Is oral lichen planus a risk factor for peri-implant diseases? A systematic review and meta-analysis

Xiaoqin Xiong

Guangzhou Medical University

Ting Yu (✉ dent_yu@163.com)

<https://orcid.org/0000-0002-8381-010X>

Tiantian Xu

Guangzhou Medical University

Xinhong Wang

Guangzhou Medical University

Wenguang Qin

Guangzhou Medical University

Luo Gang

Guangzhou Medical University

Research article

Keywords: dental implants; peri-implant diseases; oral lichen planus; systematic review; meta-analysis.

Posted Date: March 4th, 2020

DOI: <https://doi.org/10.21203/rs.3.rs-15960/v1>

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Version of Record: A version of this preprint was published at BMC Oral Health on May 20th, 2020. See the published version at <https://doi.org/10.1186/s12903-020-01134-2>.

Abstract

Background : To evaluate whether oral lichen planus (OLP) is a risk factor for peri-implant diseases (PIDs) with a systematic review and meta-analysis. **Methods :** Six electronic databases including Medline, Web of Science, etc. were searched. Included studies are observational human studies written in English. Population of interest were those with/without OLP who received dental implant treatment. Follow-up time after implantation is from one month to 20 years. The quality of the included literature regarding risk of bias and methodology was assessed with Newcastle-Ottawa Scale or the Agency for Healthcare Research and Quality. The data involving exposure (OLP), primary outcomes (implants having PIDs) and secondary outcomes (probing depth/PD, bleeding on probing/BOP and bone loss/BL) and potential confounders were extracted. Heterogeneity was assessed by I^2 tests. Dichotomous data were expressed as risk ratio (RR) and 95% confidence interval (CI) which were calculated with a fixed effect model. **Results :** Of 139 literatures, two studies were enrolled and evaluated as high quality, which totally contained 68 participants receiving 222 (OLP vs. non-OLP, 112 vs. 110) implants with 12 to 120-month follow-up time. Proportions of implants with PIDs between OLP and non-OLP groups were as follows: 19.6% (22/112) vs. 22.7% (25/110) for PIM; 17.0% (19/112) vs. 10.9% (12/110) for PI. Meta-analysis found no recognizable difference in number of implants with PIDs (PI: RR = 1.49, 95% CI 0.77-2.90, P = 0.24; PIM:RR = 0.88, 95% CI 0.53 -1.46, P = 0.61; PIDs: RR = 1.08, 95% CI 0.75 -1.55, P = 0.68) or BOP (RR = 0.90, 95% CI: 0.70-1.15, P = 0.40) between OLP and non-OLP groups. **Conclusions :** Available literature regarding the effects of OLP on PIDs remains very limited. Existing evidence seems not support OLP as a suspected risk factor for PIDs. Large-scale prospective trials are required to test the findings. **Keywords :** dental implants; peri-implant diseases; oral lichen planus; systematic review; meta-analysis.

Background

Dental implant treatment is a well-established method for restoring lost teeth. Despite a high rate of survival (>94%) for more than 5 years [1, 2], a very high proportion (8%-44%) of dental implants develop peri-implant diseases (PIDs), including peri-implant mucositis (PIM) and peri-implantitis (PI) [3], which can lead to implant failure or loss. PIDs are pathological inflammatory conditions, of which PIM involves only peri-implant mucosal inflammation, while PI causes both peri-implant mucosa and alveolar bone damage [4], accompanied by bleeding on probing, suppuration, increased probing depth, and progressive marginal bone loss[5]. PID-associated risk factors include poor oral hygiene [6], smoking [7], a history of periodontitis [4, 8] and systemic diseases, such as diabetes [9].

