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Original study article

Studying the relationship between oral lichen planus and periodontal disease: Value on periodontal pathogens and oral hygiene

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ABSTRACT

BACKGROUND: Oral lichen planus is a chronic inflammatory condition of the oral mucosa characterized by white, lacy lesions. The etiology of oral lichen planus is complex and remains unclear, but it is thought to be a T-cell mediated autoimmune disease. Several factors have been implicated in the pathogenesis of oral lichen planus, including genetic predisposition, dental materials, iatrogenic factors, infections, autoimmunity, and bowel disease. The microflora of the oral cavity plays an important role in maintaining oral health and preventing disease. However, its role in the development and progression of oral lichen planus is not yet totally understood. Some studies have shown that there are differences in the microflora of oral lichen planus patients compared to healthy controls. These differences may be related to the inflammatory process in oral lichen planus, or they may be a contributing factor to the disease.

AIM: Identify the prevalence of the detection of periodontopathogenic microorganisms in oral lichen planus patients and compare their prevalence in healthy non-oral lichen planus patients.

MATERIALS AND METHODS: A cross-sectional, single-center study was conducted. A total of 75 patients were recruited, 45 with oral lichen planus and 30 healthy controls. The groups were formed by simple random sampling. The diagnosis of oral lichen planus was confirmed histologically in the main group. The forms and localization of oral lichen planus were determined in the main group based on clinical examination. In the control group, patients were divided into two subgroups depending on the presence of chronic periodontitis or gingivitis. The prevalence of periodontal pathogens was assessed based on the analysis of culture results. Among the 48 bacteria isolated from the oral mucosa of the affected site in the main and control groups, we focused on the microbiota of only periodontal bacteria to study their role and assess their impact on disease progression.

RESULTS: Among the 45 patients with a clinical diagnosis of oral lichen planus, there were 10 (22.22%) men and 35 (77.78%) women, with a mean age of 55.3±13.4 years. The control group included 30 healthy volunteers, 8 (26.67%) men and 22 (73.33%) women, with a mean age of 54.8±12.7 years. In patients with oral lichen planus regardless of their periodontal status the percentage of seropositivity for *A. actinomycetemcomitans*, *V. parvula*, *P. gingivalis*, *T. denticola* is higher compared to their healthy counterparts with gingivitis and periodontitis.

CONCLUSION: The study found an increased frequency of detection of pathogenic microorganisms, such as *A. actinomycetemcomitans*, *V. parvula*, *P. gingivalis*, and *T. denticola*, in patients with oral lichen planus, regardless of the presence of periodontitis. These periodontopathogens may be associated with oral lichen planus. However, further studies are needed to clarify their role in the pathogenesis of the disease.

Keywords: oral lichen planus; microbiota; periodontitis; oral hygiene.

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Оригинальное исследование

Изучение взаимосвязи между красным плоским лишаём слизистой оболочки полости рта и заболеваниями пародонта: значение микробиоты пародонтальных патогенов и гигиены полости рта

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АННОТАЦИЯ

Обоснование. Красный плоский лишай слизистой оболочки полости рта — это хроническое воспалительное заболевание, характеризующееся образованием белых сетчатых поражений. Этиология заболевания сложна и до сих пор не изучена, но считается, что оно имеет опосредованную Т-клетками аутоиммунную природу. В патогенез красного плоского лишая слизистой оболочки полости рта вовлечены несколько факторов, включая генетическую предрасположенность, при этом триггером выступают стоматологические материалы, ятрогенные факторы, инфекции, соматические и аутоиммунные заболевания, болезни кишечника. Микрофлора полости рта играет важную роль в поддержании здоровья и профилактике заболеваний, тем не менее её роль в развитии и прогрессировании красного плоского лишая слизистой оболочки рта полностью не изучена, однако известно, что она отличается от микрофлоры здоровых лиц. Такие различия обусловлены усилением воспалительного процесса при красном плоском лишае или являются фактором, предшествующим развитию заболевания.

Цель исследования — оценить и сравнить распространённость пародонтопатогенных микроорганизмов в группах пациентов, страдающих (основная группа) и не страдающих (группа контроля) красным плоским лишаём слизистой оболочки полости рта.

