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Original Article

Potential relationship of dyslipidemia with dietary patterns in oral lichen planus patients-A case-control study

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KEYWORDS
Oral lichen planus;
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Abstract *Background/purpose:* Dyslipidemia and a high fat diet may increase the predisposition for accumulating body fat in patients with oral lichen planus (OLP). This study aimed to investigate the risk factors obesity, dietary patterns, and lipid metabolism.
Materials and methods: A population-based case-control study was conducted between September 2020 and October 2021, recruiting 275 pairs of OLP cases and controls. Information on lipid profiles, diet frequency and waist circumference were gathered. Principal component and factor analysis were used to analyze the semi-quantitative dietary frequency survey data of patients to extract specific dietary patterns.
Results: Univariate analysis showed that total cholesterol, triglycerides, and low-density lipoprotein were significantly higher in the OLP group than the control and other oral mucosal disease groups ($P < 0.05$, $P < 0.001$, and $P < 0.001$, respectively). Compared with the baseline group, obese and overweight patients were more common in the OLP group. Dyslipidemia

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was more common in the OLP group (68%) compared to the healthy mucosa group (32%; $P < 0.001$, OR = 4.52, 95% CI = 2.49–8.18). Four dietary patterns were described among the subjects. The traditional prone animal food pattern (OR: 24.81, 95% CI: 6.05–101.71, $P < 0.001$) and animal food pattern (OR: 28.77, 95% CI: 8.10–102.15, $P = 0.001$) were associated with an increased risk of OLP.

Conclusion: The results indicated that a high-fat diet, dyslipidemia and obesity were strongly linked to disease progression in OLP. A diet high in processed food and fat could increase the risk of OLP.

Introduction

Oral lichen planus (OLP) is a long-term inflammatory oral mucosal disease mediated by cellular immunity that predominates in middle-aged female with a prevalence of 0.1%–4%.[1,2] In particular, patients with erosive and atrophic OLP often suffer from painful feeding irritation and difficulty swallowing, causing a serious impact on their quality of life.[3] Recurrent erosive lesions are at risk of malignant transformation and OLP are categorized by the World Health Organization as potentially malignant diseases of the oral cavity.[4] Although the pathogenic mechanisms and triggering factors of OLP remain unknown, it is considered to represent a disorder of immune regulation mediated by T cells, in which CD4+T and CD8+T lymphocytes trigger the apoptosis of epithelial basal cells, the target cells in OLP.[5] Some predisposing factors associated with OLP are infection, genetic and psychological factors and systemic diseases.[2]

Recent studies have suggested a link between OLP and metabolic syndrome.[6] Obese and hyperlipidemic individuals are susceptible to a number of chronic diseases, including inflammatory autoimmune diseases; the underlying mechanism of these may involve secretion of pro-inflammatory cytokines.[7] This is supported by evidence of increasing incidence of dyslipidaemia in skin conditions like psoriasis, pemphigus, lupus erythematosus and lichen planus.[8–10] At present, only three clinical studies have investigated the relationship between OLP and abnormal serum lipid metabolism. Of these, two showed a correlation between OLP and dyslipidemia, suggesting that OLP may interact with the metabolic state of the body,[11,12] while the other study showed the opposite result.[13]

A high fat diet and obesity can amplify low-grade inflammation through infiltration of pro-inflammatory macrophages, oxidative stress, hypoxia, and lipolysis.[14] The significance of dietary factors in chronic inflammatory conditions has been confirmed by several previous studies (e.g., systemic lupus erythematosus, psoriasis, and rheumatoid arthritis).[15–17] Since OLP is also a chronic inflammatory autoimmune condition, we hypothesized that dyslipidaemia, a high-fat diet and obesity are pro-inflammatory factors which contribute to the development and progression of OLP. Therefore, we conducted a case-control trial to assess obesity, dietary patterns, and lipid metabolism in patients with OLP.

