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Original article

Periodontal status in patients with chronic skin diseases: a pilot study

Hao Nguyen[a],Nguyen Pham[a], Thuy Vo[b], Huong Duong[b], Hoa Ho[b], Nhi Pham[c], Nam Huynh[b], Thuy Nguyen [b, *]

[a] *Pham Ngoc Thach University of Medicine, Ho Chi Minh City, Viet Nam*
[b] *Faculty of Dentistry, University of Medicine and Pharmacy at Ho Chi Minh City, Ho Chi Minh City, Vietnam*
[c] *Ho Chi City Hospital of Dermato-Venereology, Ho Chi Minh City, Viet Nam*

Abstract

Background: Many skin diseases, especially those that also involve oral mucosa lesions, might play an essential role in the etiopathogenesis of oral health and mucocutaneous inflammation. The presence of these oral lesions may make it challenging to maintain satisfactory oral hygiene, which increases plaque accumulation and the risk of developing periodontal disease.
Objectives: This study evaluated and compared the periodontal status of patients with chronic skin disease (CSD) with that of healthy controls.
Methods: This study included 109 patients and 37 healthy controls. The evaluated parameters included the bleeding on probing index (BOP), periodontal pocket depths (PPD), clinical attachment level (CAL), simplified debris index (DI), simplified calculus index (CI), and the presence of oral lesions. Clinical parameters were measured and compared between the two groups using the chi-square test, *t*-test, and a non-parametric test.
Results: Patients with CSD had significantly higher BOP and a lower percentage of teeth with CAL d" 3 mm compared with those in the control group. Furthermore, CAL > 6 mm was only presented in the group of patients with CSD. There were no significant differences in the PPD, PI, and CI parameters and oral lesions between the two groups.
Conclusion: This pilot study revealed that the periodontal status of patients with CSD was worse than that of healthy controls. These results suggest that patients with CSD appear to be more at risk for the development and progression of periodontal diseases.

Keywords: Chronic skin disease; oral lesions; periodontal disease; periodontal status.

Maintaining satisfactory oral health is crucial, as it can influence a person's overall health and quality of life. Individuals with inflammatory dermatological conditions often experience oral discomfort, dry mouth, and an increased risk of developing periodontal disease more frequently than those without such conditions. In addition, certain medications used to treat these conditions may have adverse effects on oral health.[1] Periodontitis is an infection caused by an imbalance in the body's defense against bacterial invasion, which results in the resorption of alveolar bone and the destruction of periodontal tissue. If this is not treated appropriately, prolonged periodontal infection can lead to tooth loss, thereby affecting masticatory function, pronunciation, and aesthetics. Periodontitis is a multifactorial disease that has various etiological factors. There is evidence that some systemic illnesses can increase an individual's risk of developing periodontitis. Acantholysis and uncomfortable oral ulceration are common outcomes of chronic skin diseases (CSDs) that affect the skin and oral mucosa. Moreover, it has been hypothesized that these oral lesions increase the possibility of developing periodontal disease.[2-9]

*Correspondence to: Thuy Nguyen, Faculty of Dentistry, University of Medicine and Pharmacy at Ho Chi Minh City, Vietnam.
E-mail: ntthuy@ump.edu.vn
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The potential influence of CSDs on periodontitis might be based on direct or indirect relationships. Indirect effects could include inadequate dental hygiene practices and fewer scaling appointments with the dentist, which would allow the buildup of plaque and thus contribute to periodontitis. Another explanation is a decreased immune response to periodontal infections as a result of immunosuppressive therapy. Based on the potential shared pathogenetic processes, direct effects are also reasonable.[5] Furthermore, autoimmune-inflammatory mechanisms are a critical part of periodontitis and CSDs.

