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Original Article

Oral lichen planus possibly associated with Hashimoto's thyroiditis: A case–control study in a northwest Chinese population

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KEYWORDS
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Abstract *Background/purpose:* Some studies have previously demonstrated possible association between Hashimoto's thyroiditis (HT) and oral lichen planus (OLP), however, data of immunity and inflammation in these patients were not well investigated. This study aimed to propose some novel findings based on a case–control study of 185 OLP patients and 185 age- and gender-matched non-OLP volunteers in northwest China.

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Female patients;
Thyroid autoantibodies;
TPO-Ab

Materials and methods: This study was designed and implemented in a tertiary teaching hospital between January 2021 and December 2024. Comprehensive evaluation was performed including thyroid function tests and seroimmunological parameters.
Results: The incidence of HT among OLP patients reached 31.9 %, demonstrating a statistically significant elevation compared to the control group's prevalence of 9.7 % ($P < 0.001$). Regarding autoimmune markers, thyroid peroxidase antibody (TPO-Ab) positivity was observed in 18.9 % of OLP patients, significantly exceeding the 4.3 % rate in controls ($P = 0.002$). Gender-based analysis revealed that female patients had a markedly higher TPO-Ab positivity rate (21.99 %) compared to males (9.09 %) ($P = 0.012$). Clinical subtype analysis demonstrated that patients with erosive OLP lesions showed a significantly higher TPO-Ab positive rate (25.74 %) than those with reticular lesions (11.63 %) or atrophic lesions (9.76 %) ($P = 0.028$). Logistic regression modeling identified two significant risk factors: female OLP patients had an 8.935-fold increased risk of TPO-Ab positivity compared to males ($P = 0.036$), while patients with erosive lesions showed a 3.199-fold higher risk than non-erosive cases ($P = 0.038$).
Conclusion: Our findings demonstrate a strong correlation between OLP, especially its erosive form, and thyroid autoimmunity, with female sex representing an independent and clinically relevant predisposing factor.

Introduction

Lichen planus is a common, chronic inflammatory disease that can affect skin and mucous membranes, including the oral mucosa.[1] The prevalence of oral lichen planus (OLP) worldwide is estimated to be between 0.5 % and 2 %, peaking during the fifth to sixth decades of life, with a female-to-male ratio of more than 2:1.[2,3] The incidence of OLP varies within the available literature, with reported rates of up to 2.2 %, around 15 % of which concurrently develop cutaneous lesions.[4] Although there is consensus that OLP's pathogenesis involves epithelium-directed, T-cell mediated inflammation in response to unknown antigens, the specific etiology of OLP remains unclear.[1,4,5] Many risk factors including mental health, immune and endocrine functions, infections (notably hepatitis C), and systemic diseases (such as dyslipidemia, hypertension, diabetes, chronic liver disease, and thyroid disorders) are critical for the development of OLP.[6–8]

Hashimoto's thyroiditis (HT), an organ-specific autoimmune disorder mediated by T lymphocytes,[9] has a prevalence of around 2 % in the general population with an increasing incidence trend, demonstrating significant female predominance.[10] The exact etiopathogenesis of HT remains incompletely understood, involving complex interactions between genetic predisposition, epigenetic modifications, and environmental triggers.[11] Emerging evidence suggests a potential association between OLP and HT.[12–14] Clinical investigations have revealed that individuals diagnosed with OLP exhibit a substantially higher incidence of HT when compared to healthy control populations.[15,16] This epidemiological observation implies a possible pathophysiological link between these two autoimmune conditions.[12,15,16] However, no definite conclusion can be drawn from the findings of previous studies regarding the relationship between HT and OLP.

