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Original Article

Gastric parietal cell and thyroid autoantibodies in patients with oral lichen planus

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Abstract *Background/purpose:* Gastric parietal cell antibody (GPCA), thyroglobulin antibody (TGA), and thyroid microsomal antibody (TMA) are organ-specific autoantibodies associated with autoimmune disorders. This study primarily aimed to determine the frequencies of serum GPCA, TGA, and TMA positivity in patients with oral lichen planus (OLP).
Materials and methods: Serum levels of GPCA, TGA, and TMA were evaluated in 588 OLP patients and in 588 age- and sex-matched healthy control subjects.
Results: Among 588 OLP patients, 23.6 %, 27.4 %, and 28.4 % tested positive for GPCA, TGA, and TMA, respectively, compared with 2.2 %, 2.0 %, and 2.6 % in the control group. Each autoantibody was significantly more prevalent in OLP patients than in healthy control subjects (all *P-values* < 0.001). In addition, 31 (5.3 %), 122 (20.7 %), and 130 (22.1 %) OLP patients demonstrated triple (GPCA + TGA + TMA), dual (GPCA + TGA, GPCA + TMA, or TGA + TMA), or single (GPCA only, TGA only, or TMA only) autoantibody positivity, respectively, compared with 3

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(0.5 %), 10 (1.7 %), and 11 (1.9 %) healthy control subjects. Among the 159 TGA/TMA-positive OLP patients whose serum thyroid-stimulating hormone (TSH) levels were assessed, 81.1 % exhibited TSH levels within the normal reference range, whereas 11.3 % and 7.6 % showed decreased and elevated TSH levels, respectively.
Conclusion: Approximately 48.1 % of the 588 OLP patients demonstrated positivity for GPCA, TGA, and/or TMA. GPCA-positive individuals may be at risk for pernicious anemia, autoimmune atrophic gastritis, and gastric carcinoma, and TGA/TMA-positive individuals may exhibit thyroid dysfunction; thus, these autoantibody-positive patients should be referred for appropriate medical evaluation and follow-up.
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Introduction

Oral lichen planus (OLP) is a chronic, immune-mediated mucocutaneous disorder characterized by relapsing inflammatory lesions of the oral mucosa.[1,2] It affects approximately 1–2% of the general population and is more frequently observed in middle-aged and older adults, particularly women. Clinically, OLP presents in several forms, ranging from asymptomatic reticular lesions to painful erosive or ulcerative variants that significantly impair patients' quality of life.[3,4] Although the precise etiology of OLP remains unclear, increasing evidence supports the concept that it represents a T-cell-mediated autoimmune disease directed against basal keratinocytes.[1–6] This immunological basis has led to ongoing scientific interest in evaluating systemic autoimmune markers in OLP patients.

The presence of serum gastric parietal cell antibodies (GPCAs) can lead to autoimmune-mediated destruction of parietal cells, subsequently causing atrophic gastritis and impaired secretion of intrinsic factor. Deficiency of intrinsic factor compromises vitamin B12 absorption in the terminal ileum, ultimately resulting in pernicious anemia (PA).[7–10] Serum thyroglobulin antibodies (TGAs) and thyroid microsomal antibodies (TMAs; also referred to as anti–thyroid peroxidase or anti-TPO antibodies) are markers associated with autoimmune thyroiditis, particularly Hashimoto's thyroiditis, which frequently progresses to hypothyroidism.[11] Our previous investigation demonstrated GPCA positivity in 139 (23.6 %) of 588 OLP patients.[4] Nevertheless, the prevalence of serum TGA and TMA in this large OLP cohort has not yet been documented.

In our oral mucosal disease clinic, patients presenting with atrophic glossitis (AG), burning mouth syndrome (BMS), OLP, recurrent aphthous stomatitis (RAS), oral submucous fibrosis (OSF), and oral precancers are frequently encountered, whereas Behçet's disease is observed less commonly.[12–61] For individuals with any of these six oral mucosal diseases, laboratory evaluations including complete blood count and serum iron, vitamin B12, folic acid, homocysteine, and GPCA, TGA, and TMA levels are routinely performed because a subset of these patients may exhibit anemia, hematinic deficiencies, hyperhomocysteinemia, or seropositivity for GPCA, TGA, and TMA.[12–61] Furthermore, assessment of serum GPCA, TGA, and TMA is clinically relevant, as GPCA positivity is associated with an increased likelihood of PA and autoimmune atrophic gastritis, the latter of which may subsequently progress to gastric carcinoma.[62,63] Likewise, TGA and/or TMA positivity may indicate a predisposition to autoimmune thyroid diseases that can ultimately lead to thyroid dysfunction.[11,64] To facilitate early detection and management of these potential comorbidities, it is important to determine the proportion of OLP patients who exhibit GPCA, TGA, and TMA positivity, as well as the prevalence of concurrent positivity for two or all three of these autoantibodies.

