

Association of periodontal disease with oral lichen planus: A systematic review and meta analysis

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Abstract

Background: Gingival lesions in oral lichen planus can indirectly increase the risk of plaque-induced periodontal disease when symptoms associated with such lesions hamper the proper oral hygiene maintenance by the patients and can increase the risk of periodontal tissue destruction. This systematic review analyses the existing evidence on the association between oral lichen planus and periodontal disease.

Aim: This systematic review of case-control studies aimed to analyse the association between periodontal disease and oral lichen planus.

Material and Methods: An electronic database search for randomised controlled trials, experimental studies, case-control studies, and cohort studies published in peer-reviewed Journals in the English language was conducted from the following databases: PubMed, EBSCOHost, Science Open, EMBASE, and Google Scholar.

Results: A total of 12,507 were identified on an electronic database search. Only eight studies fulfilled the eligibility criteria and were included for quantitative analysis. A data extraction sheet was prepared, and studies were analysed.

Conclusion: Bleeding on Probing and Probing depth were seen to be significantly associated with Oral Lichen Planus. The symptoms in Oral Lichen Planus impede efficient oral hygiene maintenance by a patient and predispose them to the occurrence of long-term Periodontal Disease.

Keywords: Lichen planus, periodontal disease, bleeding on probing, probing depth, bone loss

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INTRODUCTION

Periodontitis is characterised by microbially-associated; host-mediated inflammation that leads to loss of periodontal attachment. The bacterial biofilm formation initiates gingival inflammation; however, periodontitis initiation and progression rely on dysbiotic ecological changes within the microbiome in response to nutrients from gingival inflammatory and tissue breakdown products

that enrich some species and anti-bacterial mechanisms that try to contain the microbial challenge within the gingival sulcus area once the inflammation has initiated. Current evidence supports multifactorial disease influences, like smoking, on multiple immunoinflammatory responses that make the dysbiotic microbiome changes more likely for a

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patient than others and are likely to influence the severity of disease for such individuals.^[1]

Oral lichen planus (OLP) is a chronic inflammatory oral mucosal disease with cell-mediated immune pathogenesis, in which T lymphocytes accumulate beneath the epithelium of the oral mucosa and lead to hyperkeratosis and erythema in lesions.^[2] The key characteristic of this disease is an inflammatory reaction toward an unidentified antigen within the basal epithelial layer/basement membrane zone. The lesions, usually bilateral, often involve the gingiva and present as desquamative gingivitis, causing pain and discomfort during eating and toothbrushing. The clinical diagnosis depends on the presence of papular or reticular-type lesions, eventually supported by histopathologic findings of hyperkeratosis, degenerative changes of basal cells, and subepithelial inflammation dominated by lymphocytes and macrophages.^[3] Investigations indicated that the increased plaque and calculus deposits are related to a significantly higher incidence of periodontal deterioration, especially in the presence of gingival atrophic-erosive-type OLP lesions.^[4]

An international workshop for the classification of periodontal disease organised by the American Academy of Periodontology categorizes lichen planus as a gingival manifestation of systemic conditions under the non-plaque induced gingival lesion. It has been suggested that gingival lesions in oral lichen planus can indirectly increase the risk of developing plaque-induced periodontal disease when symptoms associated with such lesions interfere with the maintenance of proper oral hygiene and might enhance the risk of periodontal tissue destruction. Oral lichen planus solely may not be responsible for the worsening of the condition, but the presence of plaque and calculus due to improper oral hygiene makes it worse in cases with OLP.^[5]

The erosive form of lichen planus is mostly persistent and painful to patients, and they are not able to maintain oral hygiene procedures regularly, which results in the deposition of plaque and calculus, which may exaggerate the condition and increase the likelihood of long-term periodontal diseases.^[6]

The results of various studies have shown that periodontal parameters were higher in the case group as compared to the control group. It seems reasonable to believe that patients with desquamative gingivitis resulting from OLP may have impaired capacity to perform efficient oral hygiene practices, and hence the increased gingival inflammation levels and periodontal breakdown were observed in these patients.^[7]

RATIONALE

Although there are various studies analysing the association of periodontal disease with lichen planus, there is limited documentation regarding the same. Therefore, the aim of this systematic review and meta-analysis was to analyse the existing evidence on the association of periodontal disease with oral lichen planus.