So far, the findings of most studies on the reciprocal association between OLP and HT based on laboratory analyses is inadequate. Moreover, existing research investigating the association between OLP and HT has been constrained by several methodological challenges. Key limitations include the inconsistent application of diagnostic criteria for both conditions across different studies. This variability in diagnostic approaches has contributed to inconclusive findings regarding the potential relationship between OLP and HT in the current scientific literature. The lack of standardized diagnostic protocols has significantly hindered the ability to draw definitive conclusions from previous investigations. Furthermore, comprehensive investigations examining the simultaneous involvement of pan-immune inflammation value (PIV) and systemic immune-inflammation index (SII) in this context remain scarce. Given that both PIV and SII serve as crucial biomarkers for assessing immune-mediated inflammatory processes, and considering the established immunological associations of both HT and OLP, these parameters may potentially contribute to elucidating the pathophysiological link between these two conditions. The present case–control study aims to explore the association between HT and OLP within the Chinese demographic, while concurrently evaluating the potential correlation between HT-specific autoantibody profiles and the clinical presentation of OLP.

Materials and methods

Study design and ethical statement

To achieve the study objectives, the researchers conducted a retrospective, observational, single blind and case–control study following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.[17] The research cohort comprised patients receiving clinical care at the investigators' institution from January 2021 through December 2024. This investigation received ethical clearance from the institutional review board (IRB approval

number: K202304-11) and was conducted in strict compliance with both the ethical principles outlined in the Declaration of Helsinki and China's national laboratory guidelines. Complete study datasets have been incorporated into this manuscript, with all participants providing written informed consent prior to enrollment.

Participant selection

The case group in this study enrolled 185 consecutive patients with clinically confirmed OLP. The selection process adhered to rigorous inclusion and exclusion protocols as following. Inclusion criteria comprised: *i*) histopathologically verified OLP meeting the modified World Health Organization diagnostic criteria;[18] *ii*) confirmed HT diagnosis based on elevated thyroid peroxidase antibodies (TPO-Ab) and thyroglobulin antibodies (Tg-Ab) levels coupled with characteristic hypoechogenicity on thyroid ultrasound, consistent with established clinical manifestations;[19] *iii*) participants aged above 18 years; *iv*) documented thyroid autoantibody (TPO-Ab, Tg-Ab) and thyroid function [free triiodothyronine (FT3), free thyroxine (FT4), and thyroid-stimulating hormone (TSH)] and hemogram test results.

Exclusion criteria included: *i*) previous or concurrent diagnosis of alternative oral mucosal disorders including lichenoid lesions, leukoplakia, erythematous lesions, submucous fibrosis, discoid lupus erythematosus, or recurrent aphthous ulcers; *ii*) history of systemic or autoimmune comorbidities and associated immunopharmacotherapy; *iii*) pregnancy or breastfeeding status at time of study recruitment.

The control group comprised 185 age- and gender-matched non-OLP volunteers from our institutional health screening program during the same period. All controls underwent thorough oral examinations confirming absence of mucosal lesions prior to enrollment.

Data collection

All participants in both case and control groups received comprehensive thyroid evaluations, including B-mode ultrasonography of the thyroid gland. Demographic characteristics and clinical data pertaining to various thyroid disorders (hypothyroidism, hyperthyroidism, and thyroid nodules) were systematically documented.

Serological analysis was performed for five key thyroid function markers: Tg-Ab, TPO-Ab, TSH, FT3, and FT4. These quantitative measurements were obtained using chemiluminescent immunoassay techniques, performed by certified laboratory technicians in our hospital's Clinical Laboratory Department. The established reference ranges for these parameters were as follows: TPO-Ab (0—9 IU/mL), Tg-Ab (0—115 IU/mL), TSH (0.34—5.6 μIU/mL), FT3 (2.5—3.9 pg/mL), and FT4 (0.58—1.64 ng/dL).