In this study, serum autoantibodies including GPCA, TGA, and TMA were evaluated in 588 OLP patients and in an equal number of age- and sex-matched healthy control subjects. The objectives were to determine the proportion of OLP patients exhibiting GPCA, TGA, or TMA positivity; to assess the prevalence of concurrent positivity for two or all three of these autoantibodies; to ascertain whether individuals who were TGA- and/or TMA-positive (TGA/TMA-positive) demonstrated evidence of thyroid dysfunction; and to examine whether the frequencies of GPCA, TGA, and TMA positivity were significantly higher in OLP patients compared with healthy control subjects.

Materials and methods

Subjects

This study included 588 OLP patients (110 men and 478 women; age range, 20–88 years; mean age, 55.8 ± 14.1 years). For each OLP patient, one age- (±2 years) and sex-matched healthy control subject was recruited, resulting in 588 healthy control subjects (110 men and 478 women; age range, 20–89 years; mean age, 56.0 ± 14.1 years). All patients and healthy controls were consecutively examined, diagnosed, and treated in the Department of Dentistry at the National Taiwan University Hospital (NTUH) between July 2007 and June 2023. The diagnosis of OLP in the 588 patients was based on the following criteria: (i) the presence of characteristic clinical manifestations, including radiating grayish-white Wickham striae, papules, plaques

(alone or in combination), and areas of erosion or ulceration on the oral mucosa, and (ii) a bilateral and symmetrical distribution of the oral lesions. For the 25 patients in whom the clinical diagnosis was uncertain, an incisional biopsy of a representative oral lesion was performed. Histopathologic confirmation of OLP required the presence of established microscopic hallmarks, such as hyperkeratosis or parakeratosis, mild epithelial acanthosis with liquefaction degeneration of basal cells, a dense and band-like lymphocytic infiltrate in the lamina propria, and the absence of epithelial dysplasia.[4,45–52] Patients were excluded if they had a history of betel-quid chewing; autoimmune diseases (e.g., systemic lupus erythematosus, rheumatoid arthritis, Sjögren's syndrome, pemphigus vulgaris, cicatricial pemphigoid); inflammatory disorders; malignancy; or recent surgery. OLP patients with serum creatinine levels indicative of renal dysfunction (men >131 μmol/L; women >115 μmol/L), as well as those with a history of stroke, heavy alcohol consumption, or liver, kidney, or coronary artery disease, were also excluded.[4,45–52] Healthy control subjects presented only with dental caries or mild periodontal disease and had no oral mucosal or systemic diseases. None of the OLP patients had received any OLP-related medication for at least three months prior to enrollment.

The blood samples were drawn from 588 OLP patients and 588 healthy control subjects for measurement of serum GPCA, TGA and TMA levels mainly. All OLP patients and healthy control subjects signed the informed consent forms before entering the study. This study was reviewed and approved by the Institutional Review Board at the NTUH (202402086RINC).

Determination of serum gastric parietal cell antibody level

Serum GPCA levels were assessed using an indirect immunofluorescence assay employing rat gastric tissue as the substrate, as described in previous studies.[4,45–52] A serum sample was classified as positive when a specific fluorescent signal was observed at a dilution of 1:10 or higher.

Determination of serum thyroglobulin or thyroid microsomal antibody level

Serum TGA and TMA concentrations were quantified using a chemiluminescent microparticle immunoassay. A sample was considered positive for TGA when the concentration exceeded 14.4 IU/mL, and positive for TMA when the concentration was greater than 5.6 IU/mL.[13]

Determination of blood hemoglobin, iron, vitamin B12, folic acid, and homocysteine concentrations

In addition to measurement of serum GPCA, TGA and TMA levels, the complete blood count and serum iron, vitamin B12, folic acid, and homocysteine concentrations were also determined by the routine tests performed in the Department of Laboratory Medicine, NTUH.[4,45–52]

Statistical analysis

Comparisons of the frequencies of individual serum autoantibodies (GPCA, TGA, or TMA), as well as the frequencies of concomitant positivity for one, two, or all three autoantibodies, between the 588 OLP patients and 588 healthy control subjects were conducted using the chi-square test. The chi-square test was also used to compare the distribution of serum TSH levels between 159 TGA/TMA-positive OLP patients and 100 healthy control subjects. A *P*-value of less than 0.05 was considered statistically significant.

