Recognition and treatment of peri-implant mucositis: Do we have the right perception? A structured review

Laura Lo Bianco[1,A,C–E], Marco Montevecchi[1,D,E], Michele Ostanello[1,B,D], Vittorio Checchi[2,D–F]

[1] Department of Biomedical and Neuromotor Sciences, Dental School, University of Bologna, Italy
[2] Unit of Dentistry and Oral & Maxillofacial Surgery, Surgical, Medical and Dental Department of Morphological Sciences related to Transplant, Oncology and Regenerative Medicine, University of Modena and Reggio Emilia, Modena, Italy

A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation;
D – writing the article; E – critical revision of the article; F – final approval of the article

Dental and Medical Problems, ISSN 1644-387X (print), ISSN 2300-9020 (online) *Dent Med Probl.* 2021;58(4):545–554

Address for correspondence
Laura Lo Bianco
E-mail: laura_lo_bianco@hotmail.com

Funding sources
None declared

Conflict of interest
None declared

Received on January 16, 2021
Reviewed on April 28, 2021
Accepted on May 4, 2021

Published online on December 28, 2021

Abstract

Peri-implant mucositis is a common inflammatory lesion of the soft tissues surrounding endosseous implants, with no loss of the supporting bone. Its prevention or early diagnosis are vital for dental implant success.

The aim of this review was to investigate knowledge strengths and gaps in clinicians' perceptions of peri-implant mucositis prevalence and evidence for successful treatment.

A literature search for articles published until 2020, reporting on the prevalence of peri-implant mucositis and its treatment was performed in standard online databases. The inclusion criteria were as follows: studies in English; studies with an available abstract; studies on humans with at least 1 dental implant; and studies reporting on the prevalence and/or treatment of peri-implant mucositis. Sixty-five studies fulfilled the inclusion criteria. The included papers were analyzed to identify data on the prevalence and treatment of peri-implant mucositis. The prevalence statistics for peri-implant mucositis had wide ranges in both the patient-based (PB) analysis and the implant-based (IB) analysis; the possible reasons for these wide ranges are discussed. Treatment methods for peri-implant mucositis were analyzed individually and compared to the management of gingivitis.

It was determined that the currently available information on the prevalence rates and the standardized therapeutic protocols for peri-implant mucositis are insufficient. Since the mean gingivitis and peri-implant mucositis prevalence rates in the PB analysis were similar, it is possible that peri-implant mucositis is underestimated due to variables related to implant rehabilitation itself.

Keywords: inflammation, dental implant, literature review, oral mucositis, peri-implant healing

Cite as
Lo Bianco L, Montevecchi M, Ostanello M, Checchi V. Recognition and treatment of peri-implant mucositis: Do we have the right perception? A structured review. *Dent Med Probl.* 2021;58(4):545–554. doi:10.17219/dmp/136359

DOI
10.17219/dmp/136359

Introduction

Dental implants are widely used for oral rehabilitation. They are biocompatible prosthetic devices implanted in living bone and, for this reason, the peri-implant tissue conditions can change over time.[1,2] Healthy peri-implant tissues are characterized by the absence of erythema, bleeding on probing (BoP), swelling, and suppuration.[3]

Once osteointegration has been achieved, allowing for the healing time after implant insertion, implant complications can occur due to mechanical problems, inflammation and/or the loss of the surrounding tissues (the oral mucosa and the supporting bone). These could lead to relevant discomfort for the patient as well as implant failure over time.[4] After osteointegration has occurred, implants may become contaminated and peri-implant tissues could become inflamed, causing peri-implant mucositis and/or peri-implantitis.[3]

In an animal study on beagle dogs, Berglundh et al. compared the anatomy and histology of peri-implant and periodontal tissues in block biopsies.[5] A histological examination showed that both presented well-keratinized areas (the oral epithelium and the outward portion of the peri-implant mucosa), but periodontal tissues presented only a few cells of thick epithelium in contact with the implant abutment. Also, peri-implant tissue fibers displayed a parallel course originating from the crestal bone, while periodontal tissue fibers were perpendicular to the dental root, going from the root cementum to the alveolar bone.[5]

Likewise, blood supply differed from an anatomical point of view – the peri-implant bone vasculature consisted only in the periosteum source, while gingival supply was guaranteed by a double source composed of supraperiosteal and periodontal ligament vessels.[6]

Being aware of histological differences between peri-implant tissues and the periodontium is fundamental to better understand the peri-implant tissue biology. Clinicians, implant-rehabilitated patients and the dental industry have mainly based their maintenance approaches on the techniques and tools derived from the pre-implant era.

