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Assessment of clinical and nutrological metabolic parameters influencing vasodilation mechanisms in the initial phases of hypothyroidism and hypertension: an observational controlled clinical study

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Abstract

Introduction: Hypothyroidism is a widespread endocrine illness that has major effects on the body as a whole, especially on the regulation of blood vessels. When hypertension is also present, it makes endothelial dysfunction much worse**. Objective**: This study aimed to assess the Clinical and Nutrological Metabolic Parameters Influencing Vasodilation Mechanisms in the Initial Phases of Hypothyroidism and Hypertension. **Methods:** Tests were measured in addition to Prostaglandin E Synthase, Bradykinin, and Kallikrein levels using ELISA in early hypothyroidism and hypothyroidism with hypertension among 90 participants consisting of 35 hypothyroid participants, 35 hypothyroid participants with hypertension, and 20 controls attending the Endocrinology Department at Azadi Teaching Hospital from January to May 2025. **Results**: The hypothyroid group was dominated by females, 60.0%, while in controls the male sex was the majority, 65.0%. The hypertensive hypothyroidism group had the highest mean age of 47.1 ± 15.82 years. Obesity was significantly higher in hypothyroidism with hypertension, 54.29%. When compared to controls, thyroid hormone tests showed that both patient groups had significantly higher TSH and significantly lower T3 and T4 ($p<0.05$) when One way ANOVA applied. Vasodilatory markers were significantly lower in the patient groups. Prostaglandin E Synthase levels were much lower in hypothyroidism - 206.74 ng/mL and hypothyroidism with hypertension - 182.05 ng/ml compared to controls - 397.94 ng/mL. **Conclusion**: Bradykinin and Kallikrein also revealed significant declines, and the levels were lowest in hypertensive hypothyroidism. These findings indicate that early hypothyroidism, when combined with hypertension, is strongly associated with impaired vasodilatory signaling, thus suggesting an early evaluation of cardiovascular risk in thyroid disorders.

Keywords: Hypothyroidism. Hhypertension. Metabolic. Cardiovascular disease.

Introduction

Hypothyroidism has a multifaceted and clinically important impact on cardiovascular homeostasis, creating a pathophysiologic environment that is conducive for the development and progression of arterial hypertension. Both overt and subclinical hypothyroidism have been uniformly associated with increased systemic vascular resistance and a predisposition towards higher diastolic blood pressure values. This is mediated by the loss of the direct vasodilatory effect of thyroid hormones on the smooth muscle of the vasculature and by secondary hemodynamic factors. Global analyses and prospective studies have demonstrated that individuals with lower thyroid function have increased risk for cardiovascular events and changes in blood pressure. Subjects. Mechanistically, hypothyroidism inhibits endothelium-dependent vasodilation by diminishing endothelial nitric oxide synthase (eNOS) expression and activity, nitric oxide (NO) bioavailability, asymmetrical dimethylarginine (ADMA), and oxidative stress **[1-3]**.

These functional impairments can be quantified in clinical trials in terms of decreased flow-mediated vasodilatory responses and decreased responsiveness to traditional vasodilatory stimuli, thus providing the direct link between hypothyroid biochemistry and increased peripheral resistance. Hypothyroidism is associated with a pro-inflammatory state, including increased circulating cytokine levels, immune cell activation in autoimmune thyroiditis, and increased oxidative lipid and protein modifications. This environment results in endothelial cell activation, increased vascular permeability, and the presence of circulating blood components, lipids, and immune cells in the vascular intima and perivascular interstitium **[4,5]**.

Ultimately, matrix remodeling, microvascular rarefaction, and arterial stiffening may result, contributing to increased fixed resistance and hypertension. Complementary molecular mechanisms also exist to augment hypertensive physiology in hypothyroid participants, including alterations in components of the renin-angiotensin-aldosterone system, including upregulation of angiotensinogen and components of vascular smooth muscle cell phenotype transitions, endothelial lipotoxicity due to alterations in lipid metabolism, and decreased clearance of vasoconstrictive substances, all of which are consistent with vasoconstriction and structural remodeling of the vasculature **[1,4,6]**.