Results

Table 1 presents the comparative frequencies of serum autoantibodies including GPCA, TGA, and TMA between the 588 OLP patients and 588 healthy control subjects. Among the OLP cohort, 23.6 %, 27.4 %, and 28.4 % tested positive for GPCA, TGA, and TMA, respectively, whereas the corresponding frequencies in healthy controls were 2.2 %, 2.0 %, and 2.6 %, respectively. Each autoantibody was significantly more prevalent in OLP patients than in healthy control subjects (all *P*-values <0.001) (Table 1).

We also observed that a subset of OLP patients exhibited one, two, or all three organ-specific autoantibodies including GPCA, TGA, and TMA in their sera. The numbers and frequencies of patients with single, dual, or triple autoantibody positivity among the 588 OLP patients and 588 healthy control subjects are summarized in Table 2. Among the OLP patients, 31 (5.3 %), 122 (20.7 %), and 130 (22.1 %) demonstrated triple (GPCA + TGA + TMA), dual (GPCA + TGA, GPCA + TMA, or TGA + TMA), or single (GPCA only, TGA only, or TMA only) autoantibody positivity, respectively. In contrast, the corresponding frequencies in healthy control subjects were 3 (0.5 %), 10 (1.7 %), and 11 (1.9 %), respectively. Overall, OLP patients showed significantly higher frequencies of triple, dual, and single autoantibody positivity compared with healthy control subjects (all *P*-values <0.05) (Table 2).

Table 1 The patient number and frequencies of presence of serum autoantibodies including gastric parietal cell antibody (GPCA), thyroglobulin antibody (TGA), and thyroid microsomal antibody (TMA) in 588 oral lichen planus (OLP) patients and in 588 age- and sex-matched healthy control subjects.

Group	Autoantibody-positive patient number (%)		
	GPCA	TGA	TMA
OLP (n = 588)	139 (23.6)	161 (27.4)	167 (28.4)
P-value[a]	<0.001	<0.001	<0.001
Healthy control subjects (n = 588)	13 (2.2)	12 (2.0)	15 (2.6)

[a] Comparisons of different serum autoantibody frequencies between 588 OLP patients and 588 healthy control subjects by chi-square test.

Table 2 The patient number and frequencies of presence of one, two or three organ-specific autoantibodies such as gastric parietal cell antibody (GPCA), thyroglobulin antibody (TGA), and thyroid microsomal antibody (TMA) in 588 oral lichen planus (OLP) patients and in 588 age- and sex-matched healthy control subjects.

Autoantibodies	Patient number (%)		
	OLP patients (n = 588)	Healthy control subjects (n = 588)	*P*-value[a]
GPCA + TGA + TMA	31 (5.3)	3 (0.5)	<0.001
GPCA + TGA	10 (1.7)	2 (0.3)	0.042
GPCA + TMA	19 (3.2)	3 (0.5)	0.001
TGA + TMA	93 (15.8)	5 (0.9)	<0.001
GPCA only	79 (13.4)	5 (0.9)	<0.001
TGA only	27 (4.6)	2 (0.3)	<0.001
TMA only	24 (4.1)	4 (0.7)	<0.001
None	305 (51.9)	564 (95.9)	<0.001

[a] Comparisons of frequencies of presence of one, two or three serum autoantibodies including GPCA, TGA and TMA between 588 OLP patients and 588 healthy control subjects by chi-square test.

In this study, serum TSH levels were assessed in 159 of the 204 TGA/TMA-positive OLP patients and in 100 healthy control subjects (Table 3). All healthy control subjects and 81.1 % of the TGA/TMA-positive OLP patients exhibited TSH values within the normal reference range (0.4–4.0 μIU/mL). In contrast, 18 (11.3 %) TGA/TMA-positive OLP patients had decreased TSH levels (<0.4 μIU/mL), and 12 (7.6 %) TGA/TMA-positive OLP patients had elevated TSH levels (>4.0 μIU/mL) (Table 3).

