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Association of High-Dose Coenzyme Q10 with Thyroid Autoimmunity and Thyroid Function in Hypothyroid Patients- A Prospective Observational Study.

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Abstract

Background: Autoimmune hypothyroidism, characterised by elevated anti-thyroid peroxidase (anti-TPO) antibodies, contributes to progressive thyroid dysfunction and increased dependency on levothyroxine therapy. Oxidative stress is implicated in autoimmune thyroid disease pathogenesis. Coenzyme Q10 (CoQ10), a potent antioxidant, may modulate immune activity and improve thyroid function. ***Objective:*** To evaluate the association between high-dose Coenzyme Q10 supplementation and changes in Anti-TPO antibody levels and thyroid function parameters in patients with hypothyroidism. ***Methods:*** This prospective observational study was conducted at Amar Hospital, Mohali, Punjab, India, over a period of six months. A total of 50 patients aged 30–70 years with hypothyroidism and Anti-TPO levels greater than 100 IU/mL were included. All participants received Coenzyme Q10 supplementation at a dose of 300 mg orally once daily in addition to their routine levothyroxine therapy. Thyroid autoimmunity and thyroid function parameters including Anti-TPO, FT3, FT4, and TSH were measured at baseline and after six months. Statistical analysis was performed using SPSS version 25. Paired t-test or Wilcoxon signed-rank test was applied where appropriate, and a p-value <0.05 was considered statistically significant. ***Results:*** The study included 50 participants with a mean age of 48.7 ± 10.6 years; 34 (68%) were females and 16 (32%) were males. A significant reduction in Anti-TPO antibody levels was observed after six months of CoQ10 supplementation. The mean baseline Anti-TPO level decreased from 554.3 IU/mL to 244.9 IU/mL, with a mean reduction of −309.3 IU/mL (95% CI: −359.2 to −259.5; $p < 0.001$). Thyroid function parameters showed variable responses. FT3 and FT4 levels remained stable during the study period with no statistically significant changes ($p = 0.36$ and $p = 0.82$, respectively). In contrast, TSH levels demonstrated a significant reduction from 10.95 mIU/L to 7.71 mIU/L (mean difference −3.24 mIU/L; $p < 0.001$). Levothyroxine doses remained stable in most patients, with the majority receiving 50 mcg/day. ***Conclusion:*** High-dose Coenzyme Q10 supplementation was associated with a significant reduction in Anti-TPO antibody levels and improvement in TSH levels in patients with hypothyroidism, while FT3 and FT4 remained stable. These findings suggest that CoQ10 may have a potential adjunctive role in reducing thyroid autoimmunity and improving hormonal regulation in hypothyroid patients. Further large-scale randomized controlled trials are required to confirm these findings and establish the therapeutic role of CoQ10 in autoimmune thyroid disease.

Keywords: Hypothyroidism, Coenzyme Q10, Anti-TPO antibodies, Thyroid autoimmunity, TSH, Antioxidants.

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INTRODUCTION

Hypothyroidism is an endocrine disorder defined as an inadequate thyroid gland function or an inability of thyroid hormones to act on target tissues [1]. The clinical presentation of patients can vary from asymptomatic disease to myxedema coma [2]. Hypothyroidism is characterized by low thyroid (triiodothyronine and tetraiodothyronine) and elevated thyroid-stimulating hormone (TSH) [3]. Thyroid hormones are important in the activity of metabolism [4]. These hormones are active in the functioning of many organs related to the heart, brain, intestines, and reproductive system [5]. Thyroid hormones exert a major influence from the fetal period to adulthood by controlling gene and protein expression in almost every tissue [6].