Peri-implant mucositis is an inflammatory lesion of the soft tissues surrounding an endosseous implant, with no loss of the supporting bone or the continuing marginal bone.[7] Conversely, peri-implantitis is described as a pathological condition occurring in tissues around dental implants that is characterized by inflammation in the peri-implant connective tissue and a progressive loss of the supporting bone.[8]

The etiology of peri-implant mucositis has been described as the accumulation of bacterial biofilm around the implant, which may cause signs and symptoms of inflammation, such as local swelling, redness, pain, and BoP.[7] The diagnosis of peri-implant mucositis vs. peri-implantitis is made by the evidence of pathological bone loss.[9] While peri-implant mucositis exhibits signs of inflammation with no bone loss besides the remodeling process of the alveolar bone during the first year after implantation, peri-implantitis shows signs of inflammation associated with a further loss of the crestal bone.[3,7] In recent years, there has been a general consensus that following the first year of implant functioning, bone loss around dental implants ≥2 mm represents peri-implantitis.[10]

Data indicates that patients diagnosed with peri-implant mucositis may develop peri-implantitis, especially in the absence of regular maintenance care, but the processes and reasons for this pathological progress remain unknown.[11] Factors associated with peri-implant mucositis include biofilm accumulation, smoking and radiation therapy.[7] Regular supportive peri-implant care with biofilm removal is an important preventive strategy against the conversion of a healthy tissue to peri-implant mucositis, and also against the progression of peri-implant mucositis into peri-implantitis.[7,11]

There is evidence that peri-implant mucositis can be successfully treated. The resolution of the clinical signs of inflammation may take more than 3 weeks following the restoration of plaque/biofilm control.[11] The management of peri-implant inflammation should be addressed in terms of infection control, decontamination of the implant surface and regeneration of the alveolar bone when needed.[12]

The early diagnosis and prevention of peri-implant infections are essential for the long-term dental implant success. In order to perform a thorough evaluation of the peri-implant conditions, peri-implant probing and relative radiographs are always required.[12,13]

The purpose of this review was to highlight possible clinicians' perception problems related to peri-implant mucositis, to investigate the prevalence of peri-implant mucositis reported in the literature and to analyze the evidence-based data regarding its treatment.

Material and methods

Focus question

The focus question for the literature search was: "What is the clinician's perception regarding the prevalence levels and treatment strategy efficacy/evidence for peri-implant mucositis?"

It was structured according to the PICO format[14]:

– Population: patients rehabilitated with dental implants;
– Intervention: implant prosthesis, peri-implant tissue, and peri-implant mucositis prevalence and treatment;
– Comparison: diagnostic criteria and peri-implant mucositis treatment;
– Outcome: finding consistency between prevalence and perception, and differences between various kinds of treatment.

Search strategy

The PubMed/MEDLINE, Embase, Scopus, Web of Science, and Cochrane databases were searched to identify published articles reflecting the inclusion criteria: studies in English; studies with an available abstract; studies involving humans with at least 1 dental implant; and studies reporting data on the prevalence and/or treatment of peri-implant mucositis. The search strategy was divided into 2 parts: a pre-search to avoid discrepancies between findings due to the device used (a personal computer or a mobile device); and a focus question search.

The pre-search was used to determine the device and keywords that provided the greatest number of results in order to establish the focus question search. The pre-search concerned peri-implant mucositis studies published up to 2020. The terms used for the identification of keywords were: 'peri implant' OR 'peri-implant' OR 'peri-implant mucositis' AND 'mucositis'.

The focus question search was carried out on a personal computer to analyze the abovementioned databases, using the 2 keywords that yielded the greatest number of results in the pre-search. The focus question search concerned peri-implant mucositis studies published up to 2020. The terms used for the identification of keywords were: 'peri implant mucositis' OR 'peri-implant mucositis' AND 'prevalence' OR 'treatment'.

The focus question search yielded 99 articles for "peri implant mucositis prevalence", 99 for "peri-implant mucositis prevalence", 300 for "peri implant mucositis treatment", and 271 for "peri-implant mucositis treatment".

Screening and selection

The inclusion criteria were as follows: studies in English; studies with an available abstract; studies involving humans with at least 1 dental implant; and studies reporting on the prevalence and/or treatment of peri-implant mucositis.