Материалы и методы. Выполнено поперечное одноцентровое исследование в группах пациентов с красным плоским лишаём слизистой оболочки полости рта (n=45) и контроля (n=30), сформированных методом случайного отбора. Диагностика красного плоского лишая слизистой оболочки полости рта у пациентов основной группы была подтверждена гистологически. Форма и локализация высыпаний у пациентов основной группы определялись на основании их клинической оценки. В контрольной группе пациенты были разделены на 2 подгруппы в зависимости от наличия хронического пародонтита или гингивита. Распространённость пародонтальных патогенных микроорганизмов оценивалась по результатам культурального метода исследования. Из 48 выращенных бактерий, взятых с поражённых участков слизистой оболочки полости рта пациентов основной и контрольной групп, нами исследована микробиота (КОЕ) только пародонтальных бактерий.

Результаты. В основной группе пациентов независимо от наличия у них гингивита или пародонтита отмечается более высокая частота выявления патогенных микроорганизмов *A. actinomycetemcomitans*, *V. parvula*, *P. gingivalis*, *T. denticola*.

Заключение. Обладающие пародонтразрушающим воздействием микроорганизмы, такие как *A. actinomycetemcomitans*, *V. parvula*, *P. gingivalis* и *T. denticola*, могут быть ассоциированы с красным плоским лишаём слизистой оболочки полости рта, однако для уточнения их роли в патогенезе заболевания необходимы дополнительные исследования.

Ключевые слова: красный плоский лишай слизистой оболочки полости рта; микробиота; пародонтит; гигиена полости рта.

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BACKGROUND

Oral lichen planus (OLP) is a chronic inflammatory disease characterized by white lace-like eruptions [1].

The etiopathogenesis of OLP is unclear; however, it is found to be an autoimmune disease caused by a T-cell mechanism in which autocytotoxic CD8+ T lymphocytes cause apoptosis of basal cells of the oral mucosa [2].

An early event in the disease pathogenesis is keratinocyte antigen expression, or antigen unmasking, which may be a self-peptide or heat shock protein. Then, T lymphocytes (mainly CD8+, but also CD4+) migrate into the epithelium and are activated directly by antigen binding to the major histocompatibility complex, class 1 (MHC 1) on the keratinocyte or through activated CD4+ lymphocytes [2].

OLP is multifactorial. Cases of familial predisposition are rare. An association with HLA histocompatibility antigens (HLA-A3, A11, A26, A28, B3, B5, B7, B8, DR1, and DRW9) and a tendency to develop OLP when using dental materials were noted [3].

Various infections are associated with OLP, including candidiasis, which is debatable, and human papilloma virus, Epstein–Barr virus, human herpes virus type 6, human immunodeficiency virus, and hepatitis C virus (HCV) [4]. In OLP, HCV replication was observed in the epithelial cells of the mucous membrane in the affected areas and HCV-specific CD4 and CD8 lymphocytes in the subepithelial zone. This indicates that HCV-specific T lymphocytes may be involved in the pathogenesis of OLP [5, 6].

Gram-negative anaerobic bacilli and spirochetes are assumed to be associated with the disease; however, this information has not been confirmed [7]. The hypothesis was based on outdated data from the 1930s and 1940s, obtained using imperfect methods for diagnosing OLP and identifying bacterial agents. Additionally, these data did not consider the risk of secondary infection or contamination of samples.

Moreover, *Helicobacter pylori* [8] and several microorganisms that cause periodontopathies [9] are associated with OLP.

As mentioned earlier, the association between OLP and viruses has been established; however, the role of bacteria in the etiology of OLP remains unclear. Furthermore, OLP is associated with periodontal diseases [10].

Oral microflora is crucial in maintaining oral health and preventing disease; however, the role of microflora in OLP development and progression is not fully understood. Some studies have indicated differences in the microflora of OLP patients and healthy controls [11]. Several oral diseases, particularly gingivitis and periodontitis, have been associated with changes in oral microbiota composition; however, the order of disease development, whether OLP causes periodontitis or vice versa, remains unknown.