Oral lichen planus (OLP), a chronic systemic autoimmune disease, affects one to two percent of the general population, especially middle-aged and elderly females [10]. Typically, OLP manifests as plaque-like or white reticular lesions, an erythematous area or ulceration [11, 12], and it can affect the oral mucosa, tongue or gingiva [13]. Recently, OLP has been suspected as a potential risk factor for PIDs. Some scholars have speculated that OLP may directly alter the nature of the mucosal barrier around dental implants, promoting bacterial invasion [14]. Furthermore, OLP patients have been found to have poor oral hygiene[15] and quality of life [16]. These concerns have previously made OLP a

contraindication to implant treatment[14]. However, some studies, in which 66 short implants were implanted in 23 OLP patients, with a survival rate of 98.5%, did not observe any difference in the success rate between OLP patients and normal controls [17, 18]. Notably, most of the studies regarding implantation in OLP populations were observational studies (including case reports, retrospective studies, etc.) with small sample sizes. Hence, whether dental implantation in OLP patients is safe remains inconclusive. The present systematic review aimed to analyze the existing literature to determine whether OLP is a risk factor for PIDs.

Methods

Focused question

Compared with non-OLP populations, are there changes in the rates of probing depth, bone loss or bleeding on probing around implants in OLP patients? Are there different rates of PIM and PI prevalence in OLP patients compared with non-OLP populations? This manuscript has been prepared based on the Preferred Reporting Items for Systematic Review and Meta-analyses (PRISMA) guidelines for reporting systematic reviews.

Population and exposure

The population of interest was patients with and without OLP who received dental implant treatment. The studies included participants aged ≥ 18 years with no periodontitis or stable periodontitis after treatment. Their OLP status was diagnosed before implant treatment. It was preferred that OLP patients had a diagnosis of the OLP subtype or concomitant symptoms, such as desquamative gingivitis which could be considered separate exposure factors. Detailed records of treatments, including medications, were included for all OLP patients. It was assumed that implant treatment was performed during the remission stage of OLP. The primary outcomes were PIDs, and the secondary outcomes were probing depth, bleeding on probing, and bone loss. Implant failure was also considered if its infective origin was clearly stated.

Disease determinants, risk factors, and etiologic agents

The potential association of OLP with PID determinants, including sex, age, educational background, socioeconomic position, genetics, lifestyle, nutrition, health behaviors, and microbiological factors, was recorded when available. Whenever possible, the quality of the determinant/exposure measurements were assessed.

There are some general risk indicators/factors for PI and PIM, including a history of periodontitis[19], systemic diseases, genetic traits, and smoking. Local risk indicators/factors for PI and PIM include oral hygiene/poor plaque control, poor compliance with supportive implant therapy, presence of excess cement, implant materials and surface characteristics, design of implant-supported prostheses and dimensions of keratinized mucosa [4, 8].

Study follow-up duration

All studies with a mean duration or follow-up interval of at least one month were included because the earliest time for PID diagnosis was approximately one month after loading restorations [4, 20]. If the follow-up time was reported as a range, the low value of the range was more than one month.

Types of studies

With the goal of identifying studies for inclusion, certain methods were designed based on acceptability criteria in this study. Eligible studies, including observational or longitudinal studies or prospective research, were those with a follow-up time ≥ 1 month, with primary outcomes of PIDs. The secondary outcomes were probing depth, bleeding on probing and bone loss.

Inclusion criteria

1. Observational human studies, such as case-control, cross-sectional, retrospective or prospective cohort studies;
2. Studies in which OLP and PIDs were the main exposure factor and outcome, respectively;
3. Studies in which both OLP and PIDs were the definitive diagnoses, and the diagnosis of OLP was made according to clinical and pathological evidence;
4. Studies with a follow-up time of ≥ 1 month after implant placement; and
5. Studies published in the English language.

Exclusion criteria

1. Studies lacking control groups (i.e., a non-OLP group);
2. Narrative reviews, comments, letters, case reports or series, and conference abstracts; and
3. Studies for which the full-text articles or original data could not be obtained.

Search strategy

The search was performed in June 2019. All available articles published online before this time in six electronic databases, including Medline, Web of Science, the Cochrane Library, John Wiley, Scopus and Springer, were retrieved. Manual searching in dental journals, especially those focusing on implantology, was also performed. The search strategy was established with adequate discussion among all the coauthors (Table 1).

Study selection

Two researchers independently screened the headlines and abstracts of the studies identified in the searches. Afterward, the full texts of all the publications that met the inclusion criteria were obtained; however, it was not possible to make a decision about inclusion due to a lack of information in the titles and abstracts. Any research that was considered relevant by one of the reviewers was included in the

following screening stage. Subsequently, the articles were independently assessed in duplicate by the reviewers. Any disparity about research eligibility was settled by discussion among the two reviewers, and if no consensus could be reached, the decision was resolved through arbitration by a third reviewer. All articles that did not fulfill the eligibility criteria were excluded.