Biomarkers that are clinically relevant, such as those that measure vasodilatory ability, including nitric oxide metabolites, asymmetric dimethylarginine, endothelial progenitor cells, inflammatory markers, as well as noninvasive vascular studies such as Flow-Mediated Dilation (FMD) and pulse wave velocity, offer a mechanistic approach to studying the role of thyroid function in hypertension. Importantly, these studies also offer a potential therapeutic approach to studying hypertension, as normalization of hypothyroidism with levothyroxine improves endothelial dysfunction and some measures of vasodilatory ability, implying that hypertension, or at least some aspect of it, is reversible **[6-8]**. These studies highlight the need to consider normalization of thyroid hormone levels in hypertensive participants with underlying hypothyroidism as part of a broader approach to mitigating cardiovascular risk, and also support a model whereby loss of support from thyroid hormones on vasodilatory ability, inflammation, plasma infiltration, and vascular remodeling, as well as maladaptive neurohormonal responses, feed into a feed-forward model of hypertension from hypothyroidism **[9-11]**.

Thus, This study aimed to assess the Clinical and Nutrological Metabolic Parameters Influencing Vasodilation Mechanisms in the Initial Phases of Hypothyroidism and Hypertension.

Materials and Methods

Study Design

The study was designed, conducted and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies **[12]**, and the completed STROBE checklist (available at: https://www.strobe-statement.org/. Accessed on: May 25, 2025) is provided as a supplementary file with this submission.

Participants and Settings

The present study was executed at Azadi Teaching Hospital/Endocrinology Unit from January 2025 to June 2025, enrolling 90 participants (35 with hypothyroidism, 35 with both hypothyroidism and hypertension, and 20 healthy controls). A doctor looked at the cases and validated them with thyroid function tests. Blood samples were collected from all participants, and the sera were isolated and preserved at -20 °C until utilized for thyroid function testing and ELISA assays.

Thyroid function tests

All subjects in the study were assessed for thyroid function tests (T3, T4 and TSH) according to manufacturer protocols using Cobas e411 automated immunology analyzer (Roche, Germany).

ELISA tests

An ELISA test dedicated to serum Prostaglandin E Synthase, Bradykinin, and Kallikrein (BT LAB, China) was utilized to assess the levels of these hormones in participants and control sera. The test is based on sandwich ELISA, in which antibodies specific to the target protein are coated on to an ELISA plate and bind to the target protein once sera are added and incubated at 37°C. Then a washing step is performed to remove unbound substances, and conjugated antibodies are loaded into the wells to bind the target protein. After washing, the substrate is incubated with labelled antibodies at 37°C. The reaction is stopped using a stopped solution, and the developed color is measured at 450 nm.

Ethical Approval

The study was performed subsequent to receiving ethical permission from the Ethical permission Committee at Tikrit University (issue number: 3/7/314 at 6/11/2024). All participants were given a consent form before commencing specimen collection.

Statistical analysis

Data were presented in the study as a descriptive table and comparisons between groups were preformed using One Way ANOVA (Kruskal–Walli's test) in Graph Pad Prisim version 10.1.

Results

Regarding gender distribution, Table 1 illustrates that females predominated in the hypothyroidism (21/35, 60.0%) and hypothyroidism-with-hypertension (19/35, 54.3%) groups, but males predominated in the control group (13/20, 65.0%).

Table 1. Gender distribution of study population.

Gender distribution	Hypothyroidism		Hypothyroidism with hypertension		Control	
	No.	%	No.	%	No.	%
Females	21	60.00	19	54.29	7	35.00
Males	14	40.00	16	45.71	13	65.00
Total	35	100.00	35	100.00	20	100.00

Source: Own authorship.

