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Prevalence of Oral Lichen Planus in Northern Thai Patients with Hepatitis C Virus Infection

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Abstract

Objectives: This study aimed to investigate the prevalence of oral lichen planus (OLP) among individuals with hepatitis C virus (HCV) infection in upper northern Thailand and the potential association between OLP and HCV infection.

Methods: This cross-sectional study involved 1,025 participants, comprising 126 patients with confirmed HCV infection and 899 participants without HCV. All participants underwent structured interviews and clinical oral examinations by calibrated examiners. The diagnosis of OLP was established based on characteristic clinical features and confirmed by histopathological examination of oral mucosal biopsies. The statistical analysis used Pearson's Chi-square test and Fisher's exact test, with a significance level of $p<0.05$.

Results: The prevalence of OLP was 2.38% in patients with HCV infection and 0.33% in those without HCV. The difference in OLP prevalence between the two groups was statistically significant ($p=0.027$), suggesting a possible association between HCV infection and the development of OLP.

Conclusions: The findings indicated a significant association between HCV infection and OLP in this northern Thai cohort. Therefore, screening for OLP in patients with HCV is recommended for early detection and appropriate management.

Keywords: cross-sectional studies, hepatitis c virus, oral lichen planus, prevalence, Thailand

Introduction

Hepatitis C virus (HCV), an RNA virus first identified in 1989, is transmitted through contact with infected blood. Common routes of transmission include sharing injecting equipment, sexual contact involving blood, tattooing, and receiving unscreened blood transfusions.[1] In 2022, approximately 50 million people worldwide were living with chronic HCV infection, resulting in an estimated 242,000 deaths and 1 million new infections annually.[2] Left untreated, chronic HCV infection can progress to cirrhosis, hepatocellular carcinoma, or liver-related death.[2] In addition to liver disease, HCV infection is associated with various extrahepatic manifestations, including mixed cryoglobulinemia, non-Hodgkin's lymphoma, diabetes, porphyria cutanea tarda, and oral lichen planus (OLP).[3]

OLP is a chronic T-cell-mediated inflammatory disease that affects the oral mucosa.[4] The global prevalence of OLP is 1.01%, with significant geographical variations.[5] It is commonly found in patients aged 30-60 years. The five clinical types of OLP are reticular, plaque, papular, atrophic, and ulcerative.[6] Histopathological features include a well-defined band-like infiltration of lymphocytes beneath the basement membrane, liquefaction degeneration of the basal cell layer, and an absence of epithelial dysplasia.[7] Since 2005, the World Health Organization has classified OLP as an oral potentially malignant disorder (OPMD), with a malignant transformation rate of approximately 1-2%.[8]

The association between OLP and HCV was first reported in 1991.[9] Subsequently, many studies globally have investigated this relationship, with conflicting findings. Although several studies have demonstrated a significant association between OLP and HCV[10-22], others have not demonstrated a consistent association.[23-32] In Thailand, two case-control studies have shown the association between OLP and HCV infection. Klanrit *et al*.,[19] reported that 8.33% of OLP patients were anti-HCV positive, while Manomaivat *et al*.,[22] found an 8.9% HCV seroprevalence in OLP cases versus 0.99% among controls (odds ratio ≈ 9.78). Additionally, international studies have revealed notable geographical variations in this association. Recent meta-analyses indicate that approximately 7% of HCV patients worldwide present with OLP, a prevalence markedly higher than the general population (<1%).[33]

Although previous Thai investigations suggest an association, no study has examined the prevalence of OLP among individuals with confirmed HCV infection in Thailand, including the northern region where demographic patterns differ. These variations highlight the need for local data. This study therefore aimed to determine the prevalence of OLP in HCV-infected and non-infected individuals in upper northern Thailand and to evaluate their potential association.

Materials and Methods

Ethical approval

This cross-sectional study received ethical approval from the Ethics Committee for Research on Humans at Chiangrai Prachanukroh Hospital (approval number: CR 0033.102/EC.66-451) before the commencement of data collection. The study was conducted at Chiangrai Prachanukroh Hospital, Chiang Rai, Thailand, with participant recruitment between July 2023 and May 2024. All participants were provided with comprehensive information on the study objectives and procedures and their rights as research participants. Written informed consent was obtained from all participants before enrollment, in accordance with ethical guidelines.

Participants

For the HCV infection group, patients were recruited from the Gastrointestinal and Liver Center at Chiangrai Prachanukroh Hospital. The inclusion criteria were (1) patients with anti-HCV antibodies detected using a chemiluminescent microparticle immunoassay (ARCHITECT anti-HCV assay; Abbott Laboratories, North Chicago, IL, USA) and (2) patients with serum HCV-RNA confirmed by reverse transcription-polymerase chain reaction (amplification test; Abbott Laboratories, North Chicago, IL, USA). Patients with antibodies against other hepatitis viruses, those with autoimmune mucocutaneous diseases, those taking medications known to cause lichenoid drug reactions[34], and those who declined biopsy when indicated were excluded.