For studies with missing data or those for which a clear decision on study eligibility could not be made after assessment of the full text, the corresponding authors were e-mailed to ask for the information needed for a final decision. Messages via social media or academic websites were also sent to the corresponding authors in the absence of response by e-mail.

Data extraction and management

A detailed form designed specifically for data extraction was used, and all the relevant data from the collected articles were independently extracted by two independently. Any disagreements were settled through evaluation by the third examiner or group consensus.

The following research information was extracted (Table 2):

1. Type of research;
2. Sample framework (e.g., community, university);
3. Sample size, sex and age of participants;
4. Diagnostic criteria for PI, PIM and OLP;
5. Follow-up time; and
6. Confounding factors relative to exposure factors.

Outcomes

1. probing depth change;
2. bone loss level change;
3. bleeding on probing level change; and
4. Number of implants with/without PI and PIM;

Quality assessment

The Newcastle Ottawa Scale (NOS) [21] and the Agency for Healthcare Research and Quality (AHRQ) [22] standards were used to assess the quality of cohort and cross-sectional studies, respectively. The cohort study degree of bias was categorized with a star-based quality assessment system that considers three categories as follows: the selection of the study groups (four stars), the comparability of the groups (two stars), and the ascertainment of either the exposure or outcome of interest (three stars). An 11-item checklist recommended by the AHRQ was used to assess the methodological quality of the cross-sectional studies. Article quality was assessed as follows: low quality (0 to 3); moderate quality (4 to 7); and high quality (8 to 11).

Data synthesis

Statistical heterogeneity in risk in patients suffering from PIDs in the collected studies was evaluated using both risk ratios (RRs) and I^2 measures. The use of I^2 values was based on the Cochrane Handbook: $0 < I^2 < 40\%$ represents a lack of heterogeneity; $30\% < I^2 < 60\%$ represents moderate heterogeneity; $50\% < I^2 < 90\%$ signals substantial heterogeneity; and $75\% < I^2 < 100\%$ shows considerable heterogeneity.

When suitable, meta-analysis was used to assess risk in patients suffering from PIDs based on implant and patient characteristics. A random effects approach was used if there was mild to moderate statistical heterogeneity; otherwise, a fixed-effect approach was chosen. Considering that only observational studies were included in the present systematic review, the primary outcome variable was the RRs of PI and PIM. The RRs were calculated with 95% confidence intervals (CIs) (Review Manager, v5.2, The Nordic Cochrane Center, The Cochrane Collaboration, Copenhagen, Denmark) to quantify the association between OLP and the risk of PI and PIM. Forest plots were generated showing the RRs and 95% CIs of the involved studies. If there were more than 10 studies included in the meta-analysis, publication bias was evaluated qualitatively by a funnel plot [23].

Results

Search

A total of 139 studies were identified after electronic and manual searches (Fig 1). After removing duplicates, 83 articles were retained. Then, 58 articles were excluded after evaluating the titles and abstracts, and the remaining 25 articles were selected for full-text evaluation. Of the 25 articles, 13 articles involved both OLP and implant treatment. Then, 11 articles were excluded for various reasons, such as being case reports, reviews, etc., and the remaining 2 articles were retained for final review. The screening outcomes were similar regardless of whether the literature was published in English [24].

Study characteristics

Location and sample characteristics

The two included studies included one prospective [25] study and one cross-sectional study [16] (Table 2). Both studies were conducted in the same country (Spain) but by different institutions. The two studies contained 34 OLP patients and 34 non-OLP controls aged between 29 and 79 years. The 68 participants received 224 implants (OLP vs. non-OLP, 112 vs. 110) with a follow-up time of 12 to 120 months.

At the implant level, 19.6% (22/112) and 22.7% (25/110) developed PIM in the OLP and non-OLP groups, while 17.0% (19/112) and 10.9% (12/110) developed PI in the two groups, respectively. Additionally, bleeding on probing was present at the sites of 42.6% (48/112) and 50.0% (55/110) of implants in the two groups, respectively.