However, previous studies have shown that various oral diseases, especially periodontitis, might influence the development or prognosis of several CSDs, including aphthous stomatitis, atopic dermatitis, lichen planus, pemphigus, pemphigoid, and psoriasis.[10-12] Deep diseased periodontal pockets are an ideal environment for anaerobe colonization, which includes many pathogenic organisms.[13] In addition, substantial levels of inflammatory mediators, including tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1, IL-2, and IL-8, and prostaglandins (PGEs), can be released into the circulation from the diseased periodontium and may contribute to systemic inflammation.[14]

Several studies have been conducted to detect the relationship between periodontitis and CSDs; however, no definitive conclusions have been established. Previous studies have reported that no significant differences were noted in periodontal parameters between control and case groups for mucous membrane pemphigoid (MMP), oral lichen planus, or desquamative gingivitis.[4-7, 9] Meanwhile, it has been reported that patients affected by pemphigus vulgaris (PV)[2, 8] or MMP[3] exhibit a diminished periodontal status.

This study aimed to investigate and compare the periodontal status and treatment needs between healthy controls and patients with CSD who were treated at Ho Chi Minh City Hospital of Dermato-Venereology in February 2023.

Materials and methods

Study design

This prospective case-control study was performed at Ho Chi Minh City Hospital of Dermato-Venereology in February 2023 to assess the periodontal status of patients with CSD. The study included 109 patients with CSD, including psoriasis, atopic dermatitis, lupus, and common autoimmune bullous skin diseases, and 37 healthy controls. This study was reviewed and approved by the Institutional Review Board of Ho Chi Minh City Hospital of Dermato-Venereology, Vietnam (no. 107/CN-BVDL, January 18, 2023), and it was conducted in accordance with the 1975 Helsinki Declaration as revised in 2000. All participants provided written consent after being fully informed of the study procedures, risks, and benefits.

Eligibility criteria

Patients with CSDs (psoriasis, atopic dermatitis, autoimmune bullous diseases, or systemic lupus erythematosus) who were diagnosed by dermatologists with more than 5 years of experience and supportive laboratory tests, aged 18 years or older, and willing to participate in the study were included. The control group included healthy individuals aged 18 years or older and free of any CSDs. These individuals were willing to participate in the study and would be used as a comparison group for the case group.

Clinical parameter collection

Information related to the patients' epidemiological characteristics and chronic inflammatory skin conditions was recorded from medical records, and their oral health status was collected from clinical examinations performed by postgraduate doctors and lecturers of the Faculty of Dentistry, University of Medicine and Pharmacy at Ho Chi Minh City. The investigators were trained by a specialist in public dentistry and had an intra- and inter-rater reliability of ≥ 0.8.

Oral clinical measurements

Periodontal parameters were measured as per the standard guidelines.[15] The following oral clinical parameters were evaluated:

1) Full mouth bleeding on probing scores was recorded and considered positive when bleeding occurred within 20 s after the probe was removed after application with light pressure. The bleeding percentage was calculated for each patient by dividing the total number of positive bleeding sites by the total number of probed sites. Patients with a bleeding on probing index (BOP) index $\geq 10.0\%$ are considered in need of periodontal treatment regardless of the presence of periodontal pockets or clinical attachment

loss, with intact or impaired periodontal tissue.[16-18]; 2) pocket probing depth (PPD) was measured from the gingival margin to the base of the gingival sulcus/ periodontal pocket with the aid of a 15-mm University of North Carolina (UNC) periodontal probe; 3) the clinical attachment level (CAL) was measured from the cemento-enamel junction (CEJ) to the base of the gingival sulcus/periodontal pocket using a UNC periodontal probe. The CAL was considered zero if the attachment was at the level of the CEJ. However, if the free gingival margin was coronal to the CEJ, the CAL was determined by measuring the PPD and subtracting the distance from the CEJ to the free gingival margin. When the free gingival margin was apical to the CEJ, the CAL was determined by measuring the distance from the CEJ to the free gingival margin and adding it to the measured PPD. Furthermore, the 4) debris index (DI); and 5) calculus index (CI) were evaluated by green and vermillion as suggested by the Simplified Oral Hygiene Index (OHI-S) (1964).[19] 6) the number of oral lesions was counted.