Coenzyme Q10 (CoQ10), also known as ubiquinone, is a fat-soluble, vitamin-like molecule naturally present in every cellular membrane within our bodies. This enzyme is a regular component of our diet, although it is also synthesized endogenously. CoQ10 is crucial for efficiently transferring electrons within the mitochondrial oxidative respiratory chain and producing adenosine triphosphate (ATP). CoQ10 can potentially increase the production of vital antioxidants, such as

superoxide dismutase, an enzyme that effectively mitigates vascular oxidative stress in individuals with hypertension. In addition, CoQ10 lowers lipid peroxidation levels by diminishing pro-oxidative compounds. Furthermore, CoQ10 can improve blood flow and safeguard blood vessels by preserving nitric oxide. [7]

Looking at levels in human plasma, it has been shown that the concentration of CoQ10 is different in hypothyroid subjects compared to healthy subjects and that there is a significant inverse correlation between CoQ10 levels and thyroid hormone levels.[8] Emerging evidence suggests that oxidative stress plays a central role in autoimmune mechanisms. Coenzyme Q10, a mitochondrial electron transport cofactor, exhibits strong antioxidant and anti-inflammatory properties. While its role has been explored in cardiovascular and metabolic diseases, limited data exist regarding its impact on autoimmune thyroid conditions.[9,10]

This study aimed to evaluate the association between high-dose CoQ10 supplementation and changes in anti-TPO antibody levels and thyroid function.

MATERIALS AND METHODS

The present prospective observational study was conducted at Amar Hospital, Mohali, Punjab, India for a period of 6 months among patients of hypothyroidism. Ethical clearance for conducting the research was taken from institute's ethics committee of hospital before commencement of study. Written informed consent was taken from patients after explaining them about procedure of study.

Through consecutive sampling a total of 50 patients diagnosed with hypothyroidism were selected for the study on the basis of inclusion and exclusion criteria.

Inclusion criteria-

1. Patients diagnosed with hypothyroidism
2. Patients with Anti-TPO >100 IU/mL
3. Patients with Age 30–70 years

Exclusion criteria-

1. Patients with Anti-TPO <100 IU/mL
2. Patients who were taking steroids or immunosuppressive therapy

All enrolled participants received Coenzyme Q10 supplementation at a dose of 300 mg orally once daily for a total duration of six months. The supplement was administered in addition to their routine thyroid hormone replacement therapy (levothyroxine), which was continued as prescribed by the treating physician. Compliance with the supplementation regimen was monitored through monthly follow-up visits and patient self-reporting.

At the time of enrollment, demographic and clinical details including age, gender, duration of hypothyroidism, current levothyroxine dose, and relevant medical history were recorded. Blood samples were collected under standardized laboratory conditions for the evaluation of thyroid function parameters and autoimmune markers.

Laboratory Investigations- Laboratory assessments were performed at baseline (before initiation of CoQ10 supplementation) and repeated after six months of treatment.

The following biochemical parameters were measured:

- Anti-thyroid peroxidase antibodies (Anti-TPO)
- Free triiodothyronine (FT3)
- Free thyroxine (FT4)
- Thyroid stimulating hormone (TSH)

Venous blood samples were obtained after an overnight fast. Serum was separated and analyzed using standard automated immunoassay techniques in the hospital's central laboratory.

Outcome Measures- The primary outcome measure of the study was: Change in Anti-TPO antibody levels between baseline and 6 months after CoQ10 supplementation. Secondary Outcomes measure was: Changes in thyroid function parameters (FT3, FT4, and TSH), Changes in the required dose of levothyroxine and Overall biochemical improvement in thyroid function during the study period

Statistical Analysis- Data were entered and analyzed using Statistical Package for the Social Sciences (SPSS) software version 25.0. Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data and median with interquartile range (IQR) for non-normally distributed data. Categorical variables were presented as frequencies and percentages. To evaluate changes between baseline and post-intervention values: Paired t-test was used for normally distributed variables, Wilcoxon signed-rank test was used for non-normally distributed variables. Effect sizes

were reported using mean differences along with 95% confidence intervals (CI). A two-sided p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1 shows baseline characteristics of patients. A total of 50 patients with hypothyroidism were included in the study. The mean age of participants was 48.7 ± 10.6 years, with an age range of 30–70 years. The study population included 34 females (68%) and 16 males (32%), reflecting the higher prevalence of hypothyroidism among females.