Materials and methods

Participants

This case-control study was designed and implemented in accordance with STROBE specifications. Three groups were included in this study: the OLP group, control group and other oral mucosal diseases group. Patients with OLP were diagnosed based on the 2003 clinicopathological criteria.[18] The inclusion criteria for OLP were: 1) aged 18 to 75 year, and 2) patients who met the clinical and pathological criteria of OLP and where no medication has been used to treat the lesion.[18] Patients with healthy oral mucosa and patients with other oral mucosa diseases including recurrent oral ulcer, oral leukoplakia, burning mouth syndrome, and oral candidiasis in our hospital during the same period were selected as the baseline groups, whose age and gender were matched with the OLP group. Exclusion criteria for all subjects were: 1) presence of other systemic diseases besides dyslipidemia, 2) acute infection or malignancy of the body, 3) patients had suffered from lichenoid drug eruption or were receiving treatment for OLP such as systemic corticosteroids, retinoic acid or methotrexate and 4) pregnancy. A final total of 275 were enrolled in this study patients from September 2020 to October 2021. This study was approved by the Ethical Committee of Peking University (permission number PKUSSIRB-202162036). All subjects completed informed consent before participating in the study. The dataset had no missing values.

Data collection

A questionnaire was designed to collect participants' basic information (such as gender, age, educational background, family income, smoking etc.), and diet status (Dietary Frequency Questionnaire, FFQ-25). The questionnaire was conducted in the form of face-to-face interviews at the patient's first visit. Another researcher checked the daily data, and those who failed to complete more than 30% of the questionnaire were eliminated.

A 25-item food Frequency Questionnaire (FFQ-25), which has been shown to be moderately equivalent and reproducible in the Chinese population,[19] was utilized to assess participants' dietary intake over the past 3 months. The design of FFQ-25 mainly refers to previous studies on dietary

patterns abroad and semi-quantitative food frequency questionnaires in Chinese studies that are related to chronic diseases of middle-aged and elderly people, which is in line with the prevalence of OLP in middle-aged and elderly women. A total of 25 food categories are included: rice, porridge, flour food, sweets, fried food, stuffing food, coarse grains, potatoes, red meat, processed meat products, fresh river, seafood, bean products et al. The frequency of food intake was defined as: 0) never eat; 1) less than once a month; 2) 1—3 times per month; 3) 1—2 times a week; 4) 3—4 times a week; 5) 5—6 times a week; 6) once a day; 7) twice a day; 8) more than 3 times a day. Food intake is divided into 6 categories: 1) 50 g and below; 2) 100 g; 3) 150 g; 4) 200 g; 5) 250 g and above; 6) not applicable. Products such as eggs, drinks, and beer were selected as discrete quantities.

Anthropometric measurements were obtained using standardized techniques and calibrated equipment. Body mass index (BMI) was calculated as weight (kg) divided by the square of height. According to the BMI grouping method recommended by the Chinese Adiposity Group, the BMI measurement for Chinese adults is $<$ 18.4 for underweight, 18.5—23.9 for normal, 24—27.9 for overweight and $>$28 for obese.[20] Waist circumference was measured with a plastic tape measure at the midpoint between the base of the rib cage and the top of the iliac crest at minimal breathing, to an accuracy of 0.5 cm.

Lipid level measurement: total serum cholesterol (CHO), triglyceride (TG), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), and glucose levels were studied in samples drawn between 8 a.m. and 9 a.m. after a 12-h fasting period. Dyslipidemia criteria were: TG $>$ 150 mg/dL, total cholesterol $>$200 mg/dL, LDL $>$130 mg/dL, and HDL $<$40 mg/dL.[21]

Statistical analysis

A *t*-test or chi-square test was used to describe differences in demographic variables between the case and control groups. Principal component and factor analysis (PCFA) was performed using relevant matrices for 25 dietary groups to derive dietary patterns. Bartlett's test of sphericity and the Kaiser-Meyer-Olkin sampling test (KMO) were used to identify the linearity of the individual variables and the applicability of PCFA. An orthogonal (varimax) transformation was used to simplify the structure and make the outcomes interpretable. To identify the number of factors, the eigenvalues ($>$1.0), the construction of scree plots and the total variance and interpretability of the factors were considered.[22] In determining the number of retained factors, in addition to the size of the characteristic roots of each factor and their placement on the screen plots, the rationality of the food combination was also considered.