For the control group, participants were recruited from the Health Checkup Center at Chiangrai Prachanukroh Hospital. Patients aged at least 30 years were included. The exclusion criteria were (1) patients with a history of hepatitis virus infection and (2) patients with a history of using drugs reported to cause lichenoid drug

reactions.[34] HCV infection in this group was excluded based on medical history only, as serologic testing was not performed; therefore, the possibility of unrecognized HCV infection cannot be entirely ruled out.

Sample size calculation

The sample size was calculated using a two-proportion formula, with a two-sided significance level of 0.05 and a statistical power of 0.80. Based on a previous study[16], the estimated prevalence of OLP was 4.76% in patients with HCV infection and 0.67% in control patients. A control-to-case ratio of 7.13 was applied. Consequently, 126 patients with HCV infection and 899 without HCV were included in the study.

Data collection

Demographic data (including age and sex) and clinical data (including chief complaint, present illness, medical and dental history, and medication history) were collected from all patients. The clinical characteristics of OLP or OPMDs, including type, location, distribution, and photographs, were recorded.

Clinical assessment and definitive diagnosis

Oral examinations were performed by a single examiner (CA), a resident in general dentistry at Chiangrai Prachanukroh Hospital. The screening protocol for OLP and OPMDs was standardized through calibration sessions in collaboration with a Thai board-certified oral medicine (KI). This process achieved inter-examiner and intra-examiner reliability, with kappa values of 0.84 and 1.0, respectively.

Patients with clinical features of OLP or OPMDs were referred for confirmatory histopathological examination. Incisional biopsies were performed at the representative sites by an oral medicine specialist (DM). The tissue specimens were fixed in 10% neutral-buffered formalin and submitted for histopathological examination at the Faculty of Dentistry, Chiang Mai University. Histopathological diagnoses were performed by Thai board-certified oral and maxillofacial pathologists. The diagnosis of OLP was based on the clinical and histopathological criteria established by van der Meij and van der Waal in 2003.[7] Only cases that fulfilled the strict diagnostic criteria for OLP were included. Lesions merely ‘compatible with OLP’ or with overlapping lichenoid features were not classified as OLP.

Statistical analysis

The statistical analyses were performed using SPSS (version 26.0; IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize demographic and clinical data. Categorical variables were presented as frequencies and percentages, and continuous variables were reported as means with standard deviations or medians, depending on data distribution. To compare the prevalence of OLP between the two groups, Pearson’s Chi-square test and Fisher’s exact test were applied, with the significance level set at $p<0.05$.

Results

Demographic data

A total of 1,025 participants were enrolled, comprising 126 patients with HCV infection and 899 without HCV. The demographic data for both groups are summarized in Table 1. Significant differences in mean age and sex were observed between the groups. Among the patients with HCV infection, 71 were receiving sofosbuvir or velpatasvir (SOF/VEL), with or without ribavirin (RBV), and 55 had not initiated therapy at the time of the oral examination. There were statistically significant differences in age ($p<0.001$) and sex distribution ($p=0.005$) between groups. HCV-positive participants were older and included a higher proportion of males. These demographic differences may influence OLP risk and were therefore considered potential confounding factors in the interpretation of results.

Significantly higher prevalence of OLP in patients with HCV infection

Thirteen participants with clinically suspected OLP or OPMDs underwent biopsy, including four with HCV infection and nine without HCV. Definitive diagnoses included OLP, lichenoid reaction, discoid lupus erythematosus, and premalignant epithelial dysplasia, as shown in Table 2. The prevalence of OLP was 2.38% (95% CI: 0.49-6.80%) in HCV-positive patients and 0.33% (95% CI: 0.07–0.96%) in HCV-negative patients, representing a statistically significant difference between groups ($p=0.027$).

Clinical characteristics of OLP in patients with and without HCV infection

In patients with HCV, the three cases of OLP included one man (aged 70 years) and two women (aged 40 and 58 years). The mean age of these three patients was 56.0 years, and clinically, one case presented with the ulcerative type while the remaining two exhibited the atrophic type. Representative clinical features are shown in Figure 1. One patient had been receiving SOF/VEL plus RBV for one month at the time of the oral examination and reported a burning sensation before treatment. The remaining two patients had not started antiviral therapy. No significant difference in the prevalence of OLP was observed between patients who received SOF/VEL and those who did not (p=0.58).

In patients without HCV, the three cases of OLP comprised two men (aged 52 and 65 years) and one woman (aged 54 years). Their mean age was 57.0 years, and all three cases presented with the atrophic clinical type. Representative clinical features are shown in Figures 2A-2C. Additionally, one case of a lichenoid reaction associated with amalgam restoration is presented in Figure 2D. Clinical characteristics of OLP lesions in both groups are summarized in Table 3.