The association between OLP and periodontitis is complex and multifaceted. It is hypothesized that gingival lesions in OLP may indirectly contribute to the development of periodontitis caused by dental plaque. This process may be because similar symptoms hinder the maintenance of proper oral hygiene, which increases the risk of periodontal tissue destruction [9]. However, OLP cannot directly deteriorate periodontal condition. It is more probable that the factors that aggravate the clinical manifestations of OLP and contribute to the development of periodontitis in these patients are dental plaque and tartar, which often accumulate due to poor oral hygiene [9].

Thus, theoretically, although OLP can contribute to the development of periodontitis, the combination of OLP and poor oral hygiene often leads to a more severe course of the disease.

Given the complex interaction between OLP and periodontal disease and the potential influence of periodontal bacteria on OLP onset or progression, it is critical to investigate the prevalence of periodontal pathogens in patients with OLP compared with healthy controls.

This study evaluated the prevalence of periodontal pathogenic microorganisms in patients with OLP compared with a control group without OLP.

MATERIALS AND METHODS

Study design

A cross-sectional one-stage study.

Compliance criteria

Inclusion criteria were newly or previously established diagnosis of OLP, periodontal disease on probing, voluntary desire and availability of written informed consent of the patient to participate in the study, consent to the processing of personal data, patients of different sexes aged >18 years, nonintake of systemic antibacterial drugs for at least 30 days, and the use of topical agents for at least 3 days before sampling.

Noninclusion criteria were failure to meet inclusion criteria, severe concomitant pathology or other autoimmune diseases in the anamnesis, and patient's reluctance to participate in the study for any reason.

Exclusion criteria were the patient's desire to withdraw from the study; noncompliance by the patient with the regimen, and prescribed examination and treatment regimen.

Conditions

The study was conducted at V.A. Rakhmanov Clinic of Skin and Sexually Transmitted Diseases of the I.M. Sechenov First Moscow State Medical University and the I.I. Mechnikov Research Institute of Vaccines and Serums (Mechnikov RIVS), Center for Collective Use of the Mechnikov RIVS, with financial support from the project of the Russian Federation represented by the Ministry

of Education and Science of Russia (agreement no. 075-15-2021-676; July 28, 2021).

Study duration

The study was conducted from January 2022 to November 2023.

Methods of outcome registration

The study involved 75 patients who were randomly selected and divided into two groups. The main group included 45 patients with a histologically confirmed OLP diagnosis; chronic periodontitis was diagnosed in 18 patients, and 9 patients had gingivitis. The control group consisted of 30 patients without OLP, 22 of whom had chronic periodontitis diagnosed for the first time, and gingivitis was diagnosed in 8 patients. For a more detailed analysis, participants in both groups were further divided into two subgroups.

Samples were taken from the oral mucosa of all study participants using sterile probes (cotton swab), which were then placed in a nutrient transport medium (1 ml of sucrose–gelatin medium) and sent to the laboratory. The sample was examined using cultural and microbiological methods. For the cultivation of microorganisms, the nutrient media Schaedler agar + 5% sheep red blood cells + vancomycin + neomycin (to exclude contaminated microflora) were used for *Aggregatibacter actinomycetemcomitans*, *Veillonella parvula*, *Porphyromonas gingivalis*, and *Treponema denticola*.

After the material arrived at the laboratory, the samples were processed and inoculated (a swab in 1 ml of sucrose–gelatin medium) according to the following scheme. Each sample was shaken using a shaker for 30 s, inoculated on nutrient media, and incubated under anaerobic conditions for 1–2 days at a temperature of 37°C. After incubation, the inoculations were examined for anaerobic bacteria growth; if the growth of anaerobic bacteria was not detected, the incubation was extended to 3–4 days. Then, pure cultures of microorganisms were obtained by reinoculation.