The PIV and SII - extracted from blood routine examination - were calculated using the following formula: PIV = neutrophil counts ($\times 10^9$/L) $\times$ platelet counts ($\times 10^9$/L) $\times$ monocyte counts ($\times 10^9$/L) $\div$ lymphocyte counts ($\times 10^9$/L); SII = neutrophil counts ($\times 10^9$/L) $\times$ platelet counts ($\times 10^9$/L) $\div$ lymphocyte counts ($\times 10^9$/L).[20] The medical materials of total population was collected by retrieving for the registered cases in Hospital Information and Management System (HIMS).

Statistical analyses

For categorical data analysis, chi-square tests or Fisher's exact tests were employed, while continuous variables were assessed using Student's t-tests. When normality assumptions were violated for quantitative measures, non-parametric alternatives were implemented. Multivariate logistic regression models were constructed to examine variable associations, expressed as odds ratios (ORs) with corresponding 95 % confidence intervals (CIs). Variables demonstrating significance in univariate analyses were subsequently incorporated into the multivariate models. All statistical computations were executed using R packages, version 4.2.1 (R Foundation for Statistical Computing, Vienna, Austria), with a predetermined significance threshold of $\alpha = 0.05$ for two-tailed tests. Statistical significance was set at P values < 0.05.

Results

General information of study population

The present investigation enrolled a cohort of 370 individuals, comprising 185 cases diagnosed with OLP and an equal number of age- and sex-matched non-OLP controls. Demographic analysis revealed comparable age distributions between groups, with OLP patients averaging 46.52 $\pm$ 11.67 years versus 47.66 $\pm$ 12.98 years in controls ($P = 0.385$). Gender stratification demonstrated similar proportions, with 23.8 % males (n = 44) and 76.2 % females (n = 141) in the OLP cohort, compared to 24.3 % males (n = 45) and 75.7 % females (n = 140) among controls ($P = 0.790$). These findings confirm balanced baseline characteristics between groups (all P-values >0.05), ensuring appropriate comparability for subsequent analyses, as comprehensively presented in Table 1.

Comparison of the prevalence of HT and thyroid autoantibody profiles between OLP cohorts and matched controls

The study revealed a markedly higher prevalence of HT among OLP patients (31.9 %, 59/185) compared to the controls (9.7 %, 18/185), with this difference reaching statistical significance ($P < 0.001$). Comparative analysis of thyroid-specific autoantibodies yielded the following results: *i*) Tg-Ab was detected in 9.2 % (17/185) of OLP cases versus 3.2 % (6/185) of non-OLP subjects, with this difference not achieving statistical significance ($P = 0.166$). *ii*) TPO-Ab showed significantly greater positivity in the OLP group (18.9 %, 35/185) relative to controls (4.3 %, 8/185) ($P = 0.002$). *iii*) Concurrent positivity for both Tg-Ab and TPO-Ab was observed in 7.0 % (13/125) of OLP patients and 3.2 % (6/185) of controls, without reaching statistical significance ($P = 0.123$) (Table 1).

Table 1 Demographic data and HT prevalence and patients characteristics.

Variate	OLP group (n = 185)	non-OLP control (n = 185)	t/χ^2 value	*P* value
Age, yr			0.870	0.385
Range	20–72	21–75		
Mean ± SD	46.52 ± 11.67	47.66 ± 12.98		
Gender, [n (%)]			0.081	0.790
Male	44 (23.8)	45 (24.3)		
Female	141 (76.2)	140 (75.7)		
Smoking history			0.659	0.417
Yes	34 (18.4)	32 (17.3)		
No	151 (81.6)	153 (82.7)		
HT, [n (%)]			18.516	<0.001
With	59 (31.9)	18 (9.7)		
Without	126 (68.1)	167 (90.3)		
Tg-Ab, [n (%)]			2.191	0.166
Negative	168 (90.8)	179 (96.8)		
Positive	17 (9.2)	6 (3.2)		
TPO-Ab, [n (%)]			10.989	0.002
Negative	150 (80.1)	177 (95.7)		
Positive	35 (18.9)	8 (4.3)		
Tg-Ab + TPO-Ab double-positive, [n (%)]	13 (7.0)	6 (3.2)	2.377	0.123
Primary site[a]				
Vermilion border	1 (0.5)			
Labial commissure	6 (3.2)			
Oral vestibule	3 (1.6)			
Cheek mucosa	102 (55.1)			
Tongue	40 (21.6)			
Floor of mouth	5 (2.7)			
Alveolar crest	8 (4.3)			
Retromolar trigone	12 (6.5)			
Palate	6 (3.2)			
Tuber maxillae	2 (1.1)			
Tonsil	0			