Probed sites were classified into three categories based on the PPD and CAL (≤ 3 mm, 4–5 mm, and > 6 mm). The investigator examined one tooth (index teeth) from each sextant, including the incisor and the left and right molar regions of the maxillary and mandibular dental arches, to record the CAL, DI, and CI.

Statistical analysis

All statistical analysis was performed using JASP software, version 0.17.2.1 (University of Amsterdam, Nieuwe Achtergracht 129B, Amsterdam, The Netherlands). Data were presented as the mean ± standard deviation (SD) or the median and interquartile range (IQR). The normality of the data distribution was verified using the Shapiro-Wilk test. For variables that exhibited normal distribution, an independent *t*-test was used to detect statistically significant differences between the two groups. For variables that were not normally distributed, the Mann-Whitney U test was used to compare the two groups. $P < 0.05$ was considered statistically significant.

Results

A total of 146 participants **(Table 1)** were enrolled in this study, including 109 patients in the disease group (D) and 37 healthy participants in the control group (C). **Table 2** shows the frequency of CSDs.

Table 1. Demographic features of participants in the disease and control groups.

	Disease	**Control**	***P***
Number	109	37	
Male (%)	46 (42.2%)	9 (24.3%)	0.052492(Chi-square test)
Female (%)	63 (57.8%)	28 (75.7%)	
Mean age ± SD	44.8 ± 15.2	47.0 ± 16.0	
Median age [IQR]	43 (34–58)	47 (39–54)	0.059 (Mann-Whitney U test)

Table 2. Frequencies for CSDs.

CSDs	**Frequency**	**(%)**	**Valid (%)**	**Cumulative (%)**
Pemphigus	17	11.6	15.6	15.6
Mixed connective tissue disease	17	11.6	15.6	31.2
Atopic dermatitis	7	4.8	6.4	37.6
Psoriasis	64	43.8	58.7	96.3
Erythroderma	4	2.7	3.7	100
Disease-free (control)	37	25.3		
Total	**146**	**100**		

Table 3. Periodontal parameters in the disease **(D)**; and control **(C)** groups.

Parameter	Group	Mean	Median	IQR	*P**
BOP (% sites)	D	29.5	21.9%	0.0% – 50.0%	< 0.001
	C	9.4	0	0.0% – 10.0%	
PPD 0 - 3 mm	D	92	100	100.0% – 100.0%	0.48
(% sites)	C	96	100	100.0% – 100.0%	
PPD 4 - 5 mm	D	5	0	0 – 0	**NA**
(% sites)	C	4	0	0 – 0	
CAL 0 - 3 mm	D	82	100	80.0% – 100.0%	0.02
(% sites)	C	96	100	100.0% – 100.0%	
CAL 4 - 5 mm	D	13	0	0.0% – 17.0%	**NA**
(% sites)	C	4	0	0	
CAL > 6 mm	D	1.6	0	0	**NA**
(% sites)	C	0	0	0	
Oral lesions	D	0.2	0	0	**NA**
(number/person)	C	0.1	0	0	

*Mann-Whitney U test. BOP, bleeding on probing scores; PPD, pocket probing depth; CAL, clinical attachment level.