The data in Table 2 revealed considerable differences between the three groups: the hypothyroid-only group has a mean age of 39.4 ± 12.38 years (n = 35), the hypothyroid-with-hypertension group is more aged with a mean of 47.1 ± 15.82 years (n = 35), whereas the control group is more youthful with a mean of 31.92 ± 10.63 years (n = 20).

Table 2. Age distribution of study groups.

Age	Mean	±SD	Total
Hypothyroidism	39.4	12.38	35
Hypothyroidism with hypertension	47.1	15.82	35
Control	31.92	10.63	20

Source: Own authorship.

In terms of the distribution of body mass index Table 3 depicted hypothyrodism, hypothyroidism with hypertension, and control subjects and illuminates the metabolic impact of thyroid dysfunction and its cardiovascular comorbidity. The highest proportion of participants in the hypothyroidism group was from the overweight (26–30 kg/m²; 37.14%) and obese (>30 kg/m²; 34.29%) groups, indicative of the strong association between thyroid hormone deficiency and lipid metabolism disturbances that lead to weight gain and adiposity. This was more pronounced with hypothyroidism with hypertension, where more than half of the participants (54.29%) were obese, with another 31.43% being overweight.

Table 3. BMI distribution in patents and control groups.

BMI	Hypothyroidism		Hypothyroidism with hypertension		Control	
	No.	%	No.	%	No.	%
<20	3	8.57	1	2.86	5	25.00
20-25	7	20.00	4	11.43	9	45.00
26-30	13	37.14	11	31.43	4	20.00
>30	12	34.29	19	54.29	2	10.00
Total	35	100.00	35	100.00	20	100.00

Source: Own authorship.

According to the thyroid function tests, Table 4 demonstrates that apparent alterations in thyroid and its corresponding metabolic markers in participants with hypothyroidism, particularly those with underpinning hypertension, compared with controls. TSH was significantly elevated in both hypothyroid groups (10.06 ± 4.27and 12.15 ± 3.07μIU/mL, respectively) compared with controls (2.45 ± 0.91 μIU/mL; $p<0.05$), reflecting the impaired negative feedback control of the hypothalamic-pituitary-thyroid axis. At the same time, the levels of T3 and T4 in the blood were significantly lower in both groups of participants, but more strongly dropped in the hypothyroidism and hypertension cohort (T3: 0.38 ± 0.10 ng/mL; T4: 1.14 ± 0.83μg/dL), which indicates greater thyroid impairment in the combined pathology participants.

Table 4. Mean ± SD values for the variables TSH, T3, T4 in the study groups.

ELISA parameters	Groups	No.	Mean± SD	Differences
TSH (0.27 - 4.2 μIU/mL)	Hypothyroidism	35	10.06 ±4.27	A
	Hypothyroidism with hypertension	35	12.15 ±3.07	B
	Control	20	2.45 ± 0.91	C
T3 (0.80-2.0 ng/mL)	Hypothyroidism	35	0.42 ± 0.11	A
	Hypothyroidism with hypertension	35	0.38 ± 0.10	B
	Control	20	1.29 ± 0.82	C

T4 (5.1 to 14.1 µg/dL)	Hypothyroidism	35	1.85 ± 1.03	A
	Hypothyroidism with hypertension	35	1.14 ± 0.83	A
	Control	20	10.07 ±3.98	B

* If the letters are the same, p>0.05; if the letters are different, p≤0.05. Source: Own authorship.