The identification of pure cultures of microorganisms was performed using MALDI-TOF mass spectrometry (time-of-flight mass spectrometry with matrix-associated laser desorption/ionization) on a MALDI Biotyper Sirius RUO System tool (Bruker, Germany). As such, one isolated colony of a fresh pure culture of the microorganism was applied with a disposable microbiological loop onto the target well of a special plate (MSP chip). Immediately after the biomass dried, the targets were treated with 1–2 μL of 70% formic acid to extract microbial proteins. Afterward, 1–2 μl of a matrix (alpha-cyano-4-hydroxycinnamic acid in an aqueous solution of acetonitrile and trifluoroacetic acid) was applied to the targets to ionize microbial peptides. Furthermore, the plate was placed in the device, and mass spectrometry was performed. The identification result was considered valid if the coefficient of correspondence with the database (score) was ≥2.0.

Ethical considerations

The study was approved by the local ethics committee of Sechenov University (protocol no. 01-22; January, 20, 2022). Signed voluntary informed consent to participate in the study was obtained from all the study patients. Patients were fully informed about the study, courses of therapy, possible outcomes, and side effects of the therapy.

Statistical analysis

The sample size was not previously calculated.

For a statistical description of quantitative indicators, the mean value, standard deviation, and median were calculated. To analyze the study results, the statistical software package SPSS version 26 (IBM, USA) was used. Quantitative data are presented in numerical format. The normality of the distribution of quantitative data values was tested using the Shapiro–Wilk test. Student's *t*-test was used to compare the mean values of the two independent groups.

RESULTS

Subjects (participants) of the study

Overall, 75 patients were included in the study; 45 of them had a clinical diagnosis of OLP (10 [22.22%] men and 35 [77.78%] women; average age, 55.3 ± 13.4 years) and made up the main group, and 30 volunteers (8 [26.67%] men and 22 [73.33%] women; mean age, 54. 8 ± 12.7 years) were included in the control group (Table 1).

In the main (n = 45) and comparison (n = 30) groups, no statistically significant differences were noted in the distribution of men and women (22.22% versus 26.67%, $p > 0.05$), average age (55.3 ± 13.4 vs. 54.8 ± 12.7 years, $p > 0.05$), and smoking history (17.3 ± 3 vs. 19.1 ± 2.4 years, $p > 0.05$). Thus, the results obtained indicate the absence of statistically significant differences between the study groups, which ensures comparability of the main demographic characteristics.

Regarding disease symptoms, the median pain score on the visual analog scale was five points (Q_1–Q_3: 2–8), indicating the presence of moderate manifestations of the disease in most patients.

The disease duration in patients of the main group was 3 years (Me, Q_1–Q_3: 1–5), i.e., in 50% of patients, OLP lasted from 1 to 5 years.

In 26.6% of cases, the provoking factor for developing OLP was the use of medications. Traumatization (Koebner's phenomenon) accounted for 8 (17.8%) cases, a history of stress was found in 5 (11.1%) cases, the presence of crowns made of different materials and galvanization was noted in 12 (26.6%) cases, and vaccination against COVID-19 was performed in 18 (40%) patients.

In the main group, a higher proportion of patients with a history of viral infections was noticeable. In particular, statistically significant differences were identified in

Table 1. Distribution of demographic and clinical characteristics in study groups
Таблица 1. Демографические и клинические характеристики пациентов исследуемых групп

Categories	Main group *n*=45 (%)	Control group *n*=30 (%)	*p*
Sex:			
• men	men	8 (26.67)	>0.05
• women	women	22 (73.33)	>0.05
Age, years, M ±	55.3±13.4	54.8±12.7	>0.05
Smoking, years, M ±	17.3±3	19.1±2.4	>0.05
VAS scale, Me (Q_1–Q_3)	5 (2.8)	-	-
OLP duration, Me (Q_1–Q_3)	3 (1.5)	-	-
Development of OLP during the intake of medications	12 (26.6)	-	-
Provoking factors:			
• traumatization and tooth extraction (Koebner's phenomenon)	8 (17.8)	-	-
• history of stress	5 (11.1)	-	-
• crowns and galvanization	12 (26.6)	-	-
• vaccination against COVID-19	18 (40)	-	-
History of viral infections:			
• cytomegalovirus	5 (11.1)	1 (3.3)	<0.05
• hepatitis C	12 (26.6)	4 (13.3)	<0.05
• Epstein–Barr	7 (15.5)	2 (6.6)	<0.05
Concomitant periodontal disease:			
• gingivitis	35 (77.7)	8 (26.6)	>0.05
• periodontitis	10 (22.2)	22 (73.3)	>0.05