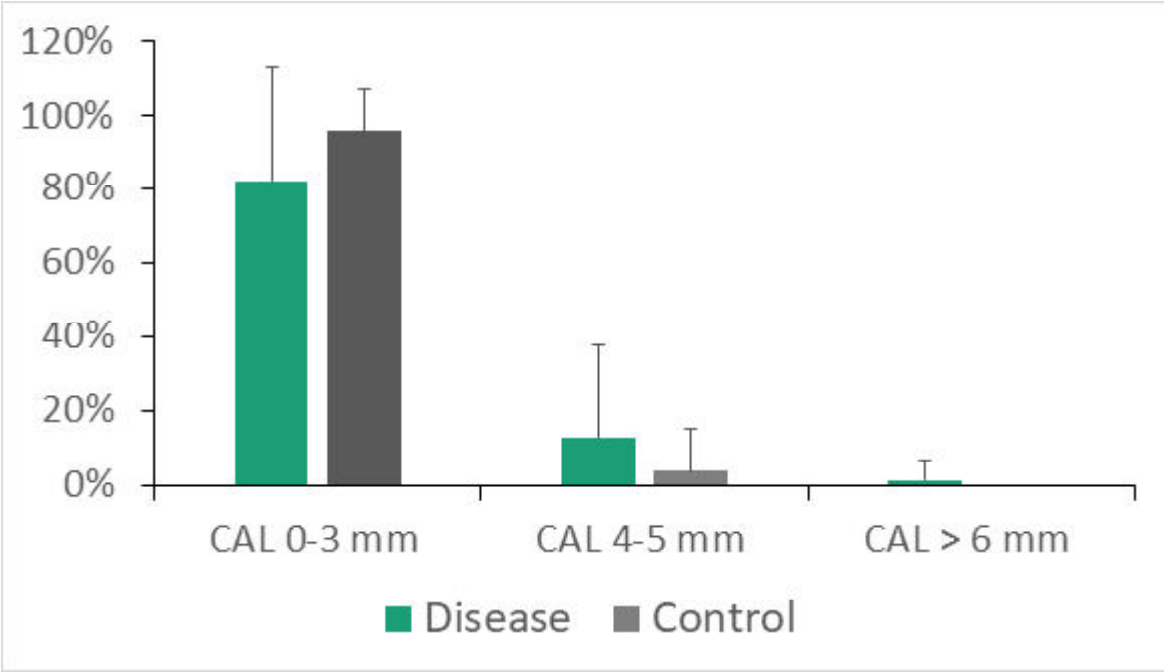

Figure 1. The distribution of CAL in the two groups. CAL, clinical attachment level.

Periodontal status

The periodontal data analysis is detailed in **Table 3.** There was a significant difference in BOP score between the two groups **(Table 3)**, and the percentage of bleeding sites in the disease group was 30.0%, which is significantly higher than that of the control group (9.0%). Similarly, the prevalence of sextants with CAL ≤ 3 mm was significantly lower in the disease group when compared to that of the control group, with ratios of 82.0% and 96.0%, respectively. Moreover, there were no sextants with CAL > 6 mm in the control group, while there were 9 cases thereof in the disease group. In addition, the CAL 4–5 mm accounted for 13.0% in the disease group, whereas that of the control group was 4.0%; the difference was not significant **(Figure 1).** The results did not reveal any significant differences in the PPD score between the two groups **(Table 3).** The percentage of sites with PPD ≤ 3 mm was 92.0% and 96.0% in the disease and control groups, respectively. **Figure 2** shows that there were no significant differences in the DI and CI between the two groups. Although the percentage of individuals with a DI of 0 (no plaque) in the disease group was lower than that of the control group, 21.0% and 31.0%, respectively, the difference was not significant ($P = 0.08$).