HT Hashimoto's thyroiditis, *OLP* oral lichen planus, *SD* standard deviation, *Tg-Ab* thyroglobulin antibodies, *TPO-Ab* thyroid peroxidase antibodies, *yr* year.
[a] The presented dataset is exclusively employed for demonstrating proportional distributions in case cohort.

Role of anti-thyroid antibodies in patients with OLP, stratified by age, gender, and OLP clinical manifestations

In the OLP cohort, the positive rate for TPO-Ab was significantly elevated in female patients relative to their male counterparts ($P = 0.012$). However, no significant gender-based differences were observed in the positivity for Tg-Ab or in the dual positivity for Tg-Ab and TPO-Ab (all *P*-values >0.05). Furthermore, patients with erosive OLP exhibited a higher TPO-Ab positive rate than those with non-erosive OLP, and this difference reached statistical significance ($P = 0.028$). No statistically significant variations were detected in the positive rates of Tg-Ab, TPO-Ab, or the dual positivity of Tg-Ab and TPO-Ab across different age subgroups among OLP patients (all *P*-values >0.05). Similarly, when comparing OLP patients with a disease duration exceeding six months to those with a duration of less than six months, no significant differences were found in the positive rates of Tg-Ab, TPO-Ab, or their dual positivity (all *P*-values >0.05) (Table 2).

Table 2 HT autoantibody in different ages, sexes, lesion types, and disease duration of patients with OLP.[a]

Variate	Total number (n)	Tg-Ab positive [n (%)]	TPO-Ab positive [n (%)]	Tg-Ab + TPO-Ab double-positive [n (%)]
Gender				
Female	141	14 (9.93)	31 (21.99)	11 (7.80)
Male	44	3 (6.82)	4 (9.09)	2 (4.55)
χ^2 value		1.118	5.210	0.825
P value		0.292	0.012	0.458
Age (yr)				
Youth (20–44)	77	6 (7.79)	14 (18.18)	5 (6.49)
Middle-aged (45–59)	82	7 (8.54)	16 (19.51)	5 (6.10)
Elderly (60–72)	26	4 (15.38)	5 (19.23)	3 (11.54)
χ^2 value		0.785	0.473	5.072
P value		0.675	0.789	0.079
Histopathology				
Reticular type	43	3 (6.98)	5 (11.63)	2 (4.65)
Atrophic type	41	4 (9.76)	4 (9.76)	2 (4.88)
Erosive type	101	10 (9.90)	26 (25.74)	9 (8.91)
χ^2 value		0.062	4.831	0.003
P value		0.806	0.028	0.967
OLP scoring[b]				
1	80	0	1 (1.25)	0
2	25	2 (8.00)	2 (8.00)	0
3	15	2 (13.33)	4 (26.67)	1 (6.67)
4	49	6 (12.24)	12 (24.49)	6 (12.24)
5	16	7 (43.75)	16 (100.00)	6 (37.50)
χ^2 value		8.506	10.989	12.029
P value		0.196	0.055	<0.001
Course of disease				
<6 months	78	5 (6.41)	12 (15.38)	4 (5.13)
≥6 months	107	12 (11.21)	23 (21.50)	9 (8.41)
χ^2 value		0.271	0.192	0.084
P value		0.603	0.662	0.772

HT Hashimoto's thyroiditis, *OLP* oral lichen planus, *Tg-Ab* thyroglobulin antibodies, *TPO-Ab* thyroid peroxidase antibodies.
[a] The sample size for survey of clinicopathological characteristics was 185 patients with OLP, as shown in Table 1.
[b] OLP scoring: 5 = white striae with erosive areas >1 cm^2; 4 = white striae with erosive areas <1 cm^2; 3 = white striae with atrophic areas >1 cm^2; 2 = white striae with atrophic areas <1 cm^2; 1 = mild white striae only, no atrophic areas; and 0 = no lesion, normal mucosa.