Focused question

Is there an association of periodontal disease with oral lichen planus?

Another research question

Is there a difference in the occurrence of periodontal disease in patients with oral lichen planus?

Primary objective

To evaluate the association of periodontal disease with oral lichen planus.

Secondary objective

To analyse the occurrence of periodontal disease in patients with oral lichen planus.

MATERIALS AND METHODS

Protocol and Registration: The systematic review was conducted in accordance with the Preferred Reporting Items of Systematic Reviews (PRISMA) and Meta-analysis statement.

Registration number: CRD42022306085.

Study design

This is a systematic review and meta-analysis of observational, case-controls, and clinical trials studies, which was aimed to evaluate the existing evidence of the association of periodontal disease with oral lichen planus.

Inclusion criteria

1. Case-control studies
2. Full-text articles published in peer-reviewed journals
3. Articles in which periodontal parameters Bleeding on probing, Probing Depth, Clinical Attachment Level, Plaque Index, and Gingival Index were assessed.
4. Articles published in English language only.

Exclusion criteria

1. Unpublished research
2. Animal model studies and in-vitro studies
3. Case series and case reports
4. Articles whose full text was not available

- Articles in which periodontal parameters that were assessed were not mentioned.

Information sources and search strategy

An electronic database search for randomised controlled trials, experimental studies, case-control studies, and cohort studies published in peer-reviewed journals in the English language was conducted from the following databases: PubMed, EBSCOHost, ScienceOpen, EMBASE, and Google Scholar.

The search terms used were

Periodontal Disease AND Oral Lichen Planus.

Periodontal Disease OR Oral Lichen Planus.

Periodontitis AND Oral Lichen Planus.

Periodontitis OR Oral Lichen Planus.

Study selection

Study selection was carried out in two phases:

- Assessment of titles and abstracts
- Assessment of full text.

Data collection process

A data extraction sheet was prepared based upon variables associated, and the articles were analysed. Using data extraction sheet, the following data were collected: authors, year of publication, country, aim, type of study, sample size, comparison group and control group, methodology, and conclusion.

RESULTS

A total of 12507 articles were found after electronic search. 12884 articles, which were of other languages and duplicates, were excluded leaving 23 articles after title screening. 7 articles were excluded as they did not fulfill the eligibility criteria leaving 8 articles. Figure 1 shows the flow chart of literature search results and study selection.

Studies included for the analysis

Eight studies were included for the quantitative synthesis. Out of the 8 studies, 1 was pilot study and 7 studies were case control studies. An overview of the included studies for the analysis is presented in Table 1.

Studies excluded for the analysis

The characteristics of studies excluded for the analysis and the reason for exclusion has been presented in Table 2.

Statistics

The odds ratio (OR) with a 95% confidence interval (CI)

was calculated for dichotomous outcomes. A fixed-effects model (Mantel-Haenszel method) was used if there was no heterogeneity ($P > 0.05$ or $I^2 \leq 24\%$), otherwise, a random-effects model (Der Simonian-Laird method) was used. All statistical analyses were performed using the RevMan 5.3 (Cochrane Collaboration, Software Update, Oxford, UK).

Assessment of heterogeneity

The significance of any discrepancies in the estimates of the treatment effects of the different trials was assessed by means of Cochran's test for heterogeneity and the I^2 statistics, which describes the percentage of the total variation across studies that is due to heterogeneity rather than chance. Heterogeneity was considered statistically significant if $P < 0.1$. A rough guide to the interpretation of I^2 given in the Cochrane handbook is as follows: (1) from 0 to 40%, the heterogeneity might not be important; (2) from 30% to 60%, it may represent moderate heterogeneity; (3) from 50% to 90%, it may represent substantial heterogeneity; (4) from 75% to 100%, there is considerable heterogeneity.