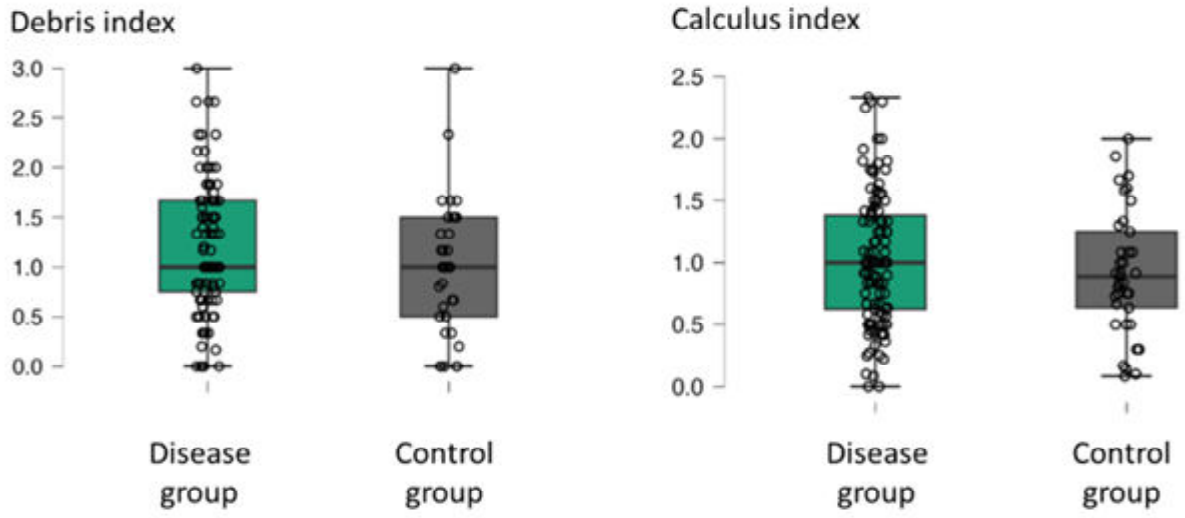

Figure 2. The debris index and calculus index in the two groups were presented by box plots (median and IQR).

Need for periodontal treatment

The percentage of patients that required periodontal disease treatment (BOP ≥ 10.0%) was 62.3% in the disease group, which was significantly higher than that of the control group (29.7%) ($P < 0.001$).

Oral lesion number

The mean number of oral lesions in the disease group was 0.2/person, which was higher than that of the control group (0.1/person); however, the difference was not significant.
(Table 3).

Discussion

This is the first large-cohort study to report the periodontal status of patients with CSD compared with age- and gender-matched controls. Our case-control study revealed that patients with CSD had significantly higher BOP than the controls. In addition, the frequency distribution of the number of teeth with CAL ≤ 3 mm was significantly lower in the cases than that of the controls. The data presented is consistent with the concept that the periodontal status of patients with CSD is worse than that of controls. Previous evidence supporting our findings is scarce, as only a few studies have described the gingival status of patients with gingival MMP.[5, 7, 9] Moreover, the presence of CAL > 6 mm only in patients with CSD suggests that CSD could increase the risk for periodontal tissue breakdown. This potential influence may be due to the immunosuppressive treatment-associated reduction in the patient's immune response to periodontal pathogens. Another possibility is the shared pathogenic mechanisms between autoantibody-induced and bacterial-elicited inflammatory tissue damage.

This study failed to detect significant differences in PPD, despite the differences in CAL between the two groups. This may be attributed to differences in the gingival recession between cases and controls, which were not evaluated in this study. Indeed, Lo Russo L, *et al.*[4, 5] reported that the gingival recession of patients with MMP was significantly greater than that of controls. The presence of oral lesions because of CSDs, combined with the application of topical corticosteroids, may have contributed to mucosal tissue thinning and increased susceptibility to gingival recession. Previous studies have shown differences in PPD and CAL between controls and patients with PV.[4, 8] The higher PPD and CAL in patients with PV could be explained by the role of plaque (PV exhibited a high plaque score) and various inflammatory cytokines involved in the development of periodontitis.

Patients with a BOP index ≥ 10.0% are considered in need of periodontal treatment, regardless of the presence of periodontal pockets or CAL, with intact or impaired periodontal tissue.[16-18] The percentage of patients in need of periodontal treatment in the disease group was higher than that of the control group. This may be explained by the fact that patients with dermatological diseases have periodontal tissues that are more sensitive to pathogens; they may also have inadequate dental hygiene practices and less frequent dentist visits.[1] Regular dental examinations and adequate treatment play an important role in improving the quality of life of patients with dermatological diseases.