Thyroid function-related indicators and seroimmunological parameters in OLP patients with or without HT

Regarding the differences between subgroups of HT comorbidity or not, OLP patients coexist alongside HT had significantly higher levels of TPO-Ab (233.5 IU/mL, $P = 0.012$), TSH (2.85 μIU/mL, $P = 0.048$), but lower concentrations of FT3 (2.4 pg/mL, $P = 0.039$) (Table 3). The seroimmunological parameters on admission such as PIV and SII had no statistical difference (all $P > 0.05$, Table 3). A summary of thyroid function features in patients with OLP is presented in Table 3.

Logistic regression analysis for risk factors related to the positive TPO-Ab in patients with OLP

Multivariate logistic regression analysis revealed significant gender differences in TPO-Ab positivity among OLP patients, with female patients exhibiting an 8.935-fold higher risk compared to males (OR = 8.935, 95 % CI: 1.134—17.388, $P = 0.036$). Additionally, patients presenting with erosive OLP lesions demonstrated a 3.199 times greater likelihood of TPO-Ab positivity than those with non-erosive lesions (OR = 3.199, 95 % CI: 1.064—9.618, $P = 0.038$). No statistically significant associations were observed between TPO-Ab positivity and age stratification (young vs middle-aged/elderly: all $P > 0.05$) or disease duration (≥6 months vs < 6 months: $P = 0.887$) (Table 4).

Discussion

This study revealed a notably higher incidence of HT among OLP patients (31.9 %) compared to the control group (9.7 %). Furthermore, female OLP patients exhibited a substantially greater prevalence of HT (40.7 %) than their male counterparts (10.2 %), aligning with prior research findings.[15,21,22] This observation underscores the heightened vulnerability of women to autoimmune disorders. The observed gender disparity may stem from various contributing elements, such as variations in sex hormone concentrations, X chromosome-associated genetic predisposition, and inherent immunological response differences between sexes.[23] Notably, estrogen and other sex hormones play a pivotal role in modulating immune cell functionality, with T lymphocytes serving as primary effector cells.[24,25] Fluctuations in estrogen concentrations can potentially impair T cell regulatory mechanisms. The fluctuation in estrogen concentrations serves as both an initiator and promoter, inducing an imbalance in the Th1/Th2 (T helper cell) ratio with a predominant shift toward Th1

Table 3 Statistical differences regarding thyroid function and immune-inflammation level in OLP patients with or without HT.[c]

