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ASSOCIATION OF THYROID AUTOIMMUNITY WITH POST-COVID-19 TELOGEN EFFLUVIUM IN FEMALES: A CASE–CONTROL STUDY

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ABSTRACT

Introduction: Telogen effluvium is a common cause of diffuse hair loss following systemic stress. There were many reports documented post-COVID-19-telogen effluvium. Thyroid hormones have important role in the hair follicle cycle. In this study we investigated thyroid hormones profile including thyroid autoantibodies in cases of post-COVID-19-telogen effluvium
Aims: To evaluate the association between thyroid function, autoimmune markers, and post-COVID-19 telogen effluvium in females.
Settings and Design: A case–control study was conducted in Erbil Dermatology Teaching Center, Kurdistan Region, Iraq.
Methods and Material: Study conducted including 107 female patients with post-COVID-19 telogen effluvium and 99 age-matched controls without hair loss. Thyroid profile parameters (free T3, Free T4, TSH, Anti-TPO, Anti-Tg) obtained from the patients and control group subjects.
Statistical analysis used: Means of the thyroid parameters been calculated from both groups and compared. Multivariate logistic regression and ROC curve analysis were performed.
Results: Patients with hair loss had significantly higher levels of fT4, TSH, and Anti-TPO antibody ($p < 0.001$). Anti-TPO was the only independent predictor (OR = 1.06, 95% CI: 1.03–1.08). ROC analysis demonstrated excellent diagnostic performance of Anti-TPO antibody (AUC = 0.943) with an optimal cutoff of 7 IU/mL.
Conclusions: Post-COVID-19 telogen effluvium is strongly associated with thyroid autoimmunity. Anti-TPO is a valuable predictive biomarker and should be considered in the evaluation of affected patients.

KEYWORDS: Telogen effluvium, COVID-19, Thyroid autoimmunity, Anti-TPO, Hair loss

INTRODUCTION

Many COVID-19 survivors report telogen effluvium (TE) weeks to months after infection [1,2]. Evidence of COVID-19 era suggests that up to one-quarter of patients may experience significant post-COVID-19-TE [3].
Telogen effluvium is a diffuse, non-scarring hair-shedding disorder typically triggered by systemic stressors such as febrile illness, surgery, or the postpartum state [4]. Severe infections often induce an intense inflammatory response (cytokine storm), including high levels of IL-6, IL-1β, and TNF-α, which can prematurely shift hair follicles from the growth phase (anagen) into the resting phase (telogen) [5]. In addition, a case series of 19 patients reported that 95% of post-COVID19-TE were women, and hair loss began approximately 2.7 months after COVID-19 diagnosis [6]. The epidemiology of post-COVID-19-telogen effluvium suggests a relatively high prevalence, estimated at approximately 20–30%, with a clear women predominance; however, the specific contributing risk factors beyond systemic illness-related stress remain poorly understood [3,7].
Thyroid hormones play a critical role in regulating hair follicle cycling and keratinocyte proliferation; therefore, thyroid dysfunction or autoimmunity may exacerbate or independently trigger telogen phase transition [8]. These thyroid abnormalities may range from transient inflammatory thyroiditis to persistent autoimmune thyroid disease [9]. Sparingly, thyroid disorders are a common cause of diffuse hair thinning. Both hypothyroidism and hyperthyroidism can slow hair growth and produce diffuse alopecia [10,11]. There is growing evidence that SARS-CoV-2 infection can directly affect the thyroid. The thyroid gland expresses ACE2 receptors, making it susceptible to viral invasion. Case series have documented new-onset subacute thyroiditis, Graves' disease, and Hashimoto's thyroiditis within weeks of COVID-19 [12]. In one report of three patients, a previously healthy 38-year-old woman developed profound hypothyroidism (TSH > 136 mIU/L) with extremely high anti-TPO antibodies (>900 IU/mL) about 6 weeks post-infection [12]. Additionally, population studies show that COVID-19 provokes a broad autoantibody response. A proteomics analysis of healthcare workers found that anti-thyroid peroxidase (anti-TPO) antibodies were among the most common new autoantibodies induced by SARS-CoV-2 infection [13]. Inflammatory mechanisms may involve molecular mimicry, bystander T-cell activation, or epitope spreading as the thyroid becomes inflamed. Thus, SARS-CoV-2 can precipitate thyroid autoimmunity in previously euthyroid patients [6,13]. Despite these observations, no controlled studies have quantitatively evaluated thyroid dysfunction and thyroid autoimmunity as potential contributing mechanisms in psot-COVID-19–telogen effluvium. This case–control design allows assessment of thyroid-related variables as potential predictors of post-COVID-19 telogen effluvium.