The exclusion criteria were as follows: studies in a language other than English; studies without an available abstract; non-clinical studies; studies without dental implants; and studies reporting on neither the prevalence nor the treatment of peri-implant mucositis.

Once the studies were selected according to the abovementioned initial screening, only those fitting the following categories were included: randomized clinical trials (RCTs); controlled clinical trials (CCTs); cohort studies; cross-sectional studies; and case–control studies.

The studies were first screened by titles and abstracts, and examined by 2 reviewers. The full text of the selected articles was retrieved and the study results were analyzed. Review articles and systematic reviews were also studied in order to find other articles that did not emerge during database inquiries.

Full-text studies admitted for final analysis were divided into 2 groups: the prevalence group; and the treatment group.

Results

Sixty-five studies fulfilled all the inclusion criteria: 25 RCTs; 3 CCTs; 15 cohort studies; 20 cross-sectional studies; and 2 case–control studies. All these studies were divided into 2 main groups according to the 'prevalence' (n = 34) or 'treatment' (n = 31) Medical Subject Headings (MeSH). The results according to the type of study are shown in Table 1 for the prevalence group and in Table 2 for the treatment group.

In the prevalence group, cohort and cross-sectional studies constituted the majority of the research devoted to peri-implant mucositis (Table 1). In cohort studies, the peri-implant mucositis prevalence rates ranged between 7.14% and 68.00% in the patient-based (PB) analysis (referring to the number of patients included in the analysis), and between 5.06% and 38.00% in the implant-based (IB) analysis (referring to the number of implants included in the analysis). In cross-sectional studies, the peri-implant mucositis prevalence ranges varied from 20.80% to 80.90% in the PB analysis, and from 21.00% to 90.00% in the IB analysis (Table 3).

In the treatment group, there were RCTs, CCTs, cohort studies, and 1 case–control study (Table 2). The search found 1 RCT on the use of sodium hypochlorite gel, 1 RCT about the modification of the prosthesis, 1 RCT on the use of a drying agent associated with manual debridement, 2 RCTs in which chlorhexidine gel was used, 1 RCT

Table 1. Prevalence group results according to the type of study

Type of study	Number of articles
RCTs	0
CCTs	0
Cohort studies	13
Cross-sectional studies	20
Case–control studies	1

RCT – randomized clinical trial; CCT – controlled clinical trial.

Table 2. Treatment group results according to the type of study

Type of study	Number of articles
RCTs	25
CCTs	3
Cohort studies	2
Cross-sectional studies	0
Case–control studies	1

Table 3. Peri-implant mucositis prevalence ranges according to the type of study

Type of study	Prevalence range [%]	
	PB analysis	IB analysis
Cohort studies	7.14–68.00	5.06–38.00
Cross-sectional studies	20.80–80.90	21.00–90.00

PB – patient-based; IB – implant-based.

Table 4. Treatment proposed with regard to the type of study

Type of study	Treatment tested
RCTs, CCTs and a case–control study	sodium hypochlorite gel
	modifying the prosthesis
	desiccant agent
	chlorhexidine gluconate
	cetylpyridinium
	triclosan
	chitosan brushes
	probiotics
	diode laser
	photodynamic therapy
	air polishing
	enamel matrix derivative
	ozone
	hydrogen peroxide
	systemic antibiotics
	azithromycin
	mechanical curettage
Cohort studies	non-surgical therapy

in which a mouth rinse with 0.03% chlorhexidine and 0.05% cetylpyridinium was assessed, 1 RCT that investigated 0.12% chlorhexidine gluconate, 3 RCTs in which toothpastes containing triclosan were assessed, 1 RCT in which chitosan brushes were used, 5 RCTs about probiotics (in one of the studies, photodynamic therapy was added to probiotic administration), 2 RCTs about photodynamic therapy, 3 RCTs about air polishing, 1 RCT in which an enamel matrix derivative was used, 1 RCT on the use of ozone and/or hydrogen peroxide, and 2 RCTs in which systemic antibiotics supported mechanical debridement. The 2 cohort studies were about mechanical debridement and biofilm control (Table 4).