Variate	OLP with HT (n = 59)	OLP without HT (n = 126)	Statistics	*P* value
Gender, [n (%)]			15.772	<0.001
Male	6 (10.2)	38 (30.2)		
Female	24 (40.7)	117 (92.9)		
BMI (kg/m^2), mean ± SD	29.0 ± 6.9	28.2 ± 5.5	8.960	0.044
Hypothyroidism, [n (%)]			9.379	0.041
Yes	12 (20.3)	3 (2.4)		
No	47 (79.7)	123 (97.6)		
Hyperthyroidism, [n (%)]			2.820	0.156
Yes	5 (8.5)	2 (1.6)		
No	54 (91.5)	124 (98.4)		
Thyroid volume (mm^3), [median (Q_L, Q_U)]	10.99 (8.33, 13.71)	9.09 (7.19, 11.52)	1.844	0.941
Tg-Ab (IU/mL)[a], [median (Q_L, Q_U)]	15.9 (2.6, 240.8)	12.6 (2.8, 56.1)	3.375	0.350
TPO-Ab (IU/mL)[b], [median (Q_L, Q_U)]	233.5 (21.4, 772.3)	38.1 (1.7, 199.0)	7.921	0.012
FT3 (pg/mL), mean ± SD	2.4 ± 0.5	2.8 ± 0.7	6.171	0.039
FT4 (ng/dL), mean ± SD	1.1 ± 0.3	1.2 ± 0.6	0.551	0.595
FT3/FT4 ratio, mean ± SD	2.1 ± 0.7	2.3 ± 0.1	6.905	0.036
TSH (μIU/mL), [median (Q_L, Q_U)]	2.85 (1.66, 3.80)	2.69 (1.71, 3.53)	5.859	0.048
PIV on admission, mean ± SD	239.4 ± 11.8	215.9 ± 9.7	0.75	0.766
SII on admission, mean ± SD	410.6 ± 8.3	399.0 ± 8.8	0.97	0.628

FT3 free triiodothyronine, *FT4* free thyroxine, *HT* Hashimoto's thyroiditis, *OLP* oral lichen planus, *PIV* pan-immune inflammation value, Q_L lower quartile, Q_U upper quartile, *SD* standard deviation, *SII* systemic immune-inflammation index, *Tg-Ab* thyroglobulin antibodies, *TPO-Ab* thyroid peroxidase antibodies, *TSH* thyroid-stimulating hormone.

[a] The Tg-Ab values were detected using serum from Tg-Ab positive individuals, as exhibited in Table 2.

[b] The TPO-Ab values were detected using serum from TPO-Ab positive individuals, as exhibited in Table 2.

[c] The sample size for survey of clinicopathological characteristics was 185 patients with OLP, as shown in Table 1.

Table 4 Comparative and logistic regression analyses of TPO-Ab positive rate in patients with OLP.

Variate	β	SE	Wald	OR	95 % CI	*P* value
Age[a]						
Middle-aged (*vs* youth)	0.003	0.036	0.031	1.025	0.958–1.055	0.966
Elderly (*vs* youth)	0.005	0.016	0.027	0.985	0.956–1.051	0.865
Gender						
Female (*vs* male)	2.199	1.055	4.325	8.935	1.134–17.388	0.036
Histopathology						
Erosive (*vs* non-erosive[b])	1.176	0.562	4.286	3.199	1.064–9.618	0.038
Course of disease						
≥6 months (*vs* < 6 months)	0.087	0.520	0.022	1.079	0.389–2.990	0.887

CI confidence interval, *OLP* oral lichen planus, *OR* odds ratio, *SE* standard error, *TPO-Ab* thyroid peroxidase antibodies.
[a] Age: youth group: 20–44 years old; middle-aged group: 45–59 years old; and elderly group: 60–72 years old.
[b] Non-erosive: atrophic and reticular.

cells. This alteration facilitates Th1-mediated cellular immune responses,[26] consequently modulating immune cell functionality and autoantibody generation.[27] Such immunological perturbations may contribute to the higher susceptibility of females to autoimmune disorders, including HT and OLP.[16]

