Table II. Comparison between cutaneous and extracutaneous PTL group

Category	All PTL, n = 115	Cutaneous PTL, n = 11	Extracutaneous PTL,* n = 104	*P* value
Male sex, n (%)	77 (67)	7 (64)	70 (67)	.806
Mean age at diagnosis of PTL, years	45.92	45.45	50.81	.370
Mean interval between first transplant and PTL, months	66.8	66.8	56.3	.044
Early onset (<12 months), n (%)	64 (56)	3 (27)	60 (58)	.054
Late onset (≥12 months), n (%)	51 (44)	8 (73)	44 (42)	
Type of transplantation, n (%)				
Liver	46 (40)	2 (18)	44 (42)	
Kidney	33 (29)	4 (36)	29 (28)	
Bone marrow	25 (22)	4 (36)	21 (20)	
Heart	9 (7.8)	1 (9)	8 (7.7)	
Pancreas	2 (1.7)		2 (1.9)	
Epstein-Barr virus positivity, n (%)	79 (69)	6 (55)	73 (70)	.287
Subtypes of lymphoma, n (%)†				
B-cell lineage lymphoma	90 (78)	3 (27)	87 (84)	<.001
Diffuse, large B-cell lymphoma	70 (61)	2 (18)	68 (65)	
Marginal zone B-cell lymphoma	11 (9.5)	1 (9)	10 (9.6)	
Hodgkin lymphoma	5 (4.3)	0	5 (4.8)	
Burkitt lymphoma	4 (3.5)	0	4 (3.8)	
T-cell lineage lymphoma	25 (22)	8 (73)	17 (16)	<.001
Peripheral T-cell lymphoma, not otherwise specified	16 (14)	3 (27)	13 (12.5)	
Anaplastic large cell lymphoma	4 (3.5)	2 (18)	2 (1.9)	
Natural killer/T-cell lymphoma	3 (2.6)	1 (9)	2 (1.9)	
Lymphomatoid papulosis	2 (1.7)	2 (18)	0	
Advanced (III or IV) Ann Arbor stage, n (%)	37 (32)	4 (36)	33 (32)	.744
Elevated serum lactate dehydrogenase, n (%)	51/89 (57)	3/7 (43)	48/82 (59)	.341
Outcome				
Median follow-up, months	79	81	79	
Alive, n (%)	66 (57)	6 (55)	60 (58)	
5-year survival rate, %	58	64	58	
Median overall survival, months	71	71	65	.586

PTL, Posttransplantation lymphoma.
*These included cases of PTL not involving the skin during follow-up. Secondary cutaneous PTLs that had a disseminated lesion on the skin during follow-up were excluded.
†This series did not contain any cases of mycosis fungoides or folliculotropic mycosis fungoides, which is inconsistent with the existing literature.

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The association among thyroid dysfunction, thyroid autoimmunity, and clinical features of alopecia areata: A retrospective study

To the Editor: Alopecia areata (AA) is a common form of nonscarring hair loss that is classified as an autoimmune disorder due to its association with other autoimmune diseases, such as thyroid autoimmunity.[1] In this study, we investigated the frequency of thyroid dysfunction and thyroid autoimmunity in AA patients and also examined the associations among thyroid dysfunction, thyroid autoimmunity, and 8 prognostic clinical features of AA.

A retrospective chart review led to the identification of 1408 Korean patients with AA diagnoses at KyungHee University Hospital in Gangdong, South Korea, during June 2006-March 2014. The severity of hair loss was assessed at the first visit in accordance with the Severity of Alopecia Tool,[2] and thyroid autoantibodies and thyroid hormones were measured. We defined thyroid dysfunction as any thyroid hormone level >10% or <10% of reference ranges (thyroxine [70-180 nmol/L], triiodothyronine

Table I. Associations between thyroid dysfunction and 8 prognostic clinical features of AA