Assessment of risk of bias in included studies

The quality of included studies was evaluated based on Newcastle Ottawa Scale, and accordingly, a numeric score (NOS Score) was assigned. It was designed to evaluate bias based on participant selection, study group comparability in a cross-sectional study, attainment of exposure in case-control studies, and outcome of interest in a cohort study. The NOS uses a nine-star rating system with a maximum of four points available for selection, two for comparability, and three for the assessment of the outcome or exposure. Quality appraisal of the included studies was undertaken by the two authors, and a third author was consulted in the event of any discrepancy. A study with a score from 7 to 9 will be considered high quality, 4 to 6 will be considered moderate quality, and 0 to 3 will be considered low quality or very high risk of bias. We completed a 'Risk of bias' table for each included study. Three studies were awarded five stars and deemed to be of moderate quality (Azizi *et al.* 2012,^[7] Ertugrul *et al.* 2013^[9] and Ertugrul *et al.* 2013).^[12] Three studies were awarded four stars and deemed to be of moderate quality (Wang H *et al.* 2014,^[11] Ertugrul *et al.* 2013^[9] and Rai *et al.* 2016).^[5] At the same time, two studies were awarded three stars and deemed to be of low quality (Fluixa *et al.* 1999^[4] and Wang H *et al.* 2014).^[11] The risk of bias of the included studied is presented in Table 3.