TPO-Ab is a prevalent antibody found in thyroid tissue, classified under the IgG immunoglobulin category. It is primarily associated with immune-mediated damage in thyroid tissue and can contribute to the tissue destruction observed in HT and atrophic autoimmune thyroiditis. The underlying pathological mechanisms involve complement activation, antibody-dependent cell-mediated cytotoxicity, and direct destruction of thyroid follicular cells by activated cytotoxic T lymphocytes.[15,28] In this investigation, the serum positivity rate for TPO-Ab was markedly elevated in the OLP case group compared to the control group. Both Tg-Ab and TPO-Ab, as autoantibodies targeting thyroid tissue, have been demonstrated to mediate the destruction of thyroid follicular cells in patients with HT. The present investigation revealed a markedly elevated positive rate of thyroid autoantibodies among OLP patients, indicating potential associations between their immune dysregulation and thyroid autoimmune responses. Consequently, we hypothesize that these antibodies might contribute to the pathogenesis of oral mucosal lesions in OLP through certain molecular pathways,[26,29] though the precise mechanisms warrant further elucidation. Notably, our data demonstrated that female OLP patients exhibited a significantly higher TPO-Ab positive rate compared to their male counterparts. Moreover, patients with erosive OLP displayed an increased TPO-Ab positive rate relative to those with non-erosive forms. These findings imply that the clinical severity of OLP may correlate with the intensity of thyroid autoimmune activity. Erosive lesions, characterized by more pronounced local inflammatory reactions or intricate immune dysregulation,[30] could potentially exacerbate autoimmune-mediated thyroid cell damage, thereby elevating TPO-Ab seropositivity.

The findings of logistic regression analysis demonstrated a significant association between elevated TPO-Ab levels and an increased risk among female patients with erosive OLP. These findings align with previous investigations.[16,28] However, contrasting evidence exists, with certain studies reporting no statistically significant alterations in thyroid autoantibody levels among OLP patients.[15,31] These discrepancies may potentially stem from variations in study design, sample sizes, disease evaluation methodologies, population heterogeneity,[32] as well as systemic and local contributing factors.[16,33] In HT, TPO-Ab may potentially induce antigen release through molecular mimicry or tissue damage mechanisms, thereby initiating cross-reactive immune responses targeting oral mucosal tissues.[31] Three principal mechanistic pathways have been proposed to explain this association: *i*) The potential coexistence or sequential development of HT and OLP, where thyroid dysfunction-induced hormonal alterations and autoantibody production may mediate keratinocyte modifications in oral mucosa, subsequently triggering OLP pathogenesis; *ii*) Shared immunological abnormalities between the two conditions, characterized by T-cell distribution irregularities and elevated proinflammatory cytokine levels in both systemic circulation and localized lesions of OLP patients, suggesting potential cross-talk between oral and thyroid immune microenvironments through local or systemic immune activation pathways; *iii*) Genetic predisposition, with emerging evidence implicating common susceptibility loci (including *HLA-DR*, *CTLA-4*, *PTPN22*, and *FOXP3* genes) in both disorders, which play crucial roles in T-cell regulation and maintenance of immunological tolerance.[13,34–36] Genetic variations in these genes could potentially elevate an individual's predisposition to autoimmune disorders. Nevertheless, comprehensive investigations regarding the co-occurrence of OLP and HT-associated genetic factors remain lacking. Future genomic association studies are warranted to elucidate potential shared genetic mechanisms between these two conditions.[31] Given the analogous pathogenic pathways of HT and OLP - both characterized by T cell-mediated localized immune responses - their interrelationship demands greater research focus, with cellular immunity representing a particularly promising avenue for investigation.[13,37]

In conclusion, our study demonstrates a significant association between OLP and HT in the Xinjiang population. The data reveal an elevated prevalence of HT among OLP patients, with particularly high rates observed in female subjects and those presenting with erosive OLP lesions. Notably, these patient subgroups also exhibited increased

positivity for thyroid autoantibodies. These findings underscore the clinical importance of implementing routine thyroid function tests and antibody screening for OLP patients, with special attention to high-risk groups. The case—control design of the current investigation precludes definitive conclusions regarding causality between OLP and HT. Future research should employ prospective cohort studies to more rigorously examine this association and elucidate potential shared immunological pathways. Furthermore, certain methodological limitations warrant consideration, including the relatively modest sample size and geographically restricted participant recruitment, which may introduce selection bias and constrain the generalizability of our results. Subsequent investigations would benefit from adopting large-scale, multicenter prospective designs to corroborate these preliminary findings.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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