Table 1 Baseline characteristics of patients

Variable	Value
Age (years), mean ± SD	48.7 ± 10.6
Age range	30–70
Female	34 (68%)
Male	16 (32%)

Table 2 shows a significant reduction in anti-thyroid peroxidase (Anti-TPO) antibody levels was observed after 6 months of Coenzyme Q10 supplementation. The mean baseline Anti-TPO level was 554.3 IU/mL, which decreased to 244.9 IU/mL at 6 months. The mean reduction was −309.3 IU/mL (95% CI: −359.2 to −259.5) and the difference was statistically significant ($p < 0.001$).

Table 2 Change in Anti-TPO Antibody Levels

Parameter	Baseline Mean	6 Months Mean	Mean Difference	95% CI	p-value
Anti-TPO (IU/mL)	554.3	244.9	−309.3	−359.2 to −259.5	<0.001

Table 3 presents the changes in thyroid function parameters (FT3, FT4, and TSH) between baseline and after six months of Coenzyme Q10 supplementation among the study participants. At baseline, the mean FT3 level was 2.86 pg/mL, which slightly increased to 2.87 pg/mL after six months, showing a mean difference of +0.02 pg/mL. However, this change was not statistically significant ($p = 0.36$). Similarly, FT4 levels remained stable during the study period. The mean baseline FT4 was 1.15 ng/dL, which marginally increased to 1.15 ng/dL at follow-up, with a mean difference of +0.003 ng/dL. The change was statistically non-significant ($p = 0.82$). In contrast, a significant reduction in serum TSH levels was observed. The mean baseline TSH was 10.95 mIU/L, which decreased to 7.71 mIU/L after six months, corresponding to a mean reduction of −3.24 mIU/L. This change was highly statistically significant ($p < 0.001$). The reduction in TSH indicates an improvement in thyroid function control during the study period.

Table 3 Changes in Thyroid Function Parameters

Parameter	Baseline Mean	6 Months Mean	Mean Difference	p-value
FT3 (pg/mL)	2.86	2.87	+0.02	0.36
FT4 (ng/dL)	1.15	1.15	+0.003	0.82
TSH (mIU/L)	10.95	7.71	−3.24	<0.001

Table 4 shows the distribution of levothyroxine doses among the study participants at baseline. The most commonly prescribed dose was 50 mcg/day, which was used by 13 patients (26%), making it the predominant maintenance dose in the study population. The next most frequent doses were 75 mcg/day, prescribed to 9 patients (18%), followed by 62.5 mcg/day, used by 6 patients (12%). Lower doses such as 25 mcg/day and 32.5 mcg/day were prescribed to 5 patients (10%) and 6 patients (12%), respectively. Similarly, 37.5 mcg/day was administered to 4 patients (8%). Higher doses of 100 mcg/day and 125 mcg/day were used in 4 patients (8%) and 3 patients (6%), respectively.

Table 4 Distribution of Levothyroxine Dose Among Participants

Dose (mcg/day)	Number of Patients	Percentage
25 mcg	5	10%
32.5 mcg	6	12%
37.5 mcg	4	8%
50 mcg	13	26%
62.5 mcg	6	12%
75 mcg	9	18%
100 mcg	4	8%
125 mcg	3	6%

DISCUSSION

The leading biomarker for detecting autoimmune thyroid disease is the determination of TSH concentration. [11] Our study evaluated the association between high-dose Coenzyme Q10 (CoQ10) supplementation and thyroid autoimmunity as well as thyroid function parameters in patients with hypothyroidism. Studies done in past underscores the importance of TPO-Ab as a biomarker in detection of thyroid disease in general population. Some earlier prospective studies investigated TSH and TPO-Ab concentrations in patients with TSH concentrations within reference values. Thyroid disease can be caused by too little or excessive iodine intake. [12,13] The results of present study showed that after six months of taking CoQ10, Anti-TPO antibody levels went down a lot and TSH levels went down a lot, but FT3 and FT4 levels stayed about the same. These results indicate that CoQ10 may play a role in regulating autoimmune activity and enhancing biochemical thyroid function in individuals with hypothyroidism.