Characteristic	Total, N = 1408	Thyroid function: Normal, N = 1184	Thyroid function: Abnormal, N = 224	*P* value	Univariate OR (95% CI)	Multivariate[†] OR (95% CI)
Age, y, mean ± SD		37.4 ± 14.0	39.7 ± 15.8	.043*		
Sex				<.001*		
Male, n (%)	592	522 (88.18)	70 (11.82)			
Female, n (%)	816	662 (81.13)	154 (18.87)			
Onset				<.001*	2.21* (1.65-2.97)	7.89* (4.94-12.62)
>30 years, n (%)	997	872 (87.46)	125 (12.54)			
≤30 years, n (%)	411	312 (75.91)	99 (24.09)			
Personal history of AA				.001*	1.66* (1.24-2.22)	1.71* (1.27-2.29)
No, n (%)	898	778 (86.73)	120 (13.27)			
Yes, n (%)	510	406 (79.46)	104 (20.54)			
Duration				.338	0.85 (0.61-1.19)	0.84 (0.60-1.17)
<2 years, n (%)	1045	873 (83.54)	172 (16.46)			
≥2 years, n (%)	363	311 (85.67)	52 (14.33)			
Family history of AA				.6	0.90 (0.59-1.35)	0.90 (0.60-1.36)
No, n (%)	1197	1004 (83.87)	193 (16.13)			
Yes, n (%)	211	180 (85.31)	31 (14.69)			
AD history				.87	1.06 (0.53-2.12)	1.25 (0.61-2.57)
No, n (%)	1348	1134 (84.12)	214 (15.88)			
Yes, n (%)	60	50 (83.33)	10 (16.67)			
Nail involvement				.068	1.81 (0.95-3.45)	2.01* (1.04-3.88)
No, n (%)	1356	1145 (84.43)	211 (15.56)			
Yes, n (%)	52	39 (75)	13 (25)			
Other autoimmune diseases				<.001*	3.46* (2.30-5.22)	3.23* (2.13-4.90)
No, n (%)	1292	1110 (85.91)	182 (14.09)			
Yes, n (%)	116	74 (63.79)	42 (36.21)			
Severity[‡]				<.001*	19.08* (9.51-38.31)	21.62* (10.60-44.10)
<S4, n (%)	1363	1173 (86.06)	190 (13.94)			
≥S4, n (%)	45	11 (24.44)	34 (75.56)			

AA, Alopecia areata; *AD*, atopic dermatitis; *CI*, confidence Interval; *OR*, odds ratio; *SD*, standard deviation.
*Statistically significant.
[†]Adjusted for sex and age.
[‡]Severity of Alopecia Tool scores: S0 (0% hair loss), S1 (<25% hair loss), S2 (25%-49% hair loss), S3 (50%-74% hair loss), S4a (75%-95% hair loss), S4b (96%-99% hair loss), and S5 (100% hair loss).

[1.3-3.3 nmol/L], and thyroid-stimulating hormone [0.3−4.2 mlU/L]). The presence of thyroid autoantibodies indicated thyroid autoimmunity. We investigated the prognostic clinical features of AA, including age of onset, disease duration, extent of alopecia, personal or family history of AA, history of atopy, nail involvement, and other autoimmune diseases. The retrospective data were analyzed using the Statistical Package for the Social Sciences version 20.0 (SPSS Inc, Chicago, IL). Continuous data are presented as mean ± standard deviation. A comparison of the demographic and clinical variables was performed using a χ^2 test. $P < .05$ was considered statistically significant.

The sample included 592 (42.04%) male and 816 (57.96%) female patients; 224 patients (15.90%) had thyroid dysfunction, and thyroid autoimmunity was identified in 260 (18.46%). Concomitant diagnoses included clinical thyroid disorder (n = 52), type-1 diabetes (n = 20), rheumatoid arthritis (n = 19), celiac disease (n = 13), and vitiligo (n = 12). The severe AA and mild AA groups showed a notable difference in thyroid dysfunction (75.56% vs 13.94%, respectively) and thyroid autoimmunity (40.82% vs 18.22%, respectively) diagnoses. Moderate differences in concomitant autoimmune disease and nail involvement were found between these groups. In our multivariate logistic regression, we controlled for age and sex because of the higher incidence of thyroid dysfunction and thyroid autoimmunity among older women. There was a significant relationship between thyroid dysfunction and earlier disease onset, personal history of AA, concomitant autoimmune disorder, and disease severity (Table I). A significant relationship also existed between thyroid autoimmunity and a longer