DISCUSSION

Lichen planus (LP) is a common chronic inflammatory

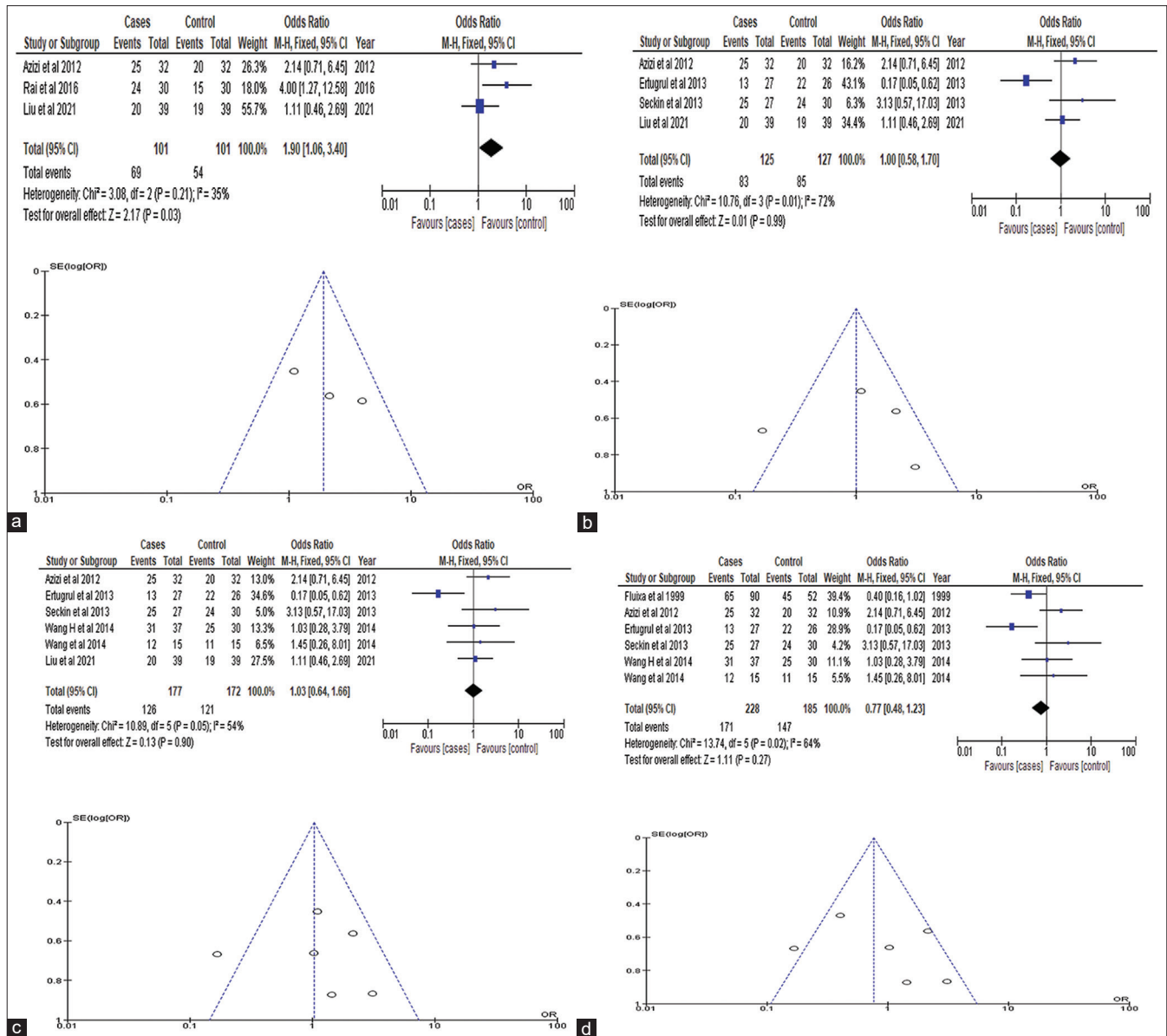


Figure 1a-d: (a) **Bleeding on Probing:** Three studies reported bleeding on probing data for the lichen planus and control group. The results of the meta-analysis revealed that with respect to bleeding on the probing parameter, the cases have 1.9 times higher odds of developing the disease through a fixed-effect model, while this difference was statistically significant with a P value < 0.05 . (b) **Clinical attachment level:** Four studies reported clinical attachment level data for the lichen planus and control group. The results of the meta-analysis revealed that with respect to clinical attachment level, the cases and controls have equal odds of developing the disease through a fixed-effect model. (c) **Gingival index:** Six studies reported gingival index data for the lichen planus and control group. The results of the meta-analysis revealed that with respect to the inference from the gingival index, the cases and controls have equal odds of developing the disease through a fixed-effect model. (d) **Plaque index:** Six studies reported plaque index data for the lichen planus and control group. The results of the meta-analysis revealed that with respect to the inferences from the plaque index, the cases have 0.7 times lesser odds of developing the disease than the controls through a fixed-effect model

condition that can affect the skin and mucous membranes, including the oral mucosa. The oral variant, oral lichen planus (OLP), is a chronic inflammatory disease affecting the oral mucosa with characteristic relapses and remissions.

The precise etiology of OLP is unknown, and only a few predisposing factors like genetic background, psychological factors, trauma, systemic associations, hepatitis C virus (HCV), hypertension and diabetes mellitus, thyroid

dysfunction, systemic medications, etc., are currently thought to potentially have a role in its pathogenesis.^[13]

Clinically, the oral lesions have been grouped into reticular, papular, plaque-like, atrophic, erosive, and bullous forms. OLP usually occurs in a bilaterally symmetrical pattern, commonly involving buccal mucosa, gingivae, and dorsum of the tongue.^[5] The lesions are usually painless, though pain and burning sensation are associated with erosive and atrophic lesions.^[14]