In our study the mean age of participants was 48.7 ± 10.6 years, with an age range of 30–70 years. The study population included 34 females (68%) and 16 males (32%), reflecting the higher prevalence of hypothyroidism among females. Chronic autoimmune thyroiditis affects 1%-2% of the total population and is one of the most common thyroid diseases. [14] The disease incidence is particularly increased in people aged between 30 and 50 years but can occur in any age group, including children.[15,16] Hypothyroidism most often occurs in areas with sufficient iodine concentration. The annual incidence of chronic autoimmune thyroiditis globally is estimated to be 0.3-1.5 per 1000 individuals, and it is 10-15 times higher in women.[17-19] The disease is more common today, not because of frequent examinations but because of the more potent immunogenicity of thyroid antigens caused by increased iodine intake.[14,15]

Several antibodies and antigen-specific T lymphocytes have been linked to autoimmune thyroid disease.[20] The significant antigens are thyroglobulin (TG), thyroid peroxidase (TPO), and the thyroid-stimulating hormone receptor (TSH receptor, thyrotropin receptor). Antibodies are highly cytotoxic microsomal antibodies to thyroid peroxidase, thyrocyte membrane antigens, and/or binding complement. These antibodies cause destruction and lysis of thyrocytes .[21] Thyroglobulin, synthesized in follicular cells, acts as both a precursor and a storage molecule for thyroid hormones and is one of the primary targets of autoimmunity in chronic autoimmune thyroiditis.[22] Studies have shown that more than 14 polymorphisms in the thyroglobulin gene are associated with an increased incidence of chronic autoimmune thyroiditis.[23] In addition, thyroid peroxidase catalyzes the iodination of tyrosine residues in thyroglobulin. Experimental autoimmune thyroiditis was induced using thyroglobulin or TPO as an antigen in mice, demonstrating the role of thyroglobulin and TPO in the pathogenesis of chronic autoimmune thyroiditis.[24] In the present study, the mean Anti-TPO antibody level decreased significantly from 554.3 IU/mL at baseline to 244.9 IU/mL after six months of CoQ10 supplementation, indicating a substantial reduction in thyroid autoimmunity. This observation supports the hypothesis that antioxidant therapy may help attenuate autoimmune-mediated inflammation within the thyroid gland.

Hypothyroidism results from autoimmune damage to the thyroid, in which autoantibodies are directed against the thyroid antigens thyroid peroxidase and thyroglobulin. It is worth noting that thyroid hormones regulate cellular energy metabolism via their effects on mitochondrial function. Mitochondria are major sites of triiodothyronine accumulation within cells, where it exerts a direct effect on mitochondrial activity and energy metabolism. Mitochondria are a major source of free radical production within cells, and the accelerating effect of triiodothyronine on basal metabolism results in an increased production of free radicals. The hypermetabolic state present in hyperthyroidism results in excessive free-radical-induced oxidative stress within cells; in contrast, the hypometabolic state induced by hypothyroidism leads to a decrease in free radical production.[25] Patients with hypothyroidism may have similar circulatory levels of CoQ10 to normal subjects or substantially increased levels, precluding the necessity of CoQ10 supplementation.[9]

This study also demonstrated a significant decrease in TSH levels from 10.95 mIU/L to 7.71 mIU/L after six months of supplementation. TSH is a key indicator of thyroid function and is commonly elevated in patients with hypothyroidism due to reduced thyroid hormone production. However, despite the improvement in TSH levels, FT3 and FT4 levels did not show statistically significant changes during the study period. Although this finding should be confirmed by studies with larger sample sizes of patients, the results suggest that measuring the concentration of TPO-Ab and TSH can be used as a marker to identify individuals at risk of developing hypothyroidism in the general population.[13]