Table II. Association between thyroid autoimmunity and 8 prognostic clinical features of AA

Characteristic	Thyroid antibody					
	Total, N = 1366	Normal, N = 1106	Abnormal, N = 260	*P* value	Univariate OR (95% CI)	Multivariate† OR (95% CI)
Age, y, mean ± SD		36.6 ± 14.3	40.2 ± 12.9	<.001*		
Sex				<.001*		
Male, n (%)	568	510 (89.79)	58 (10.21)			
Female, n (%)	798	596 (74.69)	202 (25.31)			
Onset				.365	0.86 (0.62-1.19)	1.33 (0.85-2.07)
>30 years, n (%)	952	764 (80.25)	188 (19.75)			
≤30 years, n (%)	414	342 (82.61)	72 (17.39)			
Personal history of AA				.178	1.23 (0.91-1.66)	1.30 (0.96-1.76)
No, n (%)	865	711 (82.20)	154 (17.80)			
Yes, n (%)	501	395 (78.84)	106 (21.16)			
Duration				.013*	1.49* (1.09-2.04)	1.47* (1.07-2.03)
<2 years, n (%)	1000	827 (82.7)	173 (17.3)			
≥2 years, n (%)	366	279 (73.23)	87 (23.77)			
Family history of AA				.913	1.02 (0.68-1.54)	1.03 (0.68-1.55)
No, n (%)	1161	941 (81.05)	220 (18.95)			
Yes, n (%)	205	165 (80.49)	40 (19.51)			
AD history				.907	1.04 (0.52-2.09)	1.34 (0.65-2.77)
No, n (%)	1303	1056 (81.04)	247 (18.96)			
Yes, n (%)	63	50 (79.37)	13 (20.63)			
Nail involvement				.074	1.79 (0.94-3.42)	2.15* (1.09-4.23)
No, n (%)	1311	1067 (81.39)	244 (18.61)			
Yes, n (%)	55	39 (70.91)	16 (29.09)			
Other autoimmune diseases				.005*	1.93* (1.21-3.07)	1.67* (1.04-2.70)
No, n (%)	1256	1029 (81.93)	227 (18.07)			
Yes, n (%)	110	77 (70)	33 (30)			
Severity‡				<.001*	3.07* (1.63-5.77)	3.29* (1.71-6.34)
<S4, n (%)	1317	1077 (81.78)	240 (18.22)			
≥S4, n (%)	49	29 (59.18)	20 (40.82)			

Reference ranges for thyroid antibody tests were defined as a thyroglobulin antibody of 0-30 IU/L, thyroid-stimulating hormone receptor antibody of 0-1.0 IU/L, and antithyroid microsome antibody of 0-100 IU/L.

AA, Alopecia areata; *AD*, atopic dermatitis; *CI*, confidence Interval; *OR*, odds ratio; *SD*, standard deviation.

*Statistically significant.

†Adjusted for sex and age.

‡Severity of Alopecia Tool scores: S0 (0% hair loss), S1 (<25% hair loss), S2 (25%-49% hair loss), S3 (50%-74% hair loss), S4a (75%-95% hair loss), S4b (96%-99% hair loss), and S5 (100% hair loss).

disease duration, concomitant autoimmune disorder, and disease severity (Table II). However, univariate logistic regression found no correlation between nail involvement and thyroid dysfunction or thyroid autoimmunity.

Similar to prior studies,[3,4] we observed an increased incidence of thyroid dysfunction and thyroid autoimmunity in AA patients, particularly those with severe AA. Kurtev et al[5] reported that activated T lymphocytes ($CD3^+HLA\text{-}DR$ [human leukocyte antigen, antigen-D related]$^+$) increased in the peripheral blood and infiltrated the epithelial cells of hair follicles, epidermal keratinocytes, and thyrocytes of AA patients, and activated T lymphocytes might play a role in the pathogenesis of AA and autoimmune thyroid dysfunction. Our study was limited because we were not able to identify whether thyroid dysfunction preceded the diagnosis of AA. Also, subclinical hyperthyroidism and subclinical hypothyroidism were classified as thyroid dysfunction, although we could not confirm an association between these conditions and AA.