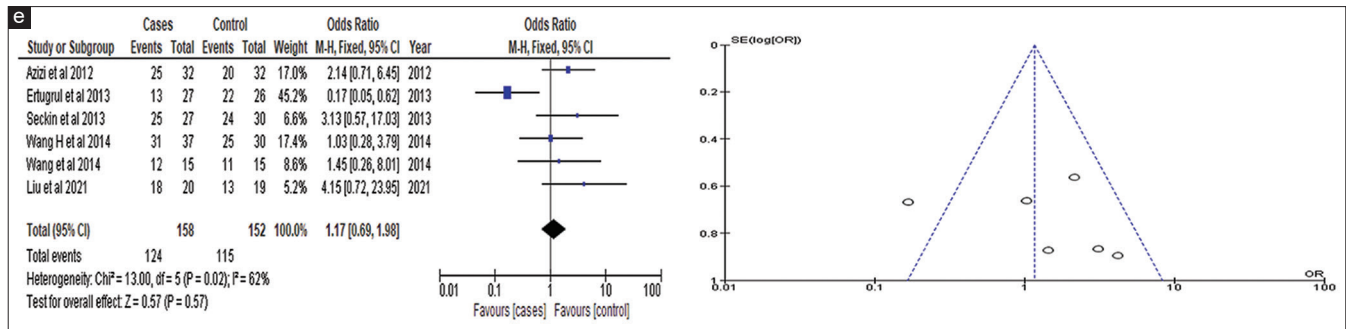


Figure 1e: Probing depth: Six studies reported probing depth data for the lichen planus and control group. The results of the meta-analysis revealed that with respect to the probing depth, the cases have 1.1 times higher odds of developing the disease through a fixed-effect model. **Funnel plots:** The funnel plot did not show any significant asymmetry, indicating the absence of publication bias, as shown in **Figures. A** funnel plot showing symmetric distribution without systematic heterogeneity of individual study treatment effects compared to the standard error indicates a lack of publication bias in the meta-analysis

When OLP is present, it is challenging for the patient to eat, drink, and function because of constant pain.^[15] In addition, discomfort caused by gingival lesions could predispose patients to limit their dental visits on a regular basis. OLP gingival lesions are usually persistent and painful, thus limiting efficient teeth brushing. This leads to plaque accumulation and could increase the possibility of long-term periodontal diseases.^[7]

The purpose of this systematic review and meta-analysis was to analyse the existing evidence on the association of periodontal disease with oral lichen planus. Eight case-control studies were included for the quantitative analysis. Studies in which periodontal parameters Bleeding on probing, Probing Depth, Clinical Attachment Level, Plaque Index, and Gingival Index were assessed were included in this systematic review and meta-analysis to study the association between periodontal disease and oral lichen planus.

Azizi A *et al.* 2012^[7] reported that periodontal parameters, namely plaque index (PI), gingival index (GI), bleeding on probing (BOP), probing depth (PD), clinical attachment level (CAL) were observed to be higher in OLP patients as compared to the control group. Fluixa CR *et al.* 1999^[4] observed that increased plaque and calculus deposits were associated with a significantly higher incidence of atrophic-erosive gingival lesions in patients with OLP.

Rai *et al.* 2016^[5] revealed that GI, PI, and BOP were observed to be higher in the OLP group compared to the control group. Wang H *et al.* 2013^[8] in a case-control study, reported that serum IL-17 level, probing depth, and plaque index in OLP-CP group patients were significantly higher as compared with chronic periodontitis patients alone.

Ertugrul AS *et al.* 2013^[9] in a study showed increased MMP-1, MMP-9 levels, and decreased TIMP-1 levels in

the gingival tissue and gingival crevicular fluid (GCF) of OLP patients. These findings, along with poor oral hygiene, may cause increased tissue breakdown. Periodontal parameters were significantly affected in OLP chronic periodontitis (OLPP) compared to the OLP gingivitis and Non-OLP periodontitis group.

Liu H *et al.* 2021^[10] concluded that subgingival plaque as an initial factor of periodontal disease might prevent OLP gingival lesions from healing, which changes the characteristics of the lesions into more aggressive forms such as erosive lesions.

Wang H 2014 *et al.*^[11] increased the expression levels of IL-17 and IL-23 in periodontal tissues from periodontitis patients with oral lichen planus, which might aggravate the inflammatory response in local lesions. The periodontal parameters were higher in CP-OLP group.

Ertugrul AS *et al.* 2013^[12] concluded that periodontopathogens percentages of detection in the OLP groups were higher than those in the non-OLP groups. The Oral lichen planus with gingivitis (OLPG) patients had higher periodontal clinical parameters than the healthy non-lichen planus patients with gingivitis (HG) patients, but a statistically significant difference was not observed.

The risk of bias existed in the studies as randomisation of study participants and blinding was not performed. This could have influenced the outcome of the studies. However, based on the studies analysed, it was observed that oral lichen planus did have an effect on BOP and PD, but significant differences were not observed with the rest of the parameters. There remains a future scope for well-designed longitudinal studies with long-term follow-up to be conducted.