Table 3 The number and percentage of individuals with different serum levels of thyroid-stimulating hormone (TSH) in 159 thyroglobulin antibody (TGA)-positive and/or thyroid microsomal antibody (TMA)-positive (TGA/TMA-positive) oral lichen planus (OLP) patients and in 100 healthy control subjects.

TSH level	TGA/TMA-positive OLP patients (n = 159)	Healthy control subjects (n = 100)	*P*-value[a]
< 0.4 μIU/mL	18 (11.3 %)	0 (0 %)	0.001
0.4–4.0 μIU/mL	129 (81.1 %)	100 (100 %)	<0.001
>4.0 μIU/mL	12 (7.6 %)	0 (0 %)	0.012

[a] Comparison of frequency of patients with different levels of TSH between 159 TGA/TMA-positive OLP patients and 100 healthy control subjects by chi-square test.

Discussion

In this study, the seropositivity rates of GPCA, TGA, and TMA were 23.6 %, 27.4 %, and 28.4 %, respectively, among 588 OLP patients, compared with 2.2 %, 2.0 %, and 2.6 % in 588 age- and sex-matched healthy controls, respectively. These results demonstrate a markedly higher prevalence of these autoantibodies in OLP patients than in healthy controls. In an earlier investigation conducted by our group, GPCA, TGA, and TMA were detected in 26.3 %, 21.3 %, and 24.4 % of 320 OLP patients, respectively.[14] Minor variations in seropositivity rates between the two studies are likely attributable to differences in sample size.

Our earlier investigations reported that the seropositivity rates of GPCA, TGA, and TMA ranged from 10.7 % to 26.7 %, 4.6 %–28.4 %, and 5.5 %–29.8 %, respectively, across six other oral mucosal diseases including AG, BMS, RAS, OSF, oral precancers, and Behçet's disease.[12,13,15–18] Comparative analyses of these six studies together with the current dataset indicate that AG or OLP patients consistently exhibit higher rates of GPCA, TGA, and TMA seropositivity than those with BMS, RAS, OSF, oral precancers, or Behçet's disease.[12,13,15–18]

Elevated serum TSH levels together with the presence of TGA and/or TMA constitute two principal diagnostic criteria for chronic autoimmune thyroiditis.[11] In the present study, serum TSH concentrations were evaluated in 159 TGA/TMA-positive OLP patients. Among them, 129 patients (81.1 %) exhibited TSH values within the normal range (consistent with euthyroid status), whereas 18 (11.3 %) and 12 (7.6 %) showed reduced (suggestive of hyperthyroidism) and elevated TSH levels (suggestive of hypothyroidism), respectively. Our earlier investigations also assessed serum TSH levels in TGA/TMA-positive patients with various oral mucosal diseases. Normal, decreased, and increased TSH levels were observed in 78.6 %, 8.0 %, and 13.4 % of 373 AG patients;[12] 87.8 %, 5.1 %, and 7.1 % of 255 BMS patients;[13] 76.9 %, 12.3 %, and 10.8 % of 65 RAS patients;[15] 80.0 %, 10.0 %, and 10.0 % of 10 oral precancer patients;[17] 87.5 %, 6.3 %, and 6.3 % of 16 Behçet's disease patients;[18] 84.3 %, 6.7 %, and 9.0 % of 210 erosive OLP patients with desquamative gingivitis.[19] Furthermore, community-based surveys have reported that 50–75 % of TGA/TMA-positive subjects are euthyroid, 25–50 % exhibit subclinical hypothyroidism, and only 5–10 % have overt hypothyroidism.[11] Overall, the findings across these studies are consistent and indicate that most TGA/TMA-positive patients with oral mucosal diseases (approximately 76.9–87.8 %) maintain euthyroid status. In contrast, only a minority demonstrate biochemical evidence of hyperthyroidism (6.3–12.3 %) or hypothyroidism (6.3–13.4 %).[12,13,15,17–19]