The ratios of PIDs in the two groups could not be calculated at the individual level due to missing data. The outcomes regarding probing depth and bone loss could not be combined because they were reported in different forms, and only one study's authors provided the original data for these parameters by online contact.

Risk of bias and methodological quality

Quality evaluation according to the NOS showed that the prospective study was rated as having 7 stars and was classified as high quality (Table 3). Based on the AHRQ criterion, the final score of the cross-sectional study was 8, and it was classified as having high quality (Table 4).

Risk of PIDs in the OLP versus non-OLP groups

Due to the small sample size and good homogeneity ($\chi^2 = 0.04$, $P = 0.84$; $I^2 = 0$) of the included literature, the fixed-effect model was employed to calculate the pooled RR in this meta-analysis. The model showed no significant difference in the number of implants suffering from PI ($RR = 1.49$, 95% CI : 0.77-2.90, $P = 0.24$, Fig 2A) or PIM ($RR = 0.88$, 95% CI : 0.53-1.46, $P = 0.61$, Fig 2B) between the OLP and non-OLP groups at the implant level. There was a similar result when combining PI and PIM into PID ($RR = 1.08$, 95% CI : 0.75-1.55, $P = 0.68$, Fig 2C). This was also the case for the presence of bleeding on probing between the two groups ($RR = 0.90$, 95% CI : 0.70-1.15, $P = 0.40$, Fig 2D).

Discussion

Key findings

The present systematic review assessed for the first time the current existing evidence on the relationship between OLP and PIDs and attempted to determine whether OLP is a potential risk factor for PIDs. Of 139 studies, two, which contained 68 participants receiving 222 (OLP vs. non-OLP, 112 vs. 110) implants, with a 12- to 120-month follow-up time, were included and evaluated as having high quality. The proportions of implants with PIDs between the OLP and non-OLP groups were as follows: 19.6% (22/112) vs. 22.7% (25/110) for PIM and 17.0% (19/112) vs. 10.9% (12/110) for PI. Existing evidence seems to suggest that OLP is not a suspected risk factor for PIDs during the 10-year follow-up period.

Overall completeness and applicability of the evidence

The diagnostic criteria for OLP were clearly stated in the two enrolled studies, while those for PIDs were absent in the cross-sectional study. Moreover, in the two enrolled studies, PIDs were evaluated at only the implant level and not at the individual level, while OLP was diagnosed at the individual level. Hence, the evidence was insufficient to determine whether OLP is a risk factor for PIDs at the individual level.

Concerning secondary outcomes of PIDs, both studies had complete bleeding on probing data. After data combination, OLP was found to not be a risk factor for bleeding on probing at the implant level. For the secondary outcomes for PIDs, i.e., probing depth and bone loss, the data were complete but reported in

different forms in the two studies. The prospective study set cutoff values for probing depth and bone loss to determine the percentages of involved implants, while the cross-sectional study directly presented the medians and ranges of the two parameters. Despite repeated contact with the authors, we obtained only the raw data of the prospective study. Hence, an attempt to combine the data of the two studies in terms of probing depth or bone loss could not be achieved. Ultimately, both studies found no significant difference in probing depth or bone loss between the OLP and non-OLP groups.

When the participant was used as the unit of analysis, the intragroup analysis of the OLP group failed to demonstrate a statistically significant relationship between the existence of desquamative gingivitis and PIM or desquamative gingivitis and PI, but 20 out of 25 implant patients with PIM were among those with desquamative gingivitis in the OLP group; hence, PIM with DG in the OLP group occurred more frequently than non-desquamative gingivitis in the OLP group in this subgroup of implant patients in the prospective study [25]. Since the cross-sectional study presented insufficient information, although the corresponding authors were contacted in various ways and only the author of the prospective study provided the original data of the parameters of interest, no subgroup analysis was performed. Therefore, subgroup combinations in the meta-analysis were not possible.