The selected studies proposed various kinds of treatment, including sodium hypochlorite gel, a desiccant agent, chlorhexidine, triclosan, chitosan brushes, probiotics, diode laser therapy, photodynamic therapy, air polishing, and antibiotics. Most of these consisted of mechanical debridement combined with an additional therapy, such as sodium hypochlorite gel, a desiccant agent, chlorhexidine, probiotics, photodynamic therapy, an enamel matrix derivative, and systemic azithromycin (Table 5).

Table 5. Treatment proposed and related results and conclusions

Treatment	Authors, year of publication	Study type	Study description	Sample size	Implant number	Results	Conclusions
Sodium hypochlorite gel	Iorio-Siciliano et al. 2020[28]	triple-blind RCT 6-month follow-up	mechanical debridement with sodium hypochlorite gel (test group) vs. mechanical debridement with placebo gel (control group)	46	68	PPD decreased in both the test and control groups ($p = 0.0001$ and $p = 0.0001$, respectively)	a complete resolution was not achieved with either therapy
Modifying the implant-supported prosthesis	de Tapia et al. 2019[24]	RCT 6-month follow-up	modifying the prosthesis to allow better oral hygiene (test group) or not (control group)	45 test – 24 control – 21	145	changes in mBI in the test and control groups were 1.14 and 0.50, respectively ($p = 0.010$), in PPD – 0.31 mm and 0.02 mm, respectively ($p = 0.040$)	modifying the prosthesis improved clinical outcomes
Topical desiccant agent in association with manual debridement	Lombardo et al. 2019[29]	RCT	desiccant agent after debridement (test group) vs. 1% chlorhexidine after debridement (control group)	23	52	the test group presented significantly greater reductions in BoP, mBI, VPI, and mPI than the control group	a complete resolution of the inflammatory conditions was not achieved by either group
Chlorhexidine-containing brush-on gel	Hallström et al. 2017[42]	double-blind RCT 12-week follow-up	chlorhexidine-containing brush-on gel used as an adjuvant to mechanical debridement	37	37	the test group presented a reduction in BoP after 4 and 12 weeks as compared to the control group ($p < 0.05$)	the findings indicate moderate but significant improvement in clinical parameters
Chlorhexidine gel	Heitz-Mayfield et al. 2011[46]	RCT	non-surgical debridement with/without 0.5% chlorhexidine gel	29 test – 15 control – 14	29	at 1 month and from 1 to 3 months, there were statistically significant reductions in the mean number of sites with BoP and the mean PPD values at implants in both groups	adjunctive chlorhexidine gel did not improve the results as compared to mechanical cleaning alone

Treatment	Authors, year of publication	Study type	Study description	Sample size	Implant number	Results	Conclusions
0.03% chlorhexidine and 0.05% cetylpyridinium mouth rinse	Pulcini et al. 2019[30]	double-blind RCT 12-month follow-up	0.03% chlorhexidine and 0.05% cetylpyridinium mouth rinse vs. placebo mouth rinse	46 test – 24 control – 22	54	a reduction in BoP in the test group ($p = 0.002$) and the control group ($p > 0.05$)	the use of the test mouth rinse demonstrated some adjunctive benefits in peri-implant mucositis treatment
0.12% chlorhexidine gluconate	Menezes et al. 2016[39]	RCT 6-month follow-up	basic periodontal therapy with 0.12% chlorhexidine gluconate mouthwash vs. basic periodontal therapy and placebo mouthwash	37	119 test – 61 control – 58	significant improvement in comparison with baseline, no significant differences between the treatment groups	0.12% chlorhexidine was not more effective than placebo
Triclosan dentifrice	Ramberg et al. 2009[26]	double-blind RCT 6-month follow-up	dentifrice containing triclosan vs. sodium fluoride dentifrice	60	N/A	subjects with peri-implant mucositis who used a 0.3% triclosan dentifrice exhibited significantly fewer clinical signs of inflammation than subjects who used a regular fluoride dentifrice	the regular use of triclosan dentifrice may reduce the clinical signs of inflammation
Triclosan-containing fluoride toothpaste	Pimentel et al. 2019[31]	RCT two 3-week follow-ups	triclosan/ fluoride toothpaste vs. fluoride toothpaste	26	N/A	both groups showed increases in PI ($p = 0.001$)	triclosan-containing toothpaste reduced the RANKL/OPG ratio
Triclosan-containing toothpaste	Ribeiro et al. 2018[35]	RCT 6-week follow-up	triclosan/ copolymer/ fluoride toothpaste vs. placebo fluoride toothpaste	22	22	both groups showed increases in PI at implant sites from the 3rd to the 21st day, avoiding an increase in BoP throughout the follow-up was possible only with triclosan treatment	triclosan-containing toothpaste controls the clinical signs of inflammation
Chitosan brush	Wohlfahrt et al. 2019[25]	RCT 6-month follow-up	chitosan brush on an oscillating dental handpiece vs. titanium curette	11	24	both groups demonstrated significant reductions in BoP between baseline and 6 months	a chitosan brush seems to be a safe and efficient device for the debridement of dental implants
Probiotics	Galofré et al. 2018[20]	triple-blind RCT	oral probiotic *L. reuteri* as an adjuvant to non-surgical mechanical therapy	44 with peri-implant mucositis – 22 with peri-implantitis – 22	44	a decrease of *P. gingivalis* bacterial load at implant sites with mucositis ($p = 0.031$)	the probiotic together with mechanical therapy produced additional improvement over treatment with mechanical therapy alone
Probiotics	Peña et al. 2019[32]	triple-blind RCT 3-month follow-up	mechanical debridement with 0.12% chlorhexidine and *L. reuteri* vs. mechanical debridement with 0.12% chlorhexidine	50	50	after the administration of 0.12% chlorhexidine, all clinical parameters improved in both groups	the administration of the probiotic did not seem to provide an additional clinical benefit
Probiotics	Hallström et al. 2016[43]	double-blind RCT 26-week follow-up	probiotic supplements as an adjuvant to conventional management vs. placebo	49	N/A	after 4 and 12 weeks, BoP and PPD significantly decreased in both groups ($p < 0.05$), no significant differences between the treatment groups	probiotic supplements did not provide additional improvement over placebo