Table 1: Characteristics of the studies included in the systematic review

Authors and year of publication	Aim	Type of study	Sample size, comparison group, and control group	Periodontal parameters assessed	Conclusion
Azizi A, Rezaee M 2012 ^[7]	To examine the periodontal status of subjects with gingival oral lichen planus (OLP) compared to control	Case-control study	Total of 64 subjects (32 with oral lichen planus, 32 without oral lichen planus controls)	Plaque index (PI), Bleeding on probing (BOP), Clinical attachment level (CAL), Gingival Index (GI), Probing depth (PD)	PI, GI, PD, BOP, and CAL were significantly higher in the case group compared with the control group.
Ramon-Fluixa C, Bagan-Sebastian JV, Milian-Masanet MA, Scully C 1999 ^[4]	To investigate the possible influence of dental plaque and derived periodontal alterations upon OLP lesions.	Case-control study	90 individuals with Oral Lichen Planus and 52 individuals without lichen planus (controls)	PI, Simplified calculus Index (CIS), Periodontal Disease Index (PDI)	Increased plaque and calculus deposits are associated with a significantly higher incidence of atrophic-erosive gingival lesions in individuals with OLP.
Rai NP, Kumar P, Mustafa SM, Divakar DD, Kheraif AA, Ramakrishnaiah R, <i>et al.</i> 2016 ^[5]	To evaluate the periodontal status of OLP patients and compare it with that of healthy controls.	Pilot study	30 (erosive and reticular) OLP and 30 controls	GI, Russell's periodontal index (RPI), and BOP	Periodontal status was poor in the study group as compared to the control group.
Wang H, Luo Z, Lei L, Sun Z, Zhou M, Dan H, <i>et al.</i> 2014 ^[8]	To compare serum expression levels of IL-23 and IL-17 in chronic oral lichen planus patients with periodontitis (OLP-CP), patients only with chronic periodontitis (CP) or OLP, and healthy controls (HC).	Case-control study	35 Oral Lichen Planus-chronic periodontitis patients (OLP-CP), 35 Oral Lichen Planus patients (OLP), 30 Chronic Periodontitis patients (CP), and 30 healthy controls (HC)	Serum IL-17 and IL-23 PD, attachment loss (AL), PI, GI	There was a significantly positive correlation between serum IL-17 level, probing depth, and plaque index in OLP-CP group patients
Ertugrul AS, Dursun R, Dundar N, Avunduk MC, Hakki SS 2013 ^[9]	To investigate matrix metalloproteinase (MMP)-1, MMP-9, and MMP inhibitor-1 (TIMP-1) levels in gingival crevicular fluid (GCF) by enzyme-linked immunosorbent assay and by immunohistochemical staining of samples from patients with and without OLP.	Case-control study	27 patients with OLP and 30 healthy non-OLP patients	MMP-1, MMP-9, and TIMP-1 levels in GCF PI, GI, PD, CAL	Increased levels of MMP-1 and MMP-9 with decreased levels of TIMP-1 in GCF and in the gingival tissue of OLP patients, in combination with poor oral hygiene, may cause increased tissue breakdown.
Liu H, Chen H, Liao Y, Li H, Shi L, Deng Y, <i>et al.</i> 2021 ^[10]	To compare the microbiota composition and bacterial diversity of subgingival plaque in chronic periodontitis patients with and without gingival erosive oral lichen planus.	Case-control study	20 chronic periodontitis patients with gingival erosive oral lichen planus (CP-OLP group) and 19 chronic periodontitis patients without gingival erosive oral lichen planus (CP group)	Subgingival microbiota, Full mouth probing depth (FMPD), CAL	Microbiome in erosive OLP with CP was significantly different from that found in only CP, and microbiome changes might be related to the presence or absence of OLP disease.
Wang H, Han Q, Luo Z, Xu C, Liu J, Dan H, <i>et al.</i> 2014 ^[11]	To compare the expression levels of interleukin (IL)-17 and IL-23 in local periodontal tissues from patients with both chronic periodontitis and oral lichen planus (CP-OLP), patients with chronic periodontitis (CP) only, patients with oral lichen planus (OLP) only, and healthy controls (HC).	Case-control study	15 CP-OLP patients, 15 CP patients, 15 OLP patients, and ten healthy controls.	CAL, PD, Silness-Loe plaque index (PI), and Loe-Silness gingival index (GI) scores.	There was an increased expression level of IL-17 and IL-23 in periodontal tissues from periodontitis patients with oral lichen planus, which might aggravate the inflammatory response in local lesions.