Overall quality, strength, and consistency of the evidence

The present systematic review included only two observational studies that met the inclusion criteria mainly because related research on implantation in OLP populations is scarce. However, the durations of the two enrolled studies were very similar (2003 to 2009 vs. 2005 to 2010), and the study populations were from the same country, which may be beneficial for data synthesis. Nevertheless, the results might be more applicable to specific ethnicities or countries and may not be easily generalized for people globally. The average age of the OLP patients in the two studies was quite different (64.5 vs. 53.7). This distinction may introduce uncertainty regarding the applicable age after pooling the data. Additionally, the mean follow-up time of the two studies was similar but did not exceed 5 years; therefore, it is unclear whether OLP is a risk factor for PIDs over an observation period longer than 5 years. The two studies contained detailed records of important confounders, such as smoking, but only the prospective study controlled for confounders. The response rate was not reported in either study; thus, the number of lost samples was unknown. In the prospective study, two implants in the non-OLP group were lost for unknown reasons, and the study did not state whether it was a PI-associated consequence. Even if that was the case, there was no difference in the results of the PI ($RR=1.31$, 95% *CI*: 0.70-2.45, $P = 0.40$) analysis between the two cases.

Case definitions for PIDs vary considerably in previous studies[8], and PID data are especially challenging to interpret. Only the prospective study, which reported clear definitions of PIM and PI, was evaluated according to the defined criteria [25, 26]. The cross-sectional study did not elaborate on the diagnostic criteria for PIDs; the two studies had different definitions of diseases, and there may have been some bias in the data. The diagnostic criteria for PI in the study were not uniform, and limited studies were included in the meta-analysis, so no meta-analysis could be conducted to prove the effect of the differences on the

meta-analysis outcomes. Clinical indicators such as bleeding on probing and bone loss are relatively easy to standardize, although no effect of OLP on these indicators has been observed.

The results of the statistical analysis show that the homogeneity between the results was good. However, the limited number of enrolled studies made it unfeasible to draw a funnel plot to assess the variation in the studies. Although the sample size was small, the strength of the study evidence was high as analyzed by quality-control assessment, and the overall estimate of the meta-analysis represents the best available evidence.

Implications for practice and policy

Although there was no clear association between OLP and the occurrence of PIDs, implant-related issues in OLP patients cannot be easily ignored, and several issues are worth noting. First, in a clinical study [15], the gingival index of the OLP group was calculated, and the mean value of the periodontal index and rate of bleeding on probing were higher than those in the control group, revealing that the periodontal condition in the OLP group was poor compared with that in the control group. Poor oral hygiene is a well-known risk factor for PIDs; therefore, OLP may have a certain impact on PIDs. Second, some scholars believe that the capacity of the epithelium to adhere to the titanium surface of the implants in OLP patients may be affected [14, 27] because the adhesion of epithelial cells decreases, affecting the epithelial barrier around the implant surface [28]. Third, desquamative gingivitis is a special type of OLP that causes gingival damage. An increased frequency of PIM was reported in cases of desquamative gingivitis compared with non-desquamative gingivitis OLP patients [25]. Another study demonstrated that the presence of desquamative gingivitis was associated with relatively deep periodontal pockets [29]. The study inferred that desquamative gingivitis lesions might circuitously enhance the long-term risk for periodontal diseases via plaque accumulation by impeding proper oral hygiene around natural teeth and implants. It is important to clarify the constructive association between desquamative gingivitis and PIDs in the future. Moreover, OLP is a T cell-mediated chronic inflammatory autoimmune disease[30]. PIDs in humans cause leukocytic infiltration through barrier epithelial migration and an increase in the proportion of T and B cells [31]. In this context, the two conditions might aggravate each other via inflammatory pathways. Additionally, dysbiosis might be a potential link between OLP and PIDs, given that both conditions have been related to microbial alterations [32, 33]. Finally, systemic corticosteroid treatment in OLP patients was frequently associated with decreased bone mineral density, especially during the first 6 months of corticosteroid therapy [34]. The osteoporotic effects of corticosteroids might cause more alveolar bone loss around dental implants during infection [35].