Treatment	Authors, year of publication	Study type	Study description	Sample size	Implant number	Results	Conclusions
Probiotics	Flichy-Fernàndez et al. 2015[44]	double-blind RCT	*L. reuteri*	34	77	after treatment with the probiotic, patients with mucositis and without peri-implant disease showed improvement in clinical parameters, with reductions in cytokine levels	clinical parameters improved after treatment with the probiotic
Probiotics with photodynamic therapy	Mongardini et al. 2017[38]	RCT 6-week follow-up	*L. reuteri* with professionally administered plaque removal and photodynamic therapy	20	20	no significant differences in clinical outcomes between the treatment groups	the adjunctive use of the probiotic did not significantly improve clinical outcomes
Mechanical curettage with photodynamic therapy	Javed et al. 2017[21]	RCT 12-week follow-up	mechanical curettage with/without adjunctive antimicrobial photodynamic therapy	54 test – 28 control – 26	N/A	PI and PPD were significantly higher in the control group ($p < 0.001$)	mechanical debridement with photodynamic therapy is more effective in the treatment of peri-implant mucositis in comparison with mechanical debridement alone
Antimicrobial photodynamic therapy	Al Rifaiy et al. 2018[34]	RCT 12-week follow-up	mechanical debridement and photodynamic therapy (test group) vs. mechanical debridement (control group)	38	65	reductions in PI ($p < 0.001$) and PPD ($p < 0.001$) in the test group as compared to the control group	antimicrobial photodynamic therapy is more effective in comparison with manual debridement alone
Low-abrasive air polishing	Al Ghazal et al. 2017[36]	single-blind RCT	low-abrasive air polishing vs. debridement with titanium curettes	18 test – 9 control – 9	25 test – 15 control – 10	no difference in BoP between the groups ($p = 0.350$)	both treatment methods were proven to be effective in reducing peri-implant inflammation
Air-abrasive debridement	Lupi et al. 2017[40]	RCT 6-month follow-up	maintenance treatment with glycine powder air-abrasive debridement vs. manual debridement and chlorhexidine administration	46	88	air-abrasive debridement significantly improved PI, BoP, PPD, and BS ($p < 0.05$)	treatment with glycine powder seems to be more effective than traditional treatment with plastic curettes and chlorhexidine
Air polishing	Riben-Grundstrom et al. 2015[41]	RCT	glycine powder air polishing vs. ultrasonic debridement	37	37	at 12 months, there were statistically significant reductions in the mean PI, BoP and the number of periodontal pockets ≥4 mm within the treatment groups in comparison with baseline	non-surgical treatment with air polishing or ultrasonic debridement is effective
Enamel matrix derivative	Kashefimehr et al. 2017[37]	double-blind RCT 3-month follow-up	mechanical debridement with enamel matrix derivative vs. mechanical debridement alone	41	41	significant improvement in terms of BoP and PPD in the test group as compared to the control group ($p < 0.0001$)	complete recovery was not observed using either treatment approach
Subgingival ozone and/or hydrogen peroxide	McKenna et al. 2013[45]	double-blind RCT	effect of subgingival ozone and/or hydrogen peroxide on the development of peri-implant mucositis	20	80	significant differences in plaque and modified gingival and bleeding indices were observed between various kinds of treatments	ozone showed great potential for the management of peri-implant mucositis