Discussion

Multiple studies have investigated the association between OLP and HCV infection, but the evidence has remained variable across populations. While several reports have demonstrated a meaningful relationship between the two conditions[10-22], others have not shown a consistent association.[23-32] These inconsistencies may reflect regional differences in host genetics, immune responses, or circulating HCV genotypes.[22] Our study provides new data from northern Thailand and demonstrates a significantly higher prevalence of OLP among patients with HCV infection compared with those without HCV. The prevalence observed in our cohort (2.38%) is consistent within the range reported internationally (1.5-4.94%).[14,16,26,29,35-38] Moreover, the prevalence of OLP in the non-HCV group (0.33%) is also comparable to previously documented rates in control populations.[16,26,29,35-37] After considering the significant differences in age and sex distribution between the two

Table 1: Demographic data in patients with and without hepatitis C virus infection.

	HCV-positive (n=126)	HCV negative (n=899)	*p*-value
Sex (n, %)			0.005*a
Male	74 (58.73)	407 (45.27)	
Female	52 (41.27)	492 (54.73)	
Age (years)			
Mean±SD	55.89±11.69	51.23±12.11	<0.001*b
Median	57	52	
Range	20-84	30-86	

HCV = hepatitis C virus, n = number of patients, SD = standard deviation
*p<0.05
[a]Chi-square test
[b]Independent-samples t-test

Table 2: Definitive diagnoses of oral lesions in patients with and without hepatitis C virus infection.

	HCV-positive (%) (n=126)	HCV negative (%) (n=899)	*p*-value[a]
Oral lichen planus	3 (2.38)	3 (0.33)	0.027*
Lichenoid reaction	0 (0)	1 (0.11)	1.000
Discoid lupus erythematosus	1 (0.79)	3 (0.33)	0.409
Premalignant epithelial dysplasia	0 (0)	2 (0.22)	1.000
Total	4 (3.17)	9 (1.00)	

HCV = hepatitis C virus, n = number of patients
* p<0.05
a Fisher's exact test

Table 3: Clinical characteristics and antiviral treatment of oral lichen planus in patients with and without hepatitis C virus infection.

	HCV-positive (%) (n=3)	HCV negative (%) (n=3)
Clinical type		
Ulcerative	1 (33.33)	0 (0)
Atrophic	2 (66.67)	3 (100)
Symptoms		
Symptomatic	3 (100)	2 (66.67)
Asymptomatic	0 (0)	1 (33.33)
Distribution		
Bilateral	3 (100)	3 (100)
Site		
Buccal mucosa	3 (100)	3 (100)
Gingiva	2 (66.67)	1 (33.33)
Lip vermilion	1 (33.33)	0 (0)
Multiple sites	3 (100)	3 (100)
SOF/VEL with or without RBV		
Yes	1 (33.33)	-
No	2 (66.67)	-
Sex		
Male	1 (33.33)	2 (66.67)
Female	2 (66.67)	1 (33.33)
Age (years)		
Mean±SD	56±15.10	57±7.00

HCV = hepatitis C virus, n = number of patients

groups, the association observed in this study remains consistent with prior evidence.[16] Because the HCV-positive group was older and more predominantly male[39], these demographic factors may have contributed to the higher prevalence of OLP. Age is known to influence the occurrence of OLP, and sex-related differences in immune responses may also play a role[5], therefore, demographic imbalance must be considered when interpreting our findings.

In Thailand, two studies have examined the prevalence of HCV infection among patients with OLP. Klanrit *et al*.,[19] reported that 8.3% of patients with OLP in the central and northeastern regions were seropositive for HCV. Similarly, Manomaivat *et al*.,[22] found that 8.9% of OLP patients in the upper northern region tested positive for anti-HCV antibodies, compared to 1% in the control group. These findings, along with ours, provide bidirectional evidence supporting a potential association between HCV infection and OLP in the Thai population.

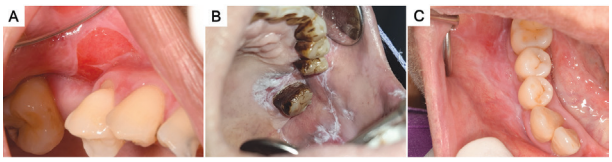

Figure 1: Clinical features of oral lichen planus (OLP) in patients with hepatitis C virus (HCV) infection. (A) Ulcerative OLP of the upper labial mucosa in a 40-year-old woman receiving sofosbuvir/velpatasvir with ribavirin; (B) Atrophic OLP involving posterior maxillary gingiva and buccal mucosa in a 70-year-old man; (C) Bilateral atrophic OLP on buccal mucosa in a 58-year-old woman.