As illustrated in Table 5, ELISA-based assessment of vasodilatory and vascular regulatory markers offers important insights into the altered biochemical milieu of hypothyroidism and its correlation with hypertension. Prostaglandin E synthase, a key enzyme in prostaglandina-mediated vasodilation and vascular tone regulation, was significantly reduced in hypothyroidism (206.74 ± 161.33 ng/mL) and hypothyroidism with hypertension (182.05 ± 98.62 ng/mL) compared to normotensive controls (397.94 ± 229.48 ng/mL, p<0.05). The bradykinin level in hypothyroid and hypertensive-hypothyroid groups was lower (6.98 ± 3.93 ng/mL and 4.14 ± 2.86 ng/mL, respectively) compared to controls (11.58 ± 5.22 ng/mL), though not statistically significant (p>0.05). Bradykinin is a potent vasodilator that promotes NO and prostacyclin release, and its reduction may further weaken vasodilatory reserve. Kallikrein, the enzyme that generates bradykinin, also showed significantly reduced levels in the two hypothyroid groups (160.12 ± 114.20 and 143.67 ± 103.27 mU/mL) compared with controls (314.85 ± 187.16 mU/mL, p<0.05).

Table 5. Mean ± SD values for the variables prostaglandin, bradykinin, and kallikrein in the study groups.

ELISA parameters	Groups	No.	Mean ± SD	Differences
Prostaglandin E Synthase (ng/mL)	Hypothyroidism	35	206.74 ±161.33	A
	Hypothyroidism with hypertension	35	182.05 ±98.62	A
	Control	20	397.94± 229.48	B
Bradykinin (ng/mL)	Hypothyroidism	35	6.98± 3.93	A
	Hypothyroidism with hypertension	35	4.14± 2.86	B
	Control	20	11.58± 5.22	C
Kallikrein (mU/mL)	Hypothyroidism	35	160.12± 114.2	A
	Hypothyroidism with hypertension	35	143.67± 103.27	A
	Control	20	314.85± 187.16	B

*Groups with similar letters mean p>0.05, different letters mean p≤0.05. Source: Own authorship.

Discussion

The impact of hypothyroidism was also significant and quantifiable. The impairment of vasodilatory mechanisms was observed and even increased in participants presenting with concurrent hypertension. The present study demonstrated significant biochemical abnormalities of thyroid function, as indicated by significantly increased levels of thyroid-stimulating hormone (TSH) among hypothyroid participants. In participants presenting with concurrent hypothyroidism and hypertensionTSH levels were significantly high in hypothyroid participants compared to controls.

Furthermore, TSH levels were significantly high in participants who suffered from hypertension and hypothyroidism compared to controls. This pattern of significant TSH levels in hypothyroid participants and in participants who suffered from hypertension and hypothyroidism was observed in all cases. TSH levels were also significantly increased among participants presenting with concurrent hypothyroidism and hypertension. TSH levels were significantly increased among hypothyroid participants compared to controls. TSH levels were also significantly increased among participants presenting with concurrent hypothyroidism and hypertension. TSH levels were significantly increased among hypothyroid participants compared to controls. TSH levels were also significantly increased among participants presenting with concurrent hypothyroidism and hypertension.

The present results show that the kallikrein-kinin system, another important vasodilatory cascade, is inhibited in a manner that is statistically and physiologically significant. Specifically, the levels of bradykinin were reduced in hypothyroidism as well as in hypothyroidism associated with hypertension. This suggests a dose-effect relationship in the reduction of this important vasodilator. Likewise, kallikrein activity is reduced by half in hypothyroid participants as well as in participants with hypothyroidism and hypertension compared to healthy controls. This suggests a reduced synthesis of bradykinin and hence a reduced stimulation of endothelial release of nitric oxide and prostacyclin. Recent mechanistic and clinical studies have confirmed that reduced bradykinin signaling

contributes to endothelial dysfunction and reduced vasodilation as well as increased vascular stiffness. This is particularly true in hypertension when degradation and downregulation of the kinin system are enhanced **[13-16]**.

Concomitantly, prostaglandin E synthase levels were significantly reduced in hypothyroid participants (206.74 ± 161.33 ng/mL) as well as in hypothyroid participants with hypertension (182.05 ± 98.62 ng/mL) compared to controls **[16,17]**. This suggests a significant reduction in prostanoid-mediated vasodilation. Note that bradykinin is known to enhance prostaglandin synthesis. These biochemical disturbances appear clinically relevant in light of patient characteristics, as the hypothyroidism with hypertension group showed greater age and a much higher prevalence of obesity (BMI >30 in 54.29%), both of which are well-recognized amplifiers of endothelial dysfunction through oxidative stress, low-grade inflammation, and impaired receptor signaling **[18]**.

