IR was 37.89, compared with 40.29 of patients with high HOMA-IR, but the difference was not significant (p=0.454). All the other differences in the means of the parameters mentioned in the (Table 4) between those with normal and high HOMA-IR were not significant as follows: TSH (p=0.712), freeT3 (p=0.919), freeT4 (p=0.183), BMI (p=0.878), waist circumference (p=0.956), anti TPO (p=0.517) (Table 4).

Table (4): Demographical and Biochemical parameters by HOMA-IR levels among patients with subclinical hypothyroidism.

parameters	HOMA-IR < 3 (n = 17)		HOMA-IR ≥ 3 (n = 63)		
	Mean	(SD)	Mean	(SD)	P*
Visfatin	(37.89)	(11.29)	(40.29)	(11.77)	0.454
TSH (micro-IU/ml)	(7.07)	(2.55)	(7.37)	(3.00)	0.712
T3(pmol/l)	(3.71)	(0.52)	(3.72)	(0.57)	0.919
T4(pmol/l)	(14.49)	(1.70)	(15.21)	(2.03)	0.183
BMI (Kg/m^2)	(28.50)	(5.01)	(28.29)	(5.09)	0.878
Waist circumference (cm)	(92.18)	(15.56)	(91.97)	(13.29)	0.956
Anti TPO(IU/ml)	(42.71)	(61.40)	(57.51)	(87.99)	0.517

*By unpaired t test.

Discussion

The correlation between thyroid dysfunction particularly hypothyroidism and increase body weight (whether BMI or WC) was commonly investigated.[19] Our study exhibited that patients with subclinical hypothyroidism was highly associated with increasing BMI and obesity, as the mean BMI and WC were higher among subclinical hypothyroid patients, as well as, near two third of them were overweight and obese. Data of various studies done in different part in the world shown the same results as of our

https://doi.org/10.56056/amj.2025.383

results.[20,21] This finding may be due some factors such as effects of high TSH level, low grade inflammation (inflammatory cytokine release), decreasing resting energy expenditure and presence or absence of leptin hormone.[21–23] Moreover, the current study shown a higher prevalence of subclinical hypothyroidism in young female comparing to male gender. Many studies show the same results.[24,25] This high rate can be explained by differences in sex hormones metabolism as there is a higher prevalence of chronic autoimmune thyroiditis (CAT) and higher estrogen levels in females.[2,26] Its well-known that the subclinical hypothyroidism can affects the insulin sensitivity leading to various metabolic.[14] The current study showed a high mean insulin level and HOMA-IR in patients with subclinical hypothyroidism, leading to early insulin resistance that can be regarded as an early manifestation of impaired glucose metabolism. The underlining causes can be obesity, increase leptin level, inflammatory cytokine release (inflammatory reaction) and most commonly due to high TSH level.[27,28] Binding of TSH to the TSH receptor of preadipocytes induce differentiation and formation of adipocyte with promotion of obesity leading to insulin resistance.[29] Moreover, TSH have an important effect on insulin secretion and glucose absorption through gastrointestinal tract (increase glucose absorption), liver (increase hepatic glucose output), skeletal and adipose tissue (increase lipolysis with release of free fatty acid).[30] Inconsistent data were available regarding the association between visfatin and subclinical hypothyroidism as well as volatile role of thyroid hormones in regulation of visfatin level.[31,32] Our data showed no difference in mean level of visfatin among subclinical hypothyroid patients and healthy control. The finding of the present study was consistent with a study done in pamukkale, that demonstrated a non-significant connection between serum visfatin level and patients with subclinical hypothyroidism as well as the serum TSH level.[33] However, a study done by Ozkaya M et al.[33] showed that patients with thyroid dysfunction specifically those associated with change in free T4 and free T3 had up-regulation of visfatin expression particularly visfatin expression in visceral fat (regulation of visfatin mRNA expression) due to a nonlinear collaboration between visfatin and T3 and T4.[34] The present study found that mean visfatin level was higher among subclinical hypothyroid patients with IR (HOMA-IR more than 3.0). These results were consistent with other study done and inconsistent with others.[35,36] This is mostly explained by the action of visfatin on insulin signaling pathway of adipocytes resulting in increased glucose uptake (protective role).[37] Moreover, interaction of visfatin with insulin receptors at locations other than insulin-binding sites, will affects the utilization of glucose by muscle and liver (increase utilization) as well as modifying insulin sensitivity leading to decreasing blood glucose levels. Therefore, adipose-tissue-specific deletion of visfatin result in glucose metabolism impairment and multiorgan IR development.[34] On the other hand, increase serum visfatin levels associated with inflammatory process as it enhances release of inflammatory mediators, regardless the presence of other causes such as metabolic syndrome and T2DM.[38]

Conclusion

The current study concludes that serum visfatin level did not changes in patients with subclinical hypothyroidism Compared to healthy control. Serum visfatin level positively correlates with insulin resistance among patients with subclinical hypothyroidism. This study demonstrated that subclinical hypothyroidism was more common among young, obese, females.

https://doi.org/10.56056/amj.2025.383

Conflict of Interest

The authors declare no conflicts of interest.

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