the cases of cytomegalovirus, hepatitis C, and Epstein–Barr virus compared with the control group, which may indicate a significant association of these viral infections with OLP in this group of patients. Additionally, an analysis of concomitant periodontal disease showed that the proportion of cases of gingivitis and periodontitis in both groups was comparable ($p > 0.05$); that is, in the main group, gingivitis was detected in 77.7% of patients and periodontitis in 22.2%; in the comparison group, these parameters were 26.6% and 73.3%, respectively.

Main research results

Thus, the present study established that in the main group patients with gingivitis, compared with individuals in the control group with gingivitis, the frequency of detection of pathogenic microorganisms such as *A. actinomycetemcomitans* prevails (67% in OLP patients vs. 43% in patients without OLP; $p < 0.05$), *V. parvula* (78% vs. 49%; $p < 0.05$), *P. gingivalis* (84% vs. 54%; $p < 0.05$), *and T. denticola* (89% vs. 57%, respectively; $p < 0.05$) (Table 2).

In OLP patients with periodontitis, in comparison with the control group also with periodontitis, the frequency of detection of the same pathogenic microorganisms dominates, namely, *A. actinomycetemcomitans* (78% vs. 22%; $p < 0.05$), *V. parvula* (84% vs. 16%; $p < 0.05$), *P. gingivalis* (91% vs. 9%; $p < 0.05$), *and T. denticola* (96% vs. 34%; $p < 0.05$) (Table 2).

DISCUSSION

In this study, a detailed analysis of oral microbiota and periodontal condition was performed in patients with OLP. According to the results, in patients with OLP, the number of periodontopathogenic bacteria significantly exceeds similar indicators in relatively healthy patients in the control group ($p < 0.05$). The identified differences in bacterial composition may contribute to the development and progression of periodontitis in patients with OLP.

Table 2. Prevalence of microbiota periodontogenic bacteria in subgroups of patients with oral lichen planus and control group
Таблица 2. Частота выявления микробиоты пародонтогенных бактерий у пациентов исследуемых групп

Microorganism	Detection frequency, %					
	M-G	C-G	*p*	M-P	C-P	*p*
A. actinomycetemcomitans	67	12.5	<0.05	67	32	<0.05
V. parvula	78	25		78	23	
P. gingivalis	89	12.5		89	59.5	
T. denticola	78	50		94.5	68.5	

Note. О-Г, subgroup of patients with CRPS accompanied by gingivitis; О-П, subgroup of patients with CRPS accompanied by periodontitis; К-Г, subgroup of control group patients with gingivitis; К-П, subgroup of control group patients with periodontitis; КПЛ СОПР, red squamous lichen planus of the oral mucosa.

The higher number of periodontal bacteria in OLP patients in the present study is consistent with that of some previous studies, indicating an association between OLP and periodontitis [7, 9]. However, to date, studies that analyzed the microbiological profile in OLP are few, in which the influence of various factors, such as oral hygiene, immune status, and drug therapy, on the composition of bacteria has not been sufficiently studied. Additionally, the patient samples in these studies were small, limiting the statistical significance and generalizability of the results [12]. Moreover, other studies did not reveal a significant difference in the composition of oral microbiota between OLP patients and a conditionally healthy cohort of control groups [10]. This discrepancy may be due to the different methods of collecting, processing, and analyzing samples and the applied criteria for diagnosing OLP and assessing periodontal condition. Additionally, oral microbiota is influenced by various factors, such as age, sex, diet, smoking, medication intake, and oral care, which may vary among different populations and studies [13].

Patients with OLP experience difficulty in general functioning, including eating and drinking, because of persistent pain caused by gingival ulcers. This discomfort sometimes prevents visits to the dentist, making it difficult to maintain proper oral hygiene and increasing the risk of plaque-related periodontal disease, which further exacerbates the destruction of periodontal tissues and increases the probability of long-term periodontal complications [14].