It is reasonable to consider that patients with CSD may have impaired capacity to perform effcient oral hygiene practices because of painful oral lesions. In addition, discomfort caused by gingival lesions may predispose patients to visit their dentists less regularly. A similar phenomenon has been reported for patients with PV (Akman A, *et al.*)[2]. Therefore, their plaque accumulation is more serious. Indeed, differences in the gingival and plaque index were reported in previous studies. [2, 4, 5, 8] However, in this study, there were no significant differences in the DI and CI between the two groups. Moreover, the percentage of individuals with a DI of 0 (no debris) in the disease group was lower than that of the control group. A possible explanation for this result is that more than half (58.7%) of patients with CSD in the aforementioned studies had psoriasis, not MMP or PV. To the best of our knowledge, no study has reported the periodontal status in patients with psoriasis. Psoriasis is characterized by the presence of keratinized tissue rather than erythematous lesions, blisters, erosions, and ulcers, which are typical symptoms of pemphigoid diseases. The fact that keratinized tissues do not impede proper oral hygiene may be a reason for the relatively low plaque index in the disease group (1.1 in the disease group vs. 1.0 in the control group).

Similarly, the number of skin lesions did not differ between the two groups. This might be because oral lesions were not always present, and some patients were already receiving treatment at the time of evaluation. The heterogeneous treatment time and treatment therapy are limitations of this study **(Figure 3)**.

This single-center case-control pilot study has several limitations. The unequal group sizes and convenient nature signify that the study was not sufficiently powered for detailed subgroup analyses across heterogeneous CSD diagnoses, thereby increasing the risk of type II errors. In addition, several potential confounders were not collected (e.g., oral hygiene practices, smoking, education, and systemic conditions); therefore, residual confounding factors cannot be excluded. Treatment exposure was heterogeneous (varying treatment timing/therapies), and some participants were already receiving treatment, thus complicating inference about temporality and therapy effects. Collectively, these factors indicate that the findings should be interpreted as hypothesis-generating and warrant confirmation in larger, multi-center, longitudinal studies with standardized periodontal and dermatologic assessments. Further expanded analyses, including regression and mixed-effect models, could be conducted in a larger-scale investigation of dermatology and periodontology in the Vietnamese population, examining other factors that contribute to periodontal disease.

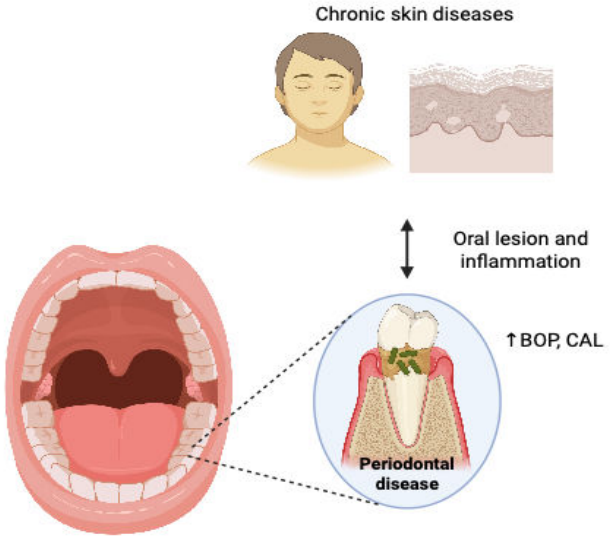

Figure 3. Concept of the relation between chronic skin diseases and periodontal disease.

Conclusion

This pilot study demonstrated that patients with CSD exhibited significantly poorer periodontal status than healthy controls, which was marked by elevated BOP and a higher prevalence of CAL. These findings suggest a potential association between mucocutaneous inflammatory conditions and periodontal tissue breakdown, possibly mediated by shared autoimmune-inflammatory mechanisms or immunosuppressive treatments. Although oral hygiene indices did not differ significantly, likely because of the high proportion of patients with psoriasis who have less oral discomfort, the greater need for periodontal treatment among patients with CSD highlights the importance of integrating dental evaluation into dermatologic care. These results emphasize the need for multidisciplinary collaboration and further large-scale research to clarify causal relationships and optimize oral-systemic health strategies for individuals with CSD.

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Conflicts of interest statement

The authors have each completed the International Committee of Medical Journal Editors Form for Uniform Disclosure of Potential Conflicts of Interest. The authors have no potential conflict of interest to disclose.

Data sharing statement

All data generated or analyzed during the present study are included in this published article. Further details are available for noncommercial purposes from the corresponding author on reasonable request.

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