In the systematic review, the available evidence did not support OLP as a risk factor for PIDs. However, this does not mean that implant restoration can be performed indiscriminately in OLP patients. For patients with acute/erosive forms of OLP or desquamative gingivitis, immune system disorder and poor oral hygiene may be potential risk factors for PIDs. OLP should not be considered a contraindication for implant treatment in patients who do not have evident symptoms or mucosal non-erosive congestion and have good oral hygiene. Furthermore, the mental state of OLP patients might be improved after implant

treatment, which in turn is better for OLP control, considering that those without implant treatment have reported a poor quality of life [16]. Additionally, for OLP patients receiving glucocorticoid treatment, assessment of alveolar bone mineral density in the area to be implanted should be considered. Finally, it is worth noting the effect of implants in the mouth on the recovery of OLP patients, although there were no significant differences in OLP signs and symptoms between the implant group (14 patients) and nonimplant group (15 patients) during the 12-24 month follow-up period [14]. For OLP patients who have undergone implant restoration, OLP conditions may need to be monitored frequently, and implant maintenance planning may need to be personalized.

Implications for further research

This review systematically analyzed the existing evidence on OLP and PIDs for the first time. Large-scale prospective trials are required to validate the findings.

The results suggest that OLP is not a potential risk factor for PIDs during the 1 to 10-year follow-up period. However, given the relatively small amount of evidence available, the final answer to this question depends on large, well-designed prospective and randomized clinical trials in the future. At present, the proportion of people undergoing implant treatment and the prevalence of OLP worldwide are not high, which may cause difficulty in researching the correlation between the two diseases; therefore, multicenter cooperative clinical research is needed. Additionally, OLP has various clinical classifications, and the prospective study included in this systematic review showed that patients with OLP with desquamative gingivitis had a higher frequency of PIM. This suggests that in future studies, a subgroup analysis of OLP may be helpful in exploring the association between OLP and PIDs. Moreover, interventional studies on the effects of OLP treatment on peri-implant status are still lacking; these studies can further elucidate the association between OLP and PIDs. Molecular biology techniques can also help to explore the microscopic effects of OLP on peri-implant pathophysiological processes at the molecular level.

Conclusions

Available literature regarding the effects of OLP on PIDs remains very limited. Existing evidence seems not support OLP as a suspected risk factor for PIDs. Large-scale prospective trials are required to test the findings.

Abbreviations

OLP	oral lichen planus
PIDs	peri-implant diseases
RR	risk ratio
CI	confidence interval
PIM	peri-implant mucositis
PI	peri-implantitis
PRISMA	Preferred Reporting Items for Systematic Review and Meta-analyses
NOS	Newcastle-Ottawa Scale
AHRQ	Agency for Healthcare Research and Quality

Declarations

Acknowledgements

The authors thank Liting Yan and Yinshen Yang for their help with meta-analysis work.

Funding

This study was supported by the National Natural Science Foundation of China, Beijing, China, Grants 81700985 and 81271159. The role of funding agencies in research: data collection, analysis, and manuscripts.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

XX and TY formulated inclusion and exclusion criteria, literature search and screening. XX and TY analyzed and interpreted the data and drafted the manuscript. XW, WQ and LG designed and supervised the study and edited and gave final approval for the manuscript to be published. All authors read and approved the final manuscript.

Ethics and consent to participate

Not Applicable

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Author details

Xiaoqin Xiong 1†, MD, Dental specialist in Periodontology and Oral Medicine; Tiantian Xu 1†, MD, Dental specialist in Periodontology; Xinhong Wang 1, DMD, Associate professor, Dental specialist in Oral Medicine; Wenguang Qin 1, MD, Dental specialist in Periodontology; Gang Luo 1*, DMD, Professor; Ting Yu 1*, DMD, Associate professor, Dental specialist in Periodontology

1 Department of Periodontology and Oral Medicine, Stomatology Hospital of Guangzhou Medical University, Guangzhou, China.

† Xiaoqin Xiong and Tiantian Xu are first authors.

* Correspondence: Ting Yu, Key Laboratory of Oral Medicine, Guangzhou Institute of Oral Disease, Department of Periodontology and Oral Medicine, Stomatology Hospital of Guangzhou Medical University, NO.195 Dongfeng West Road, Guangzhou 510140, China, Tel.: +86-020-81299940. E-mail: dent_yu@163.com

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Tables

Table 1. Electronic databases used and search strategies.