SUBJECTS AND METHODS

A comparative case–control study was conducted at Erbil Dermatology Teaching Center, Kurdistan Region, Iraq. The study included retrospectively collected data of female patients who had recovered from COVID-19 infection and subsequently developed diffuse hair loss consistent with telogen effluvium (TE). These cases were compared with age-matched women without hair loss recruited from the same center.

Inclusion criteria comprised female patients aged ≥16 years with a confirmed history of COVID-19 infection and subsequent onset of diffuse hair shedding. Patients with known thyroid disorders, pregnancy, postpartum status, or other causes of hair loss were excluded.

Data collected included age, time of onset of hair loss after COVID-19 infection (in months), and thyroid profile parameters, including free triiodothyronine (fT3), free thyroxine (fT4), thyroid-stimulating hormone (TSH), anti-thyroid peroxidase antibodies (anti-TPO Ab), and anti-thyroglobulin antibodies (anti-Tg Ab). Telogen effluvium was diagnosed clinically based on diffuse, non-scarring hair shedding following systemic illness, and by using dermatoscope by assessing the hair follicle opening, hair shafts, and excluding findings of other causes of non-scarring alopecia like alopecia areata and androgenetic alopecia.

Statistical analysis was performed using SPSS version 26 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation and compared using independent t-tests or Mann–Whitney U tests as appropriate. Effect size was calculated using Cohen's d. Receiver operating characteristic (ROC) curve analysis was conducted to assess the predictive performance of thyroid parameters for post-COVID-19 telogen effluvium. Results were reported with 95% confidence intervals, and a p-value < 0.05 was considered statistically significant.

The study was approved by the institutional ethical committee of Hawler Medical University, Erbil City, Kurdistan Region, Iraq, and conducted in accordance with the declaration of Helsinki.

RESULTS

A total of 107 female patients with post-COVID-19 telogen effluvium and 99 age-matched controls were included. The mean age was comparable between groups (27.00 ± 8.15 vs 26.55 ± 8.77 years). The mean onset of hair fall following COVID-19 infection was 3.03 ± 1.58 months.

Table 1. Baseline characteristics of study participants

Variable	Telogen effluvium (n=107)	Control (n=99)
Sample size	107	99
Age (mean ± SD)	27.00 ± 8.15	26.55 ± 8.77
Age range (years)	16–50	18–51
Onset of hair fall (months)	3.03 ± 1.58	N/A
Onset range	1–7	N/A

Significant differences were observed in thyroid parameters between groups. Patients with TE demonstrated significantly higher levels of T4, TSH, and Anti-TPO compared to controls ($p < 0.001$), whereas T3 levels did not differ significantly.

Table 2. Comparison of thyroid parameters between groups

Parameter	Telogen effluvium (mean ± SD)	Control (mean ± SD)	p-value
T3	2.44 ± 0.64	2.31 ± 0.38	0.096
T4	170.46 ± 95.21	120.76 ± 17.56	<0.001
TSH	2.55 ± 1.66	1.86 ± 1.12	0.0005
Anti-TPO	98.72 ± 145.66	10.06 ± 27.51	<0.001

Chi-square analysis demonstrated a significantly higher prevalence of abnormal thyroid parameters in patients with telogen effluvium compared to controls. Elevated Anti-TPO and Anti-thyroglobulin antibodies were strongly associated with TE, with odds ratios of 12.63 and 14.27, respectively ($p < 0.001$). Elevated T4 levels were also significantly associated with TE (OR = 6.71, $p < 0.001$). In contrast, TSH showed a borderline association, while T3 was not significantly associated with hair loss. These findings highlight the strong link between thyroid autoimmunity and post-COVID telogen effluvium.

Table 3. Comparison of elevated thyroid parameters between groups

Parameter	Telogen effluvium	Control	Odds Ratio (OR)	p-value
T3	14	8	1.71	0.349
T4	46	10	6.71	<0.001
TSH	11	3	3.67	0.073
Anti-TPO	43	5	12.63	<0.001
Anti-Tg	33	3	14.27	<0.001

ROC curve analysis demonstrated excellent predictive performance of combined thyroid parameters for post-COVID telogen effluvium (AUC = 0.922), indicating a strong association between thyroid dysfunction, particularly autoimmune markers, and hair loss following COVID-19 infection.

Table 4. ROC analysis of combined thyroid parameters

Model	AUC
Combined thyroid model (T3, T4, TSH, Anti-TPO)	0.922

Multivariate analysis identified Anti-TPO as the only statistically significant independent predictor of post-COVID-19 telogen effluvium (OR = 1.06, 95% CI: 1.03–1.08), supporting a central role of autoimmune thyroid activation in the pathogenesis of hair loss. In contrast, TSH and T4 demonstrated limited predictive value, while T3 showed no association. These findings suggest that post-COVID hair loss is primarily driven by immune-mediated mechanisms rather than isolated hormonal imbalance.