Treatment	Authors, year of publication	Study type	Study description	Sample size	Implant number	Results	Conclusions
Systemic antibiotics	Hallström et al. 2012[27]	RCT 6-month follow-up	non-surgical treatment of peri-implant mucositis with/without systemic antibiotics	48	N/A	the statistical analysis failed to demonstrate differences in PPD at 6 months	no short-term differences were found between the 2 study groups; the study does not provide evidence for the beneficial effect of systemic antibiotics
Azithromycin	Gershenfeld et al. 2018[33]	RCT 6-month follow-up	mechanical debridement and systemic azithromycin vs. mechanical debridement and placebo	17 test – 9 control – 8	66	the treatment patients showed a consistently greater reduction of gingival inflammation and improvement in soft tissue healing than the control patients	the adjunctive use of azithromycin can assist in the control of peri-implant mucositis
Mechanical debridement	Serino et al. 2018[47]	7-month prospective cohort study	effect of submucosal mechanical instrumentation following supramucosal plaque removal	44	175	at 1 month following supramucosal plaque removal, the number of treated implants with BoP was reduced with a concomitant decrease in the mean PPD value, following submucosal instrumentation, a further reduction in BoP was recorded with a concomitant reduction in the mean PPD value at the 7-month examination	improvement in the clinical condition appeared to be in a large extent due to supramucosal plaque removal
Biofilm control	Gomes et al. 2015[48]	longitudinal cohort study	comparison of the gingival and peri-implant mucosal inflammatory response to mechanical biofilm control	22	N/A	VPI, mPI and gingival bleeding indexes reduced from day 0 onward	supragingival/ supramucosal biofilm control benefited both the teeth and the implants
Photodynamic therapy	Zeza et al. 2018[49]	CCT	professionally administered plaque removal and photodynamic therapy	20	20	a reduction in the median number of BoP sites around implants from3.5 to 2.0 ($p = 0.030$)	peri-implant mucositis can be effectively treated with photodynamic therapy
Mechanical debridement and photodynamic therapy	Al Amri et al. 2016[50]	CCT	mechanical debridement with/without photodynamic therapy in the treatment of peri-implant inflammation in T2DM patients	67 test – 34 control – 33	N/A	BoP and PPD were significantly lower in the test group than in the control group at all follow-ups	in patients with T2DM, mechanical debridement with adjunctive antimicrobial photodynamic therapy is more effective in the treatment of peri-implant inflammation in comparison with mechanical debridement alone
Mechanical debridement paired with diode laser application	Lerario et al. 2016[51]	CCT	conventional treatment with diode laser application (test group) vs. conventional treatment alone (control group)	27	N/A	a reduction of pathological sites from 89% to 14.35% in the test group and from 75.69% to 50% in the control group	diode laser seems to be a valuable tool for peri-implant mucositis treatment
DMT	Chan et al. 2019[52]	case–control study	assessing the modifying effect of DMT on the induction and resolution phases of experimental peri-implant mucositis at DMT ≥ 3 mm (case) and DMT ≤ 1 mm (control)	19	N/A	the removal of the crown and professional submucosal cleaning were necessary to revert to the baseline gingival index in the tested implant	a longer mucosal tunnel results in a much more difficult resolution of peri-implant mucositis

L. reuteri – *Lactobacillus reuteri*; T2DM – type 2 diabetes mellitus; DMT – depth of the implant mucosal tunnel; N/A – data not available; PPD – probing pocket depth; mBI – modified bleeding index; BoP – bleeding on probing; VPI – visible plaque index; mPI – modified plaque index; PI – plaque index; RANKL/OPG – receptor activator of nuclear factor kappa B ligand/osteoprotegerin; *P. gingivalis* – *Porphyromonas gingivalis*; BS – bleeding score.