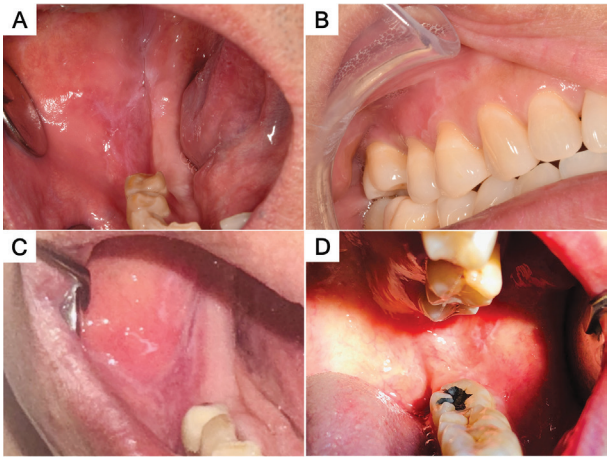

Figure 2: OLP and lichenoid reaction in HCV-negative patients. (A–C) Atrophic-type OLP: (A) 52-year-old man, buccal mucosa; (B) 54-year-old woman, upper labial gingiva; (C) 65-year-old man, buccal mucosa; (D) Oral lichenoid contact reaction adjacent to an amalgam restoration on the left buccal mucosa in a 55-year-old woman, with localized erythema and white striae.

The prevalence identified in this study aligns with international reports and underscores the relevance of evaluating OLP in patients with HCV infection in Thailand's northern region.[33]

Several mechanisms have been proposed to explain this association, including immune-mediated cytotoxicity directed against basal keratinocytes, molecular mimicry between viral antigens and epithelial structures, and T-cell–driven chronic inflammation.[40,41] Possible replication of HCV in extrahepatic tissues, including oral epithelial cells, may also contribute to mucosal immune dysregulation.[42,43] These mechanisms may help clarify the limited proportion of individuals with HCV who develop OLP. In addition to these immunopathogenic pathways, the severity and chronicity of HCV infection may further influence the clinical expression of OLP. Long-standing HCV infection is associated with persistent immune activation and elevated systemic inflammatory burden, factors that may predispose patients to more symptomatic or recalcitrant mucosal disease.

Direct-acting antivirals (DAAs) such as SOF/VEL, with or without RBV, are highly effective in achieving a sustained virologic response in patients with chronic HCV infection, with response rates exceeding 97% across all genotypes.[44] In addition to viral clearance, several studies have reported improvement in HCV-related extrahepatic manifestations, including OLP, following DAA therapy, with significant symptom improvement observed during or after treatment.[45,46] However, in our study, one patient who received SOF/VEL with RBV continued to experience symptomatic OLP despite achieving a virologic cure. This observation highlights the potential for persistent OLP symptoms following HCV eradication and indicates the need for further investigation into the mechanisms and timeline of extrahepatic response to antiviral therapy.

Since 2005, the World Health Organization has classified OLP as an OPMD, with a malignant transformation rate of 1.43%.[8] The highest transformation rates are observed in the erosive (1.7%) and atrophic (1.3%) types.[47] Gandolfo *et al.*,[48] demonstrated that concurrent HCV infection and OLP increased the risk of oral cancer, with a risk ratio of 3.16. Previous studies have also reported an association between erosive OLP and HCV infection.[11,49] In our study, one of three HCV-related OLP cases was ulcerative, and two were atrophic. These findings suggest that HCV infection may be associated with more aggressive OLP types, which carry a higher risk of malignant transformation. Therefore, close monitoring of OLP in patients with HCV infection is crucial for the early detection and prevention of progression to oral cancer.

This study has several limitations. First, the single-center and cross-sectional design limits the ability to infer causality. Second, the small number of OLP cases restricts the statistical power to detect subgroup differences. Third, demographic imbalance between groups may have influenced the results. Fourth, HCV infection in the control group was excluded based on medical history without serologic testing, raising the possibility of unrecognized HCV infection. Finally, although strict diagnostic criteria were applied, some degree of overlap between lichenoid lesions and true OLP cannot be entirely ruled out. To enhance generalizability, multicenter and population-based studies across various regions of Thailand are recommended. Additionally, longitudinal research is needed to evaluate the efficacy of SOF/VEL in resolving OLP lesions and its potential impact on the risk of malignant transformation in HCV-related OLP. Further investigation into the pathogenesis of OLP in HCV infection, including the underlying molecular and immunological mechanisms, is also warranted. Finally, assessing the cost-effectiveness of incorporating OLP screening into HCV treatment protocols could inform clinical practice and health policy.

Conclusions

Our findings indicate a significant association between HCV infection and OLP. Therefore, screening for OLP in patients with HCV could be recommended for early detection and appropriate management.

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Conflict of Interest

The authors declare no conflict of interest.

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