Within each model, patients were divided into four subgroups according to the quartiles of the various dietary pattern scores of the normal control group. Crude and adjusted odds ratios (OR) and 95% confidence intervals (95% CI) were estimated from univariate and multivariate logistic regression (forward stepwise) models, respectively. SPSS (version 19.0, IBM, Armonk, NY, USA) was used for statistical analysis and tests were two-tailed, with $P < 0.05$ regarded as significant.

Results

Table 1 displays the basic information of the participants. There were no significant differences among the three groups in gender, age, family income, smoking, height, or weight. The educational level of the OLP group was significantly lower than the other groups ($P < 0.01$). Mean waist circumference in the OLP group was significantly ($P < 0.01$) higher than the control group and other oral mucosal disease group. BMI in the OLP was significantly higher than in the control and other oral mucosal disease groups ($P < 0.001$). Compared with the control group and other oral mucosal disease group, there were significantly more obese and overweight participants in the OLP group ($P < 0.05$). Univariate analysis showed that TG, total cholesterol, and LDL were significantly higher in the OLP group than the controls and other oral mucosal disease group ($P < 0.05$, $P < 0.001$, and $P < 0.001$, respectively). The prevalence of dyslipidemia in patients with OLP was 68%, higher than the 32% in the control group and 43% in the other oral mucosal disease group (Table 2).

An overall KMO value of 0.605 ($>$0.5 is considered admissible) and a p-value of $<$0.001 for Bartlett's test of sphericity ($<$0.001 is considered admissible) indicated that there was adequate sample size for PCFA.[22] Ultimately, four common factors were derived (Table 3) and explained 44% of the total variance. The first factor, defined as 'health pattern', was characterized by relatively high factor loadings for river fish, seafood, coarse grains, rice and desserts, explaining 14.1% of the total variance. The second factor, labeled as "traditional prone animal food pattern", had a higher factor loading of red meat, poultry, white-color vegetables, fruits, dairy products, coarse grains, eggs, and soy products and explained 10.3% of total variance. The third factor, labeled as "traditional prone plant food pattern", had high loadings of wheaten food, fried food, dark-color vegetables, white-color vegetables, coarse grains, and stuffing and explained 10.1% of total variance. The fourth factor, labeled as "animal food pattern", had a high loading of rice, fried food, red meat, processed meat, dark-color vegetables, and soy products and explained 9.9% of total variance.

A multivariate analysis found that traditional prone animal food and animal food patterns were related to an increased risk of OLP, using the control group as a baseline. The highest quartile of pattern scores was related to a 25-fold risk for the traditional prone animal food pattern and the animal food pattern compared to the quartile with the lowest score. With the other oral mucosal diseases group as the control, the animal food pattern was related to disease progression in OLP. The quartile with the highest mode score had a nearly 3-fold risk of being linked to the animal food pattern compared to the quartile with the lowest score (Table 4).

Discussion

The results of this study confirmed the initial hypothesis that obesity, dyslipidaemia and a high-fat diet are risk factors for OLP. This study found higher serum lipid levels in patients with OLP than the control and other oral mucosal

Table 1 Socio-demographic, anthropometric and biochemical characteristics of the controls and the patients of oral lichen planus and other oral mucosal disease.

	OLP (n = 100)	Controls (n = 100)	OMD(n = 75)	*P*
Age (years, mean ± SD)	46.11 ± 9.54	46.17 ± 9.22	48.91 ± 15.25	0.356
Gender, n (%)				
Female	71 (71%)	66 (66%)	44 (59%)	0.235
Male	29 (29%)	34 (34%)	31 (41%)	
Education level, n (%)				
Bachelor's degree or above	21 (21%)	25 (25%)	21 (28%)	**<0.01**
High school	18 (18%)	38 (38%)	26 (35%)	
Lower than middle school	61 (61%)	37 (37%)	28 (37%)	
Family income (RMB)				
High (≥3000)	32 (32%)	25 (25%)	17 (23%)	0.339
Intermediate (1000–2999)	45 (45%)	58 (58%)	40 (53%)	
Low (<1000)	23 (23%)	17 (17%)	18 (24%)	
Smoking, n (%)				
Smoker	16 (16%)	23 (23%)	16 (21%)	0.439
Non-smoker	84 (84%)	77 (77%)	59 (79%)	
Height (cm), median (IQR)	165.0 (15)	166.5 (18)	166.0 (17)	0.532
Weight (kg), median (IQR)	65.5 (17.75)	63.0 (17.75)	67.0 (16.00)	0.431
Waist circumference (cm)	83.06 ± 6.87	80.61 ± 6.51	81.02 ± 6.13	**0.002**
BMI (kg/m^2)	24.58 ± 2.52	23.43 ± 1.76	23.76 ± 1.85	**<0.001**
BMI≥28 (obesity)	16 (16%)	6 (6%)	7 (9.3%)	
28 > BMI≥24 (overweight)	44 (44%)	32 (32%)	28 (37.3%)	
24 > BMI≥18.5 (normal)	40 (40%)	62 (62%)	40 (53.3%)	**0.021**