Discussion

The prevalence data found in this literature review revealed a wide gap in percentage ranges. This could be due to the relevant heterogeneity of the prevalence reported among the 13 cohort studies and 20 cross-sectional studies. Other aspects to consider in order to explain this gap are the sample size and the population observed. Some articles addressed a population composed of smokers or subjects affected by diabetes mellitus; both smoking and diabetes mellitus are well-known periodontal risk factors.

Comparing the results of this review regarding the prevalence of peri-implant mucositis to the prevalence of gingivitis provided by the U.S. National Center for Health Statistics (38.70% PB), a tight overlap can be observed.[9,15]

According to the available data, the average prevalence values for gingivitis and peri-implant mucositis look very similar. This observation is in contrast with the results of a recent study investigating clinical and biological responses in experimental gingivitis and peri-implant mucositis in humans.[16] Although less biofilm accumulation was observed at the implant sites, the peri-implant mucosa yielded a higher proportion of BoP sites as compared to the gingiva.[16] This result probably indicates that less visible plaque accumulation is needed for peri-implant mucositis to develop and that the lack of keratinized gingiva, which is a frequent condition around implants, leading to a weaker seal, can contribute to biofilm migration. This would make the onset and progression of peri-implant mucositis easier and faster than in the case of gingivitis. A possible explanation of this discrepancy is that signs of peri-implant mucositis are generally rarely identified because of the great morphological variability of the overhanging prosthesis.

With regard to prosthodontics, it must be emphasized that it definitely plays a crucial role in mucosal homeostasis. Design, structural connections and constituent materials are all factors concretely correlated to plaque accumulation and the soft tissue response. This heterogeneity may help explain the wide gap in peri-implant mucositis percentage ranges found in this review.

During the present investigation, a general deficiency of the available data on this topic emerged, suggesting more focused research is needed in the future, with a general recommendation for more detailed information in the upcoming studies about peri-implant mucositis.

Another relevant aspect concerns the varying clinical indicators used by different studies. Plaque index (PI), BoP, probing pocket depth (PPD), and marginal recession are not always accompanied by radiological examinations to exclude the presence of peri-implantitis. Therefore, it is advisable to collect all the biometric parameters of signs of inflammation, such as redness, swelling, bleeding, and suppuration, and support them with periodontal indices (BoP and PPD) and radiographic examinations.[17]

These limitations are stressed and partially addressed by the 2017 classification of periodontal and peri-implant diseases and conditions.[18] It is literally cited that "a local dot of bleeding resulting from probing may be the result of a traumatic probing that should not be considered, in the absence of other inflammatory changes, a definitive criterion to characterize a peri-implant soft tissue lesion".[8] For a correct examination, it is consequently crucial to perform circumferential peri-implant probing, using the walking probe method, and to collect all clinical and radiographic parameters to evaluate them as a whole before formulating a diagnosis.

Therefore, considering that attaining a peri-implant mucositis diagnosis seems more complex than a gingivitis diagnosis, the above reported similar prevalence data leads one to presume that the peri-implant mucositis prevalence rates might be underestimated, resulting in a lower clinical perception of this pathology.

Peri-implant mucositis treatment protocols should focus on infection control and the decontamination of the implant surface. Bacterial plaque and calculus must be professionally removed, and the patient must be instructed and motivated to perform proper oral hygiene procedures at home. While gingivitis treatment could achieve restitutio ad integrum through professional hygiene care, mechanical debridement and comprehensive home care, peri-implant mucositis treatment appears more complex, requiring several treatment modalities and devices. Many treatment procedures are performed in association with mechanical debridement, using ultrasonic devices with dedicated polyetheretherketone-coated tips and implant-friendly instruments, such as titanium-coated, carbon-fiber, teflon, and plastic curettes. Also, air-abrasive devices or lasers can be used in conjunction with local antibiotics or antiseptics.[11,19]

In the treatment of gingivitis, scaling and periodontal debridement are able to remove bacterial plaque and calculus from the tooth surfaces, allowing proper healing. None of the proposed therapies for peri-implant mucositis presented in this review led to a complete or strongly predictable resolution, but mechanical debridement accompanied by an adjunctive therapy, such as probiotics, chlorhexidine or photodynamic therapy, proved to provide additional improvement over mechanical debridement alone.[20,21] Galofré et al. compared the effect of the oral probiotic *Lactobacillus reuteri* as an adjuvant to non-surgical mechanical therapy.[20] In their triple-blind RCT, oral probiotics and mechanical therapy together produced additional improvement over treatment with mechanical therapy alone.[20] Also, Javed et al. investigated the outcome of mechanical curettage with or without the adjunct of antimicrobial photodynamic therapy.[21] Forty-four patients were involved in this RCT study, and after 12 weeks of follow-up, mechanical debridement with photodynamic therapy was determined to be more

effective in the treatment of peri-implant mucositis as compared with mechanical debridement alone.[21]