Table 5. Multivariate Logistic Regression Analysis

Variable	Odds Ratio (OR)	95% CI
T3	0.682	0.291–1.602
T4	1.010	0.993–1.027
TSH	1.257	0.915–1.726
Anti-TPO	1.059	1.033–1.084

Receiver operating characteristic (ROC) analysis demonstrated excellent diagnostic performance of Anti-TPO in predicting post-COVID-19 telogen effluvium, with an area under the curve (AUC) of 0.943. The optimal cutoff value was identified at 7 IU/mL, yielding a sensitivity of 95.3% and specificity of 89.9%. These findings indicate that Anti-TPO is a highly accurate biomarker for identifying patients at risk of developing hair loss following COVID-19 infection.

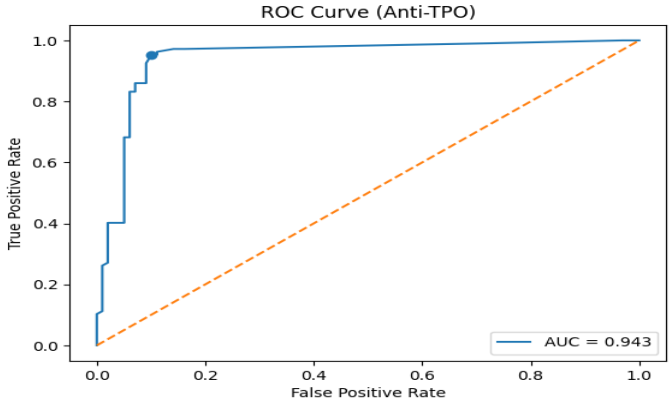


Figure 1: ROC Curve for Anti-TPO value cut-off

DISCUSSION

This study demonstrates a significant association between thyroid dysfunction, particularly autoimmune thyroid activity, and post-COVID-19-TE in females. Patients with TE exhibited significantly elevated levels of T4, TSH, and Anti-TPO compared to controls, with Anti-TPO emerging as the strongest independent predictor in multivariate analysis. Additionally, the onset of hair loss at approximately three months post-infection aligns with the classical timeline of telogen effluvium following systemic stress [1,14].

The findings support the two main factors triggering post-COVID hair loss: cytokine-mediated hair cycle disruption and autoimmune activation [15].

COVID-19 infection induces a systemic inflammatory response characterized by elevated cytokines such as IL-6 and TNF-α. These mediators are known to prematurely shift hair follicles from the anagen phase into the telogen phase, resulting in delayed shedding approximately 2–3 months later [15,16].

In the calculation of number of subjects with raised thyroid parameters between both groups, we found that there is markedly higher prevalence of autoimmune thyroid markers in patients compared to controls strongly supports an immune-mediated mechanism underlying post-COVID telogen effluvium. The relatively lower differences in TSH and T3 suggest that autoimmune activity precedes overt hormonal dysfunction, highlighting the importance of early detection of thyroid autoimmunity in affected patients.

The markedly elevated Anti-TPO levels observed in the post-COVID19-TE group suggest that autoimmune thyroiditis plays a central role. COVID-19 has been shown to trigger autoantibody production through mechanisms such as: molecular mimicry, bystander immune activation, epitope spreading [17]. This autoimmune activation may directly or indirectly affect hair follicle cycling [18].

Thyroid hormones are critical regulators of hair follicle proliferation and cycling. Even subtle thyroid dysfunction, particularly in the context of autoimmunity, may impair follicular activity and contribute to diffuse hair shedding [19,20]. As it was clear upon our results that we had combination of elevated TSH which could be compensatory, elevated T4, and strong Anti-TPO positivity. These finding are suggesting subclinical or evolving thyroid dysfunction, rather than overt disease.

Our findings are consistent with previous reports indicating association with systemic inflammation and immune dysregulation with thyroid autoimmunity [21]. However, most prior studies have focused primarily on stress-related mechanisms, whereas our study provides novel evidence linking thyroid autoimmunity to post-COVID hair loss.

CONCLUSION

Post-COVID-19 telogen effluvium in females is strongly associated with thyroid autoimmunity, particularly elevated Anti-TPO levels. These findings suggest that hair loss following COVID-19 infection is not solely a stress-induced phenomenon but may represent an immune-mediated endocrine disorder. Routine evaluation of thyroid autoimmunity should be considered in affected patients to facilitate early diagnosis and targeted management.

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