Oral mucosal disease patients who are positive for serum GPCA are considered at increased risk for PA, as GPCA-mediated autoimmune destruction of gastric parietal cells can lead to partial or complete loss of intrinsic factor secretion and consequently impair vitamin B12 absorption in the terminal ileum.[7–10] Our previous investigations reported PA in 17 (12.2 %) of 139 GPCA-positive OLP patients,[4] 22 (7.7 %) of 284 GPCA-positive AG patients,[20] 15 (13.8 %) of 109 GPCA-positive BMS patients,[34] 3 (7.3 %) of 41 GPCA-positive erosive OLP patients,[51] 13 (14.1 %) of 92 GPCA-

positive erosive OLP patients with desquamative gingivitis,[52] 4 (8.7 %) of 46 GPCA-positive RAS patients,[53] and 1 (11.1 %) of 9 GPCA-positive Behçet's disease patients.[60] Collectively, these data demonstrate that GPCA positivity does not uniformly translate into the development of PA; rather, only approximately 7.3—14.1 % of GPCA-positive patients with oral mucosal diseases exhibit clinical evidence of PA.[4,20,34,51—53,60]

As noted previously, if appropriate early diagnosis and management are not provided, patients who are positive for GPCA have an elevated risk of developing PA and autoimmune atrophic gastritis, the latter of which may progress to gastric carcinoma.[62,63] Likewise, individuals who are positive for TGA and/or TMA may develop autoimmune thyroid disease, eventually leading to thyroid dysfunction.[11,64] Therefore, oral mucosal disease patients who are TGA/TMA-positive and exhibit either hyperthyroidism or hypothyroidism should be referred to an endocrinologist for further evaluation and treatment. Additionally, GPCA-positive patients should be referred to gastroenterology for endoscopic assessment of the gastric mucosa to determine the presence of autoimmune atrophic gastritis, which can then be managed appropriately. A long-term follow-up study is also warranted to determine whether GPCA-positive patients with oral mucosal diseases, regardless of treatment status, may ultimately develop gastric carcinoma.[63]

Human leukocyte antigen (HLA) class II molecules are known to exhibit strong associations with various autoimmune diseases. In Southern Chinese populations, the HLA-DR9 allele has been linked to an increased risk of autoimmune diseases such as Graves' disease, myasthenia gravis, insulin-dependent diabetes mellitus, and Hashimoto's thyroiditis.[65—67] In Caucasian populations, HLA-DR3 has similarly been associated with systemic lupus erythematosus, Graves' disease, myasthenia gravis, insulin-dependent diabetes mellitus, and erosive OLP.[67,68] With respect to oral mucosal diseases, our previous work identified a strong correlation between HLA-DRw9 and RAS in Chinese patients, as well as an association between antiepithelial cell antibodies and the HLA-DR3 or HLA-DR7 phenotype in RAS patients.[69,70] These findings suggest that the links between HLA-DRw9 and RAS, and between HLA-DR3/DR7 and autoantibody production, may partly account for the presence of GPCA, TGA, and TMA in a minority of RAS patients.[69,70] However, whether specific HLA class II alleles are associated with AG, BMS, OLP, OSF, oral precancers, or Behçet's disease in Taiwanese patients remains unknown. Further research is therefore warranted to elucidate the mechanisms underlying autoantibody generation in subsets of patients with these oral mucosal diseases.

Several limitations should be acknowledged in this study. First, the underlying mechanisms responsible for the presence of organ-specific autoantibodies, namely GPCA, TGA, and TMA, in OLP patients remain unclear. Second, follow-up data were not available; therefore, it is unknown how many GPCA-positive OLP patients may ultimately progress to autoimmune atrophic gastritis or gastric carcinoma over an extended observation period.[62,63] Third, it has yet to be determined whether intramuscular vitamin B12 therapy can delay or prevent the development of autoimmune atrophic gastritis and subsequent gastric carcinoma in GPCA-positive OLP patients.[71] Finally, it is also unclear whether TGA/TMA-positive OLP patients who are currently euthyroid may later develop autoimmune thyroid disease and eventually experience thyroid dysfunction.[11,64]

In conclusion, 23.6 %, 27.4 %, and 28.4 % of the 588 OLP patients examined were seropositive for GPCA, TGA, and TMA, respectively. Additionally, 5.3 %, 20.7 %, and 22.1 % of these OLP patients exhibited one, two, or all three of these autoantibodies, respectively. Among the 159 TGA/TMA-positive OLP patients, 11.3 % demonstrated biochemical evidence of hyperthyroidism and 7.6 % showed hypothyroidism. Further investigation is required to determine whether GPCA-positive OLP patients may eventually progress to autoimmune atrophic gastritis or gastric carcinoma.[62,63]

Declaration of competing interest

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

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None.

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