In conclusion, thyroid dysfunction and thyroid autoimmunity are poor prognostic factors for patients with AA. Thus, thyroid function and thyroid autoantibody testing might be useful for evaluating the prognosis of affected individuals.

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Host characteristics and dynamics of *Staphylococcus aureus* colonization in patients with moderate-to-severe psoriasis before and after treatment: A prospective cohort study

To the Editor: Although psoriatic lesions are prone to staphylococcal colonization[1] and evidence has suggested that nasal decolonization could reduce recurrent infections,[2,3] the current evidence is insufficient to inform clinical practice on bacterial colonization in patients with psoriasis vulgaris.

In this study, we prospectively followed 50 patients with psoriasis from 2015 to 2017 to quantify changes in staphylococcal colonization after a course of treatment with biologics (n = 42) or nonbiologics (n = 8) for 16 weeks. At enrolment, all eligible patients were evaluated for initiation of biologics such as interleukin monoclonal antibodies (ustekinumab or secukinumab) or tumor necrosis factor inhibitors (adalimumab, golimumab, or etanercept) on the basis of disease severity (Psoriasis Area Severity Index score, >10) and a lack of response to or intolerance of traditional systemic therapeutics, including methotrexate, acitretin, cyclosporine, leflunomide, or a combination of these agents.

After written informed consent had been obtained, enrolled patients were interviewed for sociodemographic information, medical history, and clinical outcomes. The primary outcome was the culture-based staphylococcal colonization status of selected lesion sites; we also swabbed the anterior nares and nailfolds of the fingers to evaluate cross-site concordance. We reported prevalence estimates and Wilson 95% confidence intervals (CIs) as suggested[4] and performed mixed-effects regression analysis and differential expression analysis to identify host factors associated with colonization dynamics.

The study included 50 participants with an average age of 41 years (interquartile range, 34-46), with the majority being men (86%) and having had psoriasis for 20 years or longer; 7 cases (14%) were newly diagnosed at enrolment. A total of 10 users of biologics were followed for additional 8 months and appeared representative of the others.

At enrolment, 12 patients (24%) carried *Staphylococcus aureus* in their nares and 16 (32%) harbored *S. aureus* on at least 1 lesion (Fig 1, *A*) (average, 12.1% per lesion). After 16 weeks of treatment, the mean rate of lesion colonization decreased to 5.0% (adjusted odds ratio [aOR], 0.35 [$P = .024$]) but colonization of the nares or nailfolds did not decrease (both $P > .05$) (Fig 1, *A*), although site-specific variations existed (Fig 1, *B*). Colonization by methicillin-resistant *S. aureus* followed a similar pattern (data not shown).

Nailfold colonization before (aOR, 4.91 [$P = .017$]) and during (aOR, 6.20 [$P = .002$]) treatment were associated with lesion colonization, as were active smoking (aOR, 3.38 [$P = .019$]) and concurrent nasal colonization (aOR, 3.68 [$P = .002$]). Patient-reported quality of life (as quantified by the Dermatology Life Quality Index [$P = .008$]) and regular use of antiseptic-containing facial cleansers ($P < .001$) correlated with loss of colonization; first-time receipt of systemic therapeutics ($P = .008$) increased the risk of lesion recolonization. Concordance analysis showed temporal correlation of colonization of the nailfolds and nares (aOR, 26.8 [$P = .003$]) as well as temporal correlation of colonization of the nares and hairline (aOR, 4.69 [$P = .044$]); hairline colonization was highly concordant with colonization of other skin lesions (all $P \leq .022$).

Despite a limited power to compare the decolonization effects of systemic agents for treatment of psoriasis, commensal *S. aureus* on psoriatic lesions was reduced in patients receiving systemic