Database	Search strategy	Items found
Medline	#1 ([MeSH Terms] OR [Title/Abstract]) AND #2 ([MeSH Terms] OR [Title/Abstract])	32
Web of Science	TS= #1 AND TS= #2	60
Cochrane Library	#1:ti, ab, kw AND #2:ti, ab, kw	5
John Wiley	"#1" anywhere AND "#2" in Abstract	3
Springer	#1 AND #2	28
Scopus	TITLE-ABS-KEY (#1) AND TITLE-ABS-KEY (#2)	3
Manual search	#1 AND #2	8 (JP: 1;CIDRR: 3; COIR: 1; JCP: 1; COI: 1; JD: 1)

#1: peri-implantitis OR peri-implant disease OR peri-implant mucositis OR dental implant OR implant; #2: OLP OR (oral lichen planus)

IP: Journal of Periodontology; CIDRR: Clinical Implant Dentistry and Related Research; COIR: Clinical Oral Implants Research; JCP: Journal of Clinical Periodontology; COI: Clinical Oral Investigations; JD: Journal of Dentistry

Table 2. General description of the included studies.

Gonzalo Hernandez (2011)	Pia López-Jornet (2012)
Median (range) 53.7 (38-73)/52.2 (35-70)	Median (range) 64.5 (44-76)/42 (29-79)
18/18	16/16
10/26	14/18
56/60	56/50
Clinical and histopathological criteria of OLP according to the modified WHO diagnostic criteria of OLP	OLP diagnosis was based on a thorough clinical examination and histopathology of the lesions
The presence of PIM (BoP, PD $\geq$ 4 mm and no BL) and PI (BoP or pus, BL $\geq$ 3 threads at the final examination)	-
Median (IR): 56.5 (22)/52.5 (22.7)	Median (range): 42 (12-120)/48 (24-48)
PD (mm): n:<4 mm 23/18; $\geq$ 4 mm 33/42	PD (mm): median (range): 3.00 (1.12-4.90)/3.00 (2-5)
(sites): 105/114; (implants): 36/44; patients: 13/16	(No. implants): 12/11;
BL (mm): $\leq$ 1.7 18/22; 1.8-2.4 24/24; 2.5-3 8/9; 3.1-3.6 4/3; $\geq$ 3.7:2/2	(No. of implants): 10/8;
12/16	10/9
5/4	14/8
Smoking	Age, sex, smoking, alcohol consumption, frequency of tooth brushing
Lichen planus was not a prominent local factor in the genesis of implant failure.	Implants did not influence manifestations of OLP.
	OLP was not a risk factor for peri-implantitis.
-	1.32 (PI)
-	0.81-2.14 (PI)
.254 (PI)/.985 (PIM)	.257 (PI)

M/F, male/female; OLPG, oral lichen planus group; CG, control group; BL, bone loss; BOP, bleeding on probing; CAL, clinical attachment level; PD, probing depth; PI, peri-implantitis; PIM, peri-implant mucositis; S, systemic diseases.

Table 3. Quality assessment of the prospective study.

	Selection	Comparability	Exposure	Score	Quality
Study	(max 4 asterisks)	(max 2 asterisks)	(max 3 asterisks)		
Gonzalo Hernandez (2011)	***	**	**	7	Low risk

Table 4. Quality assessment of the cross-

sectional study.

Items	Yes	No	Unclear
the information (survey, record review)	1		
clusion and exclusion criteria for exposed and unexposed subjects (cases and controls) or refer to previous .tions	1		
e time period used for identifying patients	1		
e whether or not subjects were consecutive if population-based	1		
5) Indicate if evaluators of study subjects were blinded to other aspects of the status of the participants	1		
6) Describe any assessments undertaken for quality assurance purposes (e.g., test/retest of primary outcome measurements)	1		
7) Explain any patient exclusions from analysis			0
re how confounding was assessed and/or controlled	1		
cable, explain how missing data were handled in the analysis			0
alize patient response rates and completeness of data collection			0
what follow-up, if any, was expected and the percentage of patients for which incomplete data or follow-up	1		
obtained			

Figures

Figure 1. Study screening process.

Figure 1

Study screening process.

Figure 2

Forest plot of the meta-analysis of PI (A), PIM (B), PIDs (C) and BOP (D). #CI, confidence interval.