Contd...

Table 1: Contd...

Authors and year of publication	Aim	Type of study	Sample size, comparison group, and control group	Periodontal parameters assessed	Conclusion
Ertugrul AS, Arslan U, Dursun R, Hakki SS 2013 ^[12]	To identify the prevalence of the detection of periodontopathogenic microorganisms (Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythia and Treponema denticola in OLP patients and to compare with this prevalence of periodontopathogenic microorganisms in healthy non-OLP patients.	Case-control study	27 (18 chronic periodontitis (OLPP) and nine gingivitis (OLPG) patients diagnosed with OLP, along with 26 (13 chronic periodontitis (HP) and 13 gingivitis (HG) healthy non-OLP patients.	Periodontopathogenic microorganisms present in subgingival plaque samples Actinomycetemcomitans, P. Gingivalis, P. Intermedia, T. Forsythia and T. Denticola) PI, GI, PD, CAL	OLP patients have higher levels of infection with A. Actinomycetemcomitans, P. Gingivalis, P. Intermedia, T. Forsythia and T. Denticola as compared to non-OLP patients.

Table 2: Characteristics of excluded studies

Authors (Year)	Reason of Exclusion
Çakçak DS, Karakas AA, Tosun O, Kacaroglu H, Alpsoy E 2016 ^[15]	Did not meet the inclusion criteria
Abdel-Haq A, Kusnierz-Cabala B, Darczuk D, Sobuta E, Dumnicka P, Wojas-Pelc A, Chomyszyn-Gajewska M 2014 ^[16]	Did not meet the inclusion criteria
Mignogna MD, Russo LL, Fedele S 2005 ^[17]	Case series
Miricescu D, Totan A, Calenic B, Mocanu B 2015 ^[18]	Did not meet the inclusion criteria
Bianco L, Romano F, Maggiora M, Bongiovanni L, Guzzi N, Curmei E, <i>et al.</i> 2019 ^[19]	Did not meet the inclusion criteria
Stone SJ, McCracken GI, Heasman PA, Staines KS, Pennington M 2013 ^[20]	Did not meet the inclusion criteria
Guiglia R, Di Liberto C, Pizzo G, Picone L, Lo Muzio L, Gallo PD, <i>et al.</i> 2007 ^[21]	Did not meet the inclusion criteria

Table 3: Assessment of risk of bias in included studies

Author, year	Selection (Max=4)	Comparability (Max=2)	Exposure/ Outcome (Max=3)	Overall quality score (Max=9)
Azizi A, <i>et al.</i> (2012) ^[7]	***	*	*	*****
Ramon-Fluixa C, <i>et al.</i> (1999) ^[4]	*	*	*	***
Rai NP, <i>et al.</i> (2016) ^[5]	**	*	*	****
Wang H, <i>et al.</i> (2014) ^[8]	**	-	*	***
Ertugrul AS, <i>et al.</i> (2013) ^[9]	***	*	*	****
Liu H, <i>et al.</i> (2021) ^[10]	***	*	*	*****
Wang H, <i>et al.</i> (2014) ^[11]	**	*	*	****
Ertugrul AS, <i>et al.</i> (2013) ^[12]	***	*	*	*****

CONCLUSION

Oral lichen planus lesions are painful and can limit efficient oral hygiene maintenance by the patient, which can predispose them to develop long-term periodontal disease. Results of this meta-analysis revealed that Bleeding on probing and Probing Depth were significantly

associated with Lichen planus. A significant difference was not observed with the rest of the parameters in the OLP patients. The results of the included studies were inconclusive, and hence further well-designed longitudinal studies with a long-term follow-up period should be carried out for definite conclusions.

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Conflicts of interest

There are no conflicts of interest.

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