Recent comprehensive reviews and population-based studies confirm that the combination of hypothyroidism and hypertension exerts additive and sometimes multiplicative deleterious effects on vascular function, resulting in greater impairment than either one of these conditions alone and leading to a significantly increased long-term cardiovascular risk. Although restoration of euthyroidism with levothyroxine has been shown to improve endothelial function, contemporary interventional trials indicate that such improvement is often incomplete in participants with persistent hypertension or excess adiposity, emphasizing that thyroid hormone replacement alone may not normalize vasodilatory pathways **[18,19]**. Collectively, the marked quantitative downregulation of bradykinin, kallikrein, and prostaglandin E synthase in this report, in conjunction with severe thyroid hormone deprivation and presence of hypertension-related vascular stressors, provide strong evidence that suppression of multiple interlinked vasodilatory systems forms the basis of marked endothelial dysfunction in hypothyroidism complicated by hypertension **[20,21]**.

Limitations and Future

Future studies should also help clarify the role of reversible endothelial dysfunction, irreversible vascular remodeling, as well as inflammatory and plasma infiltration pathways, as well as the optimal levels of normalization of thyroid hormones to mitigate hypertension risk.

Conclusion

This study clearly indicates that endocrine and biochemical abnormalities are significant in participants with hypothyroidism, and the problem is further complicated by the presence of hypertension. The significant increase in TSH and the marked decrease in T3 and T4 clearly show that the thyroid dysfunction is severe when compared to the control groups. Furthermore, age and BMI may influence significant changes in ELISA-derived markers such as prostaglandin E synthase, bradykinin, and kallikrein. The study clearly indicates that the problem of hypothyroidism, along with hypertension, may increase the metabolic problems and accelerate the disease.

CRediT

Author contributions: **Conceptualization-** All authors; **Investigation-** All authors. **Methodology-** All authors; **Project administration-** All authors; **Supervision-**All authors; **Writing - original draft-** All authors; **Writing-review & editing-** All authors.

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Ethical Approval

The study was performed subsequent to receiving ethical permission from the Ethical permission Committee at Tikrit University (issue number: 3/7/314 at 6/11/2024). All participants were given a consent form before commencing specimen collection.

Informed Consent

It was applicable.

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The authors declare no conflict of interest.