Periodontal diseases are inflammatory conditions of the tissues supporting teeth, potentially leading to tooth loss and systemic inflammation [15]. Periodontal disease involves localized infection and inflammation caused by anaerobic gram-negative bacteria that affect various periodontal tissues, including alveolar bone, periodontal ligament, cementum, and gingiva. Disease pathogenesis involves immunological responses that promote tissue destruction and bone loss [16].

Although specific microorganisms have not been directly associated with OLP, functional aspects of the oral microbiome are crucial in its development. Host factors, such as signaling pathways associated with keratinization, inflammation, and T-cell responses, are involved in OLP.

Interactions within the microbiome and between the microbiome and the host have a significant impact on overall health. For example, *P. gingivalis*, which, instead of attacking directly the bone, causes periodontal bone loss by disrupting the balance between the commensal microbiome and the host immune response [17].

The importance of studying the microbial community, rather than a specific microorganism, is emphasized in the study of diseases associated with microorganisms [18].

Study limitations

This study had some limitations. First, we used a cross-sectional design, which did not enable us to establish a causal relationship between oral microbiota and OLP or periodontitis. Second, we did not control for potential confounding factors, including smoking, alcohol consumption, oral health habits, medication intake, and systemic diseases, that may influence oral mucosal microbiota and periodontal health. Therefore, future studies should use a longitudinal design, adjust for confounding factors, and apply more modern techniques, such as metagenomics or metatranscriptomics, to further investigate the role of the microbiota in OLP and periodontitis.

Despite some limitations, our study has several advantages. In particular, this is one of the few studies of oral microflora in patients with OLP, which may provide new insights into OLP pathophysiology.

CONCLUSION

The study showed that in OLP patients, the frequency of detection of pathogenic microorganisms, such as

A. actinomycetemcomitans, *V. parvula*, *P. gingivalis*, and *T. denticola*, is increased. Our findings are relevant regardless of the presence of periodontitis in patients.

These results indicate a possible association of OLP with an increased frequency of detection of these pathogenic microorganisms; however, additional research is required to clarify their role in disease pathogenesis, which will allow for a deeper understanding of the mechanisms of OLP development and the development of novel methods for its diagnostics and treatment.

Thus, this study provides critical contribution to the understanding of OLP pathogenesis and can serve as a basis for the development of new approaches to the diagnosis and treatment of this disease.

ADDITIONAL INFORMATION

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Authors' contribution. All authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work. O.A. Svitich, N.P. Teplyuk, M.A. Stepanov — research concept, making significant edits to the manuscript in order to increase the scientific value of the article; N.O. Vartanova — obtaining data, significant edits in order to increase the scientific value of the article; B.Sh. Damdinova — analysis of the data obtained, interpretation of the results, a significant contribution to the writing of the article.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при подготовке статьи.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Вклад авторов. Авторы подтверждают соответствие своего авторства международным критериям ICMJE (все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией). Наибольший вклад распределён следующим образом: О.А. Свитич, Н.П. Теплюк, М.А. Степанов — концепция исследования, внесение в рукопись существенных правок с целью повышения научной ценности; Н.О. Вартанова — получение данных, существенные правки с целью повышения научной ценности статьи, Б.Ш. Дамдинова — анализ полученных данных, интерпретация результатов, существенный вклад в написание статьи.

REFERENCES

1. Alrashdan MS, Cirillo N, McCullough M. Oral lichen planus: A literature review and update. *Arch Dermatol Res.* 2016;308(8):539-551. doi: 10.1007/s00403-016-1667-2

2. Roopashree MR, Gondhalekar RV, Shashikanth MC, et al. Pathogenesis of oral lichen planus--a review. *J Oral Pathol Med.* 2010;39(10):729-734. doi: 10.1111/j.1600-0714.2010.00946.x

3. Gupta S, Jawanda M. Oral lichen planus: An update on etiology, pathogenesis, clinical presentation, diagnosis and management. *Indian J Dermatol.* 2015;60(3):222. doi: 10.4103/0019-5154.156315