OLP: oral lichen planus; OMD: other oral mucosal disease; cm:centimeter; IQR: interquartile range; kg:kilogram; FFQ-25: 25-item food Frequency Questionnaire.

Table 2 Serum lipid profiles and dyslipidemia in the OLP, control and other oral mucosal disease groups.

Lipid or lipoprotein	OLP	Control	OMD	*P*	OR	95% CI
	(n = 100)	(n = 100)	(n = 75)		(OLP vs control)	
Triglycerides (mg/dL) (median)	147.96	112.08	107.21	0.002		
Total cholesterol (mg/dL)	213.02	182.28	175.90	<0.001		
LDL cholesterol (mg/dL)	125.26	102.26	104.38	<0.001		
HDL cholesterol (mg/dL)	49.87	53.16	49.48	0.01		
Dyslipidemia						
Yes	68	32	32			
No	32	68	43	<0.00	4.52	2.49–8.18

OLP: oral lichen planus; OMD: other oral mucosal disease.

diseases groups. After controlling for education level, waist circumference and BMI, patients with OLP presented a higher risk for dyslipidemia. A long-term high-fat diet is a key risk factor for dyslipidemia, which leads to inflammation and might promote the occurrence and development of OLP.[23] In this research, healthy, traditional prone plant food, traditional prone animal food and animal food dietary patterns were defined. Compared to the control group, the traditional prone animal food and the animal food groups were found to be associated with an increased risk of OLP, while the traditional prone plant food pattern was associated with a reduced risk. In addition, there was no significant correlation between healthy pattern and OLP group.

OLP is a chronic inflammatory skin mucosal disease mediated by cellular immunity, with an unknown cause. Dyslipidemia has been reported to be more common in patients with OLP. A 2016 meta-analysis showed that OLP was significantly related to a higher risk of dyslipidemia and elevated triglyceride levels (OR: 1.74, 95% CI: 1.19–2.54).[24] However, most of the included studies were retrospective trials and the diagnosis of OLP disease did not involve histopathology. In our study, lipid indexes (TG, CHO, and LDL) of OLP patients were markedly higher than in the baseline groups. The results were similar to a previous study which showed a significantly higher frequency of dyslipidemia in patients with LP.[25]

The current study found that most patients in the OLP group had lower levels of education compared to controls. Epidemiological surveys in some countries (e.g., Sweden[26] and India[27]) have also identified low levels of education

Table 3 The factor loadings of FFQ-25 in four dietary patterns.

Food groups	Healthy pattern	Traditional prone animal food pattern	Traditional prone plant food pattern	Animal food pattern
Rice	**−0.39**			**0.61**
Wheaten food	0.21	−0.17	**0.78**	0.11
Desserts	**0.70**			
Fried food	0.18		**0.62**	**0.34**
Dairy products	0.44	**0.32**		−0.28
Red meat	−0.13	**0.38**		**0.57**
Poultry	−0.15	**0.47**	−0.14	
River fish	**0.71**		0.13	0.12
Marine products	**0.68**			
Dark-color vegetables		−0.22	**−0.41**	**0.38**
White-color vegetables	−0.12	**−0.60**	**−0.31**	
Fruits	0.25	**0.65**		0.22
Processed meat	0.11			**0.61**
Coarse grains	**0.53**	**−0.32**	**−0.37**	−0.23
Stuffing		0.12	**0.50**	−0.20
Eggs	0.26	**0.32**		
Soy products	0.18	**−0.42**	−0.14	**0.30**
Variance explained VAR (%)	14.07	**10.30**	10.14	**9.92**
Cumulative explained VAR (%)	14.07	**24.36**	34.51	**44.42**

FFQ-25: 25-item food Frequency Questionnaire; VAR: Variance.