The distribution of levothyroxine doses among the participants also reflects the heterogeneity of hypothyroidism severity in the study population. The most commonly prescribed dose in this study was 50 mcg/day, followed by 75 mcg/day and 62.5 mcg/day, which is consistent with typical dosing patterns for patients with mild to moderate hypothyroidism. In their study, Okuroglu et al found a positive correlation between the antibody concentration and higher levothyroxine dosing in patients with autoimmune thyroiditis.[26]

Notwithstanding these encouraging results, it is important to recognise a number of the study's limitations. First, the study was carried out at a single facility with a rather small sample size of 50 patients, which would restrict how broadly the findings can be applied. Second, it was challenging to substantiate a clear causal link between CoQ10 supplementation and the noted improvements in thyroid autoimmunity since the study design was observational and lacked a control group. Third, the duration of follow-up was limited to six months, and longer-term effects of CoQ10 supplementation on thyroid autoimmunity and hormone regulation remain unknown.

To further explore the therapeutic potential of CoQ10 in autoimmune thyroid illness, future studies should concentrate on large-scale randomised controlled trials. To better understand the processes underlying the reported effects, such studies should incorporate longer follow-up periods, control groups, and additional indicators of immunological activation and oxidative stress. Additionally, assessing CoQ10's effects on clinical symptoms, quality of life, and long-term thyroid function results would yield important information on its function in hypothyroidism care.

CONCLUSION

This prospective observational study concluded that high-dose Coenzyme Q10 supplementation significantly reduced Anti-TPO antibody levels in hypothyroid patients after six months of treatment. A notable reduction in TSH levels was detected, indicating enhanced regulation of thyroid function. Nonetheless, FT3 and FT4 levels remained constant, suggesting that CoQ10 predominantly affected autoimmune activity rather than directly modifying circulating thyroid hormone levels. The data indicate that Coenzyme Q10 may serve a helpful supplementary role in diminishing thyroid autoimmune and enhancing biochemical markers of thyroid function. The antioxidant and anti-inflammatory characteristics of CoQ10 may facilitate these effects. Nonetheless, the findings must be regarded with care owing to the observational study design and restricted sample size. Additional extensive randomised controlled trials are advised to validate the therapeutic efficacy of CoQ10 in autoimmune hypothyroidism.