4. Lucchese A, Di Stasio D, Romano A, et al. Correlation between oral lichen planus and viral infections other than HCV: A systematic review. *J Clin Med.* 2022;11(18):5487. doi: 10.3390/jcm11185487

5. Alaizari NA, Al-Maweri SA, Al-Shamiri HM, T et al. Hepatitis C virus infections in oral lichen planus: A systematic review and meta-analysis. *Aust Dent J.* 2016;61(3):282-287. doi: 10.1111/adj.12382

6. Shengyuan L, Songpo Y, Wen W, et al. Hepatitis C virus and lichen planus: A reciprocal association determined by a meta-analysis. *Arch Dermatol.* 2009;145(9):1040-1047. doi: 10.1001/archdermatol.2009.200

7. Villa TG, Sánchez-Pérez Á, Sieiro C. Oral lichen planus: A microbiologist point of view. *Int Microbiol.* 2021;24(3):275-289. doi: 10.1007/s10123-021-00168-y

8. Li S, Zhang Y, Yang Z, et al. Helicobacter pylori infection is correlated with the incidence of erosive oral lichen planus and the alteration of the oral microbiome composition. *BMC Microbiol.* 2021;21(1):122. doi: 10.1186/s12866-021-02188-0

9. Ertugrul A, Arslan U, Dursun R, Hakki S. Periodontopathogen profile of healthy and oral lichen planus patients with gingivitis or periodontitis. *Int J Oral Sci.* 2013;5(2):92-97. doi: 10.1038/ijos.2013.30

10. Wang H, Luo Z, Lei L, et al. Interaction between oral lichen planus and chronic periodontitis with Th17-associated cytokines in serum. *Inflammation.* 2013;36(3):696-704. doi: 10.1007/s10753-013-9594-2

11. Chen J, Liu K, Sun X, et al. Microbiome landscape of lesions and adjacent normal mucosal areas in oral lichen planus patient. *Front Microbiol.* 2022;13:992065. doi: 10.3389/fmicb.2022.992065

12. Nunes GP, Pirovani BO, Nunes LP, et al. Does oral lichen planus aggravate the state of periodontal disease? A systematic review and meta-analysis. *Clin Oral Invest.* 2022;26(4):3357-3371. doi: 10.1007/s00784-022-04387-z

13. Deo P, Deshmukh R. Oral microbiome: Unveiling the fundamentals. *J Oral Maxillofac Pathol.* 2019;23(1):122. doi: 10.4103/jomfp.JOMFP_304_18

14. Sanadi R, Khandekar P, Chaudhari S, et al. Association of periodontal disease with oral lichen planus: A systematic review

and meta analysis. *J Oral Maxillofac Pathol.* 2023;27(1):173. doi: 10.4103/jomfp.jomfp_178_22
15. Kinane DF, Stathopoulou PG, Papapanou PN. Periodontal diseases. *Nat Rev Dis Primers.* 2017;3(1):17038. doi: 10.1038/nrdp.2017.38
16. Nair S, Faizuddin M, Dharmapalan J. Role of autoimmune responses in periodontal disease. *Autoimmune Diseases.* 2014;2014:1-7. doi: 10.1155/2014/596824
17. Scapoli L, Girardi A, Palmieri A, et al. Microflora and periodontal disease. *Dent Res J (Isfahan).* 2012;9(Suppl 2):S202-206. doi: 10.4103/1735-3327.109755
18. Jung W, Jang S. Oral microbiome research on oral lichen planus: Current findings and perspectives. *Biology.* 2022;11(5):723. doi: 10.3390/biology11050723