Table 4 Associations between four dietary patterns and OLP risk.

Dietary pattern	OLP/control/OMD	Crude OR (95% CI) OLP & control	Adjusted OR (95% CI) OLP & control	Crude OR (95% CI) OLP & OMD	Adjusted OR (95% CI) OLP & OMD
Healthy					
Q1	22/23/23	1.00	1.00	1.00	1.00
Q2	19/26/18	0.76 (0.33, 1.76)	0.64 (0.25, 1.69)	1.10 (0.46, 2.64)	0.96 (0.35, 2.63)
Q3	25/26/22	1.01 (0.45, 2.24)	0.93 (0.37, 2.29)	1.19 (0.52, 2.69)	1.06 (0.42, 2.70)
Q4	34/25/12	1.42 (0.65, 3.10)	1.06 (0.43, 2.59)	2.96 (1.23, 7.14)	2.02 (0.74, 5.50)
P for trend		0.88	0.29	0.88	0.42
Traditional prone animal food					
Q1	3/25/4	1.00	1.00	1.00	1.00
Q2	10/25/14	3.33 (0.82, 13.58)	4.68 (1.01, 21.80)	0.95 (0.17, 5.23)	1.46 (0.24, 9.06)
Q3	21/25/25	7.00 (1.85, 26.49)	9.18 (2.11,40.00)	1.17 (0.23, 5.82)	1.36 (0.24, 7.71)
Q4	66/25/32	22.0 (6.10, 79.36)	24.81 (6.05, 101.71)	2.67 (0.56, 12.62)	3.01 (0.57, 15.83)
P for trend		**0.001**	**<0.001**	0.71	0.43
Traditional prone plant food					
Q1	9/25/9	1.00	1.00	1.00	1.00
Q2	14/25/30	1.56 (0.57, 4.25)	1.55 (0.48, 4.96)	0.47 (0.15, 1.43)	1.23 (0.38, 3.95)
Q3	9/25/10	1.00 (0.35, 2.94)	0.89 (0.26, 3.00)	0.90 (0.25, 3.27)	2.07 (0.55, 7.72)
Q4	68/25/26	7.56 (3.11, 18.38)	9.02 (3.19, 25.47)	2.62 (0.94, 7.32)	24.49 (7.59, 79.04)
P for trend		0.01	0.08	1.00	0.11
Animal food pattern					
Q1	26/25/6	1.00	1.00	1.00	1.00
Q2	18/25/24	0.69 (0.31, 1.57)	1.45 (0.49, 4.30)	0.17 (0.06, 0.51)	0.27 (0.08, 0.94)
Q3	21/25/12	0.81 (0.36, 1.80)	3.78 (1.18, 12.06)	0.40 (0.13, 1.26)	1.28 (0.33, 5.01)
Q4	35/25/33	1.35 (0.64, 2.85)	28.77 (8.10, 102.15)	0.25 (0.09, 0.67)	2.26 (0.57, 9.00)
P for trend		0.89	**0.001**	**0.001**	**0.02**

Q: Quartile; adjusted OR: was adjusted for educational levels, waist circumference (cm), BMI (continuous, kg/m^2), stress, triglycerides, total cholesterol, LDL cholesterol, and HDL cholesterol (all mg/dL); OLP: oral lichen planus; OMD: other oral mucosal disease group.