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References

1. Kim MI, Bertot BE. Hypothyroidism in Older Adults. 2025 Dec 10. In: Feingold KR, Adler RA, Ahmed SF, Anawalt B, Blackman MR, Chrousos G, Corpas E, de Herder WW, Dhatariya K, Dungan K, Hamilton E, Hofland J, Jan de Beur S, Kalra S, Kaltsas G, Kapoor N, Kim M, Koch C, Kopp P, Korbonits M, Kovacs CS, Kuohung W, Laferrère B, Levy M, McGee EA, McLachlan R, Muzumdar R, Purnell J, Rey R, Sahay R, Shah AS, Sperling MA, Stratakis CA, Trence DL, Wilson DP, editors. Endotext **[Internet]**. South Dartmouth (MA): MDText.com, Inc.; 2000–. PMID: 25905236.
2. Guerrero-Pérez F, Iglesias P, Paja Fano M, Moure Rodríguez MD, Biagetti B, Cordido F, Novoa-Testa I, García-Centeno R, González Fernández L, Araujo-Castro M, Villar-Taibo R, García L, Fernández-Rodríguez E, Capristán-Díaz V, Simó-Servat A, Aulinas A, Asla Q, Tenorio-Jiménez C, Díez JJ; Neuroendocrinology Task Force of Spanish Society of Endocrinology and Nutrition. Central hypothyroidism and cardiovascular disease: a Spanish cohort observational study. Hormones (Athens). 2026 Mar;25(1):213-223. doi: 10.1007/s42000-025-00707-6.
3. Wiersinga WM. Adult Hypothyroidism. 2014 Mar 28. In: Feingold KR, Adler RA, Ahmed SF, Anawalt B, Blackman MR, Chrousos G, Corpas E, de Herder WW, Dhatariya K, Dungan K, Hamilton E, Hofland J, Jan de Beur S, Kalra S, Kaltsas G, Kapoor N, Kim M, Koch C, Kopp P, Korbonits M, Kovacs CS, Kuohung W, Laferrère B, Levy M, McGee EA, McLachlan R, Muzumdar R, Purnell J, Rey R, Sahay R, Shah AS, Sperling MA, Stratakis CA, Trence DL, Wilson DP, editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000–. PMID: 25905416.
4. Sheikhi V, Heidari Z. Association of Subclinical Hypothyroidism with Nonalcoholic Fatty Liver Disease in Participants with Type 2 Diabetes Mellitus: A Cross-Sectional Study. Adv Biomed Res. 2022 Dec 26;11:124. doi: 10.4103/abr.abr_15_21.
5. Chiu HH, Villanueva E 3rd, Larrazabal R Jr, Arcellana AE, Jimeno C. Characteristics and Prevalence of Metabolic Syndrome Among Adult Filipinos with Hypothyroidism: A Cross-sectional Study. J ASEAN Fed Endocr Soc. 2024;39(1):53-60. doi: 10.15605/jafes.039.01.13.
6. Kiran M, Ejaz S, Iqbal MN, Malik WN, Zahoor S, Ejaz SA. Hypothyroidism correlates with dyslipidemia and protein contents in participants with various metabolic disorders. J Int Med Res. 2022 Sep;50(9):3000605221119656. doi: 10.1177/03000605221119656.
7. Evsen A, Demir M, Günlü S. Evaluation of epicardial fat tissue and echocardiographic parameters in participants with silent enemy subclinical hypothyroidism. Echocardiography. 2022 Nov;39(11):1426-1433. doi: 10.1111/echo.15471.
8. Guo W, Hou LY, Yi X. Impact of Subclinical Hypothyroidism on Coagulation Parameters and Coronary Artery Disease Severity in Participants with Coronary Heart Disease. Cardiology. 2026;151(2):151-161. doi: 10.1159/000545904.
9. Chiu HHC, Larrazabal RB Jr, Arcellana AES, Jimeno CA. The Prevalence of Metabolic Syndrome among Adult Filipinos with Hypothyroidism: A Retrospective Cohort Study. Acta Med Philipp. 2023 Jul 27;57(7):31-37. doi: 10.47895/amp.vi0.4978.
10. Feingold KR. The Effect of Endocrine Disorders on Lipids and Lipoproteins. 2026 Mar 20. In: Feingold KR, Adler RA, Ahmed SF, Anawalt B, Blackman MR, Chrousos G, Corpas E, de Herder WW, Dhatariya K, Dungan K, Hamilton E, Hofland J, Jan de Beur S, Kalra S, Kaltsas G, Kapoor N, Kim M, Koch C, Kopp P, Korbonits M, Kovacs CS, Kuohung W, Laferrère B, Levy M, McGee EA, McLachlan R, Muzumdar R, Purnell J, Rey R, Sahay R, Shah AS, Sperling MA, Stratakis CA, Trence DL, Wilson DP, editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000–. PMID: 28121116.
11. Izkhakov E, Vaisman N, Barnes S, Barchana M, Stern N, Keinan-Boker L. Body Composition, Resting Energy Expenditure, and Metabolic Changes in Women Diagnosed with Differentiated Thyroid Carcinoma. Thyroid. 2019 Aug;29(8):1044-1051. doi: 10.1089/thy.2018.0483.
12. Von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE

Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453-7.