REFERENCES

1. Almandoz JP, Gharib H. Hypothyroidism: Etiology, diagnosis, and management. Med Clin North Am. 2012;96(2):203-221.
2. Taylor PN, Albrecht D, Scholz A, et al. Global epidemiology of hyperthyroidism and hypothyroidism. Nat Rev Endocrinol. 2018;14(5):301-316.
3. Aktümsek A. Genel Endokrinoloji. 1st edn, Ankara, Nobel Yayınevi, 2020.
4. Schwarz C, Leichtle AB, Arampatzis S, et al. Thyroid function and serum electrolytes: Does an association really exist? Swiss Med Wkly. 2012;142:w13669.
5. Ladenson PW, Singer PA, Ain KB, et al. American Thyroid Association guidelines for detection of thyroid dysfunction. Arch Intern Med. 2000 Jun 12;160(11):1573- 1575.
6. Giannocco G, Kizys MML, Maciel RM, de Souza JS. Thyroid hormone, gene expression, and central nervous system: Where we are. Semin Cell Dev Biol. 2021;114:47- 56.
7. Du J, Wang T, Huang P, Cui S, Gao C, Lin Y, Fu R, Shen J, He Y, Tan Y, Chen S. Clinical correlates of decreased plasma coenzyme Q10 levels in patients with multiple system atrophy. Parkinsonism Relat Disord. 2018 Dec;57:58-62.
8. Jiang P, Wu M, Zheng Y, et al. Analysis of coenzyme Q(10) in human plasma by column-switching liquid chromatography. J Chromatogr B Analyt Technol Biomed Life Sci. 2004;805(2):297-301.
9. Ogura F, Morii H, Ohno M, et al. Serum coenzyme Q10 levels in thyroid disorders. Horm Metab Res. 1980;12(10):537-540.
10. Atkinson Jr AJ, Colburn WA, DeGruttola VG, DeMets DL, Downing GJ, Zeger SL. Biomarkers Definitions Working Group, Biomarkers and surrogate endpoints: Preferred definitions and conceptual framework. Clin Pharm Therap. 2001;69(3),89-95.
11. D'Aurizio F, Tozzoli R, Villalta D, Pesce G, Bagnasco M. Immunoassay of thyroid peroxidase autoantibodies: diagnostic performance in automated third generation methods. A multicentre evaluation. Clin Chem Lab Med. 2015;53(3):415-21.
12. Prpić M, Franceschi M, Vidranski V, et al. Iodine status and thyroid function in lactating women and infants – a survey in the Zagreb area, Croatia. Acta Clin Croat. 2021;60(2):259- 67.
13. Vasilj-Mihaljević M, Mihaljević I, Bekić S, Zubčić Ž, Vasilj A, Daus-Šebeđak D, Škrlec I, Talapko J. Correlation of TPO Antibody Concentration with Thyroid Hormones as a Predictor of Clinical Hypothyroidism. Acta Clinica Croatica. 2024 Dec;63(3-4):542.
14. Wiersinga WM.Hashimoto's thyroiditis. In: Vitti P, Hegedüs L, editors. Thyroid Diseases. Endocrinology. Springer, Cham; 2018:205-247.
15. Bečejac B, Solter M. Upale štitnjače. In: Solter M, editor. Bolesti štitnjače – klinička tireoidologija. Zagreb, Croatia: Medicinska naklada, 2007;145-53.

16. Chiovato L, Magri F, Carlé A. Hypothyroidism in context: where ' 'we've been and where ' 'we're going. Adv Ther. 2019;36(2):47-58.
17. Iddah MA, Macharia BN. Autoimmune thyroid disorders. ISRN Endocrinol. 2013;2013:1-9.
18. Vanderpump MPJ, Tunbridge WMG, French JM, et al. The incidence of thyroid disorders in the community: A twentyyear follow-up of the Whickham Survey. Clin Endocrinol (Oxf). 1995;43(1):55-68.
19. Vanderpump MPJ, French JM, Appleton D, Tunbridge WMG, Kendall-Taylor P. The prevalence of hyperprolactinaemia and association with markers of autoimmune thyroid disease in survivors of the Whickham Survey cohort. Clin Endocrinol (Oxf). 1998;48(1):39-44.
20. Ramos-Leví AM, Marazuela M. Pathogenesis of thyroid autoimmune disease: the role of cellular mechanisms. Endocrinol y Nutr (English Ed. 2016;63(8):421-429.
21. Fröhlich E, Wahl R. Thyroid autoimmunity: role of antithyroid antibodies in thyroid and extra-thyroidal diseases. Front Immunol. 2017;8:521.
22. Thanas C, Ziros PG, Chartoumpekis D V., Renaud CO, Sykiotis GP. The Keap1/Nrf2 signaling pathway in the thyroid—2020 update. Antioxidants. 2020;9(11):1082.
23. Jacobson EM, Tomer Y. The genetic basis of thyroid autoimmunity. Thyroid. 2007;17(10):949-61.
24. Matsuoka N, Unger P, Ben-Nun A, Graves P, Davies T. Thyroglobulin-induced murine thyroiditis assessed by intrathyroidal T cell receptor sequencing – PubMed. J Immunol. 1994;152(5):2562-2568.
25. Mantle D, Hargreaves IP. Coenzyme Q10 and autoimmune disorders: An overview. International journal of molecular sciences. 2024 Apr 22;25(8):4576.
26. Okuroglu N, Ozdemir A, Sertbas Y, Sancak S. The relationship between thyroid antibody titer and levothyroxine dose in patients with overt primary hypothyroidism. Ann Saudi Med. 2017;37(3):189-93.