СПИСОК ЛИТЕРАТУРЫ

1. Alrashdan M.S., Cirillo N., McCullough M. Oral lichen planus: A literature review and update // Arch Dermatol Res. 2016. Vol. 308, N 8. P. 539-551. doi: 10.1007/s00403-016-1667-2
2. Roopashree M.R., Gondhalekar R.V., Shashikanth M.C., et al. Pathogenesis of oral lichen planus a review // J Oral Pathol Med. 2010. Vol. 39, N 10. P. 729-734. doi: 10.1111/j.1600-0714.2010.00946.x
3. Gupta S., Jawanda M. Oral lichen planus: An update on etiology, pathogenesis, clinical presentation, diagnosis and management // Indian J Dermatol. 2015. Vol. 60, N 3. P. 222. doi: 10.4103/0019-5154.156315
4. Lucchese A., Di Stasio D., Romano A., et al. Correlation between oral lichen planus and viral infections other than HCV: A systematic review // J Clin Med. 2022. Vol. 11, N 18. P. 5487. doi: 10.3390/jcm11185487
5. Alaizari N.A., Al-Maweri S.A., Al-Shamiri H.M., et al. Hepatitis C virus infections in oral lichen planus: A systematic review and meta-analysis // Aust Dent J. 2016. Vol. 61, N 3. P. 282-287. doi: 10.1111/adj.12382
6. Shengyuan L., Songpo Y., Wen W., et al. Hepatitis C virus and lichen planus: A reciprocal association determined by a meta-analysis // Arch Dermatol. 2009. Vol. 145, N 9. P. 1040-1047. doi: 10.1001/archdermatol.2009.200
7. Villa T.G., Sánchez-Pérez Á., Sieiro C. Oral lichen planus: a microbiologist point of view // Int Microbiol. 2021. Vol. 24, N 3. P. 275-289. doi: 10.1007/s10123-021-00168-y
8. Li S., Zhang Y., Yang Z., et al. Helicobacter pylori infection is correlated with the incidence of erosive oral lichen planus and the alteration of the oral microbiome composition // BMC Microbiol. 2021. Vol. 21, N 1. P. 122. doi: 10.1186/s12866-021-02188-0
9. Ertugrul A., Arslan U., Dursun R., Hakki S. Periodontopathogen profile of healthy and oral lichen planus patients with gingivitis or periodontitis // Int J Oral Sci. 2013. Vol. 5, N 2. P. 92-97. doi: 10.1038/ijos.2013.30
10. Wang H., Luo Z., Lei L., et al. Interaction between oral lichen planus and chronic periodontitis with Th17-Associated cytokines in serum // Inflammation. 2013. Vol. 36, N 3. P. 696-704. doi: 10.1007/s10753-013-9594-2
11. Chen J., Liu K., Sun X., et al. Microbiome landscape of lesions and adjacent normal mucosal areas in oral lichen planus patient // Front Microbiol. 2022. Vol. 13. P. 992065. doi: 10.3389/fmicb.2022.992065
12. Nunes G.P., Pirovani B.O., Nunes L.P., et al. Does oral lichen planus aggravate the state of periodontal disease? A systematic review and meta-analysis // Clin Oral Invest. 2022. Vol. 26, N 4. P. 3357-3371. doi: 10.1007/s00784-022-04387-z
13. Deo P., Deshmukh R. Oral microbiome: Unveiling the fundamentals // J Oral Maxillofac Pathol. 2019. Vol. 23, N 1. P. 122. doi: 10.4103/jomfp.JOMFP_304_18
14. Sanadi R., Khandekar P., Chaudhari S., et al. Association of periodontal disease with oral lichen planus: A systematic review and meta-analysis // J Oral Maxillofac Pathol. 2023. Vol. 27, N 1. P. 173. doi: 10.4103/jomfp.jomfp_178_22
15. Kinane D.F., Stathopoulou P.G., Papapanou P.N. Periodontal diseases // Nat Rev Dis Primers. 2017. Vol. 3, N 1. P. 17038. doi: 10.1038/nrdp.2017.38
16. Nair S., Faizuddin M., Dharmapalan J. Role of responses in periodontal autoimmune disease // Autoimmune Diseases. 2014. Vol. 2014. P. 1-7. doi: 10.1155/2014/596824
17. Scapoli L., Girardi A., Palmieri A., et al. Microflora and periodontal disease // Dent Res J (Isfahan). 2012. Vol. 9, Suppl. 2. P. S202-206. doi: 10.4103/1735-3327.109755
18. Jung W., Jang S. Oral microbiome research on oral lichen planus: Current findings and perspectives // Biology. 2022. Vol. 11, N 5. P. 723. doi: 10.3390/biology11050723

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