as a risk factor for hyperlipidemia. A recent prospective cohort study in China found that the less educated group was more likely to have dyslipidemia, especially in the case of high TG.[28] Therefore, we should pay more attention to the lipid profile of patients with OLP who have a low level of education, and those with dyslipidemia should be

treated with an active multidisciplinary approach. Furthermore, the effect of lipid alterations on such autoimmune inflammatory diseases suggests that the lipid profile may contribute to the development and exacerbation of inflammation in the body through a specific pathway.[29] One mechanism that might explain the relationship between dyslipidemia and OLP is that oxidized low-density lipoprotein promotes the release of pro-inflammatory cytokines and causes the body to be in a state of chronic low-grade inflammatory response, thus inducing or aggravating OLP.[23]

Dietary habits are closely related to weight and body metabolism. A long-term high fat diet is a key factor leading to overweight and dyslipidemia and can induce some chronic diseases. Therefore, the diet of OLP patients with dyslipidemia deserves our attention. In this study, the traditional prone animal pattern refers to eating more animal food, with higher factor loadings of red meat, poultry, eggs, and wheaten food. The animal food pattern is similar to a western-style diet with higher factor loadings of red meat, processed meat, and fried food. These types of foods are rich in animal fats, which are thought to be risk factors for metabolic and inflammatory diseases.[30] Both diets lead to higher intake of saturated and omega-6 fatty acids, and lower intake of omega-3 fats. They also trigger low-level inflammation in the body, leading to the development of obesity and metabolic syndrome. A permanent high-fat diet promotes a variety of lifestyle disorders that are associated with a number of physical and psychiatric problems caused by metabolic disorders, the most important of which are hyperinsulinemia, insulin resistance, dyslipidaemia, low-grade systemic inflammation, dysbiosis and increased production of oxidative stress.[31] A meta-analysis from 2021 found reduced total antioxidant capacity and overexpression of oxidative stress markers in OLP patients.[32] Therefore, dyslipidemia, low-grade systemic inflammation, dysbiosis, and increased production of oxidative stress can promote the occurrence and progress of OLP.[33]

Obesity has been regarded as a risk factor that can exacerbate the severity and chronicity of autoimmune conditions by secreting pro-inflammatory cytokines involved in the pathogenesis and progression of several autoimmune diseases.[29] We observed that OLP patients present a higher frequency of overweight and obesity than the control and other oral mucosal diseases groups. These findings suggest that overweight and dyslipidemia may play a role in the inflammatory process of OLP. Of note, increased gene and protein expression of several pro-inflammatory cytokines, such as IL-6, IL-8 and TNF-α, have been reported in patients with OLP.[30] All of the above proinflammatory factors can be secreted by white fat to promote local and systemic inflammation.[26] Therefore, the increased body fat content and high prevalence of obesity in OLP patients may promote the continued deterioration of their chronic inflammatory state.

This study is one of the few investigations to date to assess the dietary patterns of patients with OLP. Strengths of the research include the matched case-control design, the high response rate to the questionnaire and the inclusion of both normal controls and other oral mucosal disease groups. In addition, potential confounding factors that may have influenced the results were collected and adjusted for. Nevertheless, due to the retrospective nature of this study, the presence of information bias, particularly recall bias during the questionnaire process, may be an issue that needs to be addressed. We therefore attempted to reduce recall bias by blinding cases, controls and interviewers about the research hypotheses and objectives. In addition, standardized training was advocated in advance for the investigators in order to avoid human bias in the processing of information collection for the case and control groups. The FFQ used in our study was reasonably reproducible and valid in collecting information on the intake of common foods and beverages.[19] Another limitation was the small sample size; and since most patients were from northern China they are not necessarily representative of the majority of the Chinese population. Additionally, the food groups for the dietary survey are not sufficiently refined and need to be further enriched.

In conclusion, the results of this study suggest that dyslipidemia, obesity, and a high fat diet are risk factors for OLP. Traditional prone animal food and animal food patterns might contribute to the development of OLP. The study provided further support to the recommendation on monitoring the lipid profile of patients with OLP. OLP patients with obesity or hyperlipidemia are encouraged to reduce the intake of animal fat and increase the intake of vegetables and dietary fiber. Future rigorous prospective studies are needed to clarify the causal relationship between hyperlipidemia, obesity, a high-fat diet, and OLP. Further mechanistic studies could clarify the specific role of hyperlipidemia in OLP, with the aim of being able to implement interventions in the clinic to improve the cure rate of OLP.

Declaration of competing interest

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

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