13. Korevaar TIM, Leung AM, Alexander EK, Bliddal S, Boelaert K, Brenta G, Chou R, Dhillon-Smith R, Dosiou C, Eaton JL, Guan H, Kilpatrick SJ, Lasserre BJ, Lee SY, Maraka S, Meister KD, Morris-Wiseman LF, Nguyen CT, Pearce EN, Shan Z. American Thyroid Association 2026 Guidelines for Thyroid Disease in Preconception, Pregnancy, and Postpartum. Thyroid. 2026 May;36(5):481-544. doi: 10.1177/10507256261445624.
14. Zhu L, Cheung YM, Chiang C, Gallagher EJ, Hamnvik OR, Mammen J, Quandt Z, Jones R, McDunn J, Al-Bahadili H, Angell T, Bancos I, Betof Warner A, Blackwell S, Calabrese C, Cho TA, Dougan M, Ferreira M, Funchain P, Herndon J, Johnson DB, Kennedy R, Kheterpal M, Khouri M, Lechner MG, Lim AM, Markova A, Meara A, Overton L, Perdigoto AL, Sachithanandan N, Sharon E, Spain L, Tsai K, Yarchoan M, Reynolds KL, Shariff A. Consensus-based disease definitions for endocrine immune-related adverse events of immune checkpoint inhibitors. J Immunother Cancer. 2025 Oct 15;13(10):e011865. doi: 10.1136/jitc-2025-011865.
15. Delić T, Karanović Štambuk S. Iodine in Health and Disease: A Comprehensive Review. Nutrients. 2026 Apr 16;18(8):1262. doi: 10.3390/nu18081262.
16. Alharbi A, Alsaawi OK, Alhablen SA, Aljumah MK, Alharbi IK, Alshowaish MK. Prevalence and Types of Anemia in Participants With Hypothyroidism in Buraidah, Saudi Arabia. Cureus. 2026 Jul 8;18(7):e112269. doi: 10.7759/cureus.112269.
17. Liu D, Zhang Y, Chen Z, Hong Y, Liu T, Chen LH. Ultra-processed food consumption and incident autoimmune-related hypothyroidism: a sex-stratified prospective analysis from the UK Biobank. Am J Clin Nutr. 2026 Feb;123(2):101142. doi: 10.1016/j.ajcnut.2025.101142.
18. Bortolotto VC, Zborowski VA, Dahleh MMM, Borstmann SMA, Viana CE, Pinheiro FC, Balok FRM, Meichtry LB, Boeira SP, Guerra GP, Nogueira CW, Prigol M. Neuroinflammation in hypothyroidism? Chrysin treatment as a strategy to reduce neuroinflammation induced by hypothyroidism. Neuroscience. 2025 Oct 1;584:395-403. doi: 10.1016/j.neuroscience.2025.08.038.
19. Iglesias P. Hormone deficiency and cardiovascular disease: clinical impact, treatment strategies and prognostic prediction. Expert Rev Cardiovasc Ther. 2026 Apr;24(4):323-338. doi: 10.1080/14779072.2026.2649164.
20. Kochar A, Kaur H, Dayal D. Effect of timely diagnosis and treatment on growth and body proportionality of children with congenital hypothyroidism. Front Endocrinol (Lausanne). 2026 Jan 28;16:1713739. doi: 10.3389/fendo.2025.1713739.
21. Lado-Abeal J, Munir I, Al-Zubaidi K, Mohammed S, Tadisina S, Stiewig-Rapp MJ, Arvandi A, Whisenant T, Ginzo-Villamayor MJ, Tejera-Perez C, Perez-Castro P, Fernandez-Rodriguez E, Guo A, Surampudi P, Bhattacharya R, Xiang YK, Smith TW, Fernandez-Pombo A. Heart ventricular function in hospitalized participants with severe hypothyroidism and myxedema coma. J Clin Endocrinol Metab. 2026 Jun 17;111(7):2016-2026. doi: 10.1210/clinem/dgag036.