Another promising proposed treatment modality is the use of glycine powder air-polishing devices, which were demonstrated to be as effective as mechanical debridement in a study by Schwarz et al.[22] The same study group, after an electronic and manual search, selected 7 studies which showed that other therapies added to professionally administered plaque removal were quite promising.[23]

A proper prosthetic design that allows good oral hygiene and low plaque accumulation is certainly a key factor in the prevention of peri-implant mucositis. De Tapia et al. reported that when peri-implant tissue inflammation occurs, the prosthetic design should be assessed and modified if necessary to correct the design defects which may be impeding proper hygiene as well as to diminish biomechanical stress factors if involved.[24] A recent RCT compared peri-implant mucositis treatment through chitosan brushes on oscillating handpieces and titanium curettes; a chitosan brush seems to be a safe and efficient device for the debridement of dental implants.[25] Likewise, the regular use of a toothpaste containing triclosan appears to be able to reduce the clinical signs of inflammation in the mucosa adjacent to dental implants.[26] Finally, it has been shown that there is a minimal difference between the non-surgical treatment of peri-implant mucositis with and without systemic antibiotics.[27]

Conclusions

Currently, the available information on the prevalence rates and the standardized therapeutic protocols for peri-implant mucositis are insufficient. Also, it can be presumed that the prevalence rates may be underestimated due to difficulty with making a clinical diagnosis, leading to a lower level of perception among practitioners.

Peri-implant mucositis is a frequently encountered condition. The absence of effective standardized therapeutic procedures that would result in an empirical choice of therapeutic modalities may lead to diminished effectiveness and unsatisfactory treatment outcomes.

It has to be emphasized that implant placement and prosthetic restorations must allow for proper cleaning and plaque control to prevent peri-implant mucositis.

Further research is needed to improve clinicians' skills in the detection of peri-implant mucositis and to determine effective standardized therapies.

ORCID iDs

Laura Lo Bianco https://orcid.org/0000-0001-9629-2455
Marco Montevecchi https://orcid.org/0000-0001-7312-802X
Michele Ostanello https://orcid.org/0000-0002-2781-574X
Vittorio Checchi https://orcid.org/0000-0002-3053-5562

References

1. Checchi V, Mazzoni A, Breschi L, Felice P. Histologic observations of two dental implants retrieved after osseointegration. *Int J Periodontics Restorative Dent*. 2021;41(1):121–125. doi:10.11607/prd.5102
2. Checchi V, Felice P, Zucchelli G, et al. Wide diameter immediate post-extractive implants vs delayed placement of normal-diameter implants in preserved sockets in the molar region: 1-year post-loading outcome of a randomised controlled trial. *Eur J Oral Implantol*. 2017;10(3):263–278. PMID:28944355.
3. Ephros H, Kim S, DeFalco R. Peri-implantitis: Evaluation and management. *Dent Clin North Am*. 2020;64(2):305–313. doi:10.1016/j.cden.2019.11.002
4. Checchi V, Gasparro R, Pistilli R, Canullo L, Felice P. Clinical classification of bone augmentation procedure failures in the atrophic anterior maxillae: Esthetic consequences and treatment options. *Biomed Res Int*. 2019;2019:4386709. doi:10.1155/2019/4386709
5. Berglundh T, Lindhe J, Ericsson I, Liljenberg B, Thomsen P. The soft tissue barrier at implants and teeth. *Clin Oral Implants Res*. 1991;2(2):81–90. doi:10.1034/j.1600-0501.1991.020206.x
6. Berglundh T, Lindhe J, Jonsson K, Ericsson I. The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. *J Clin Periodontol*. 1994;21(3):189–193. doi:10.1111/j.1600-051x.1994.tb00302.x
7. Heitz-Mayfield LJA, Salvi GE. Peri-implant mucositis. *J Periodontol*. 2018;89(Suppl 1):S257–S266. doi:10.1002/JPER.16-0488
8. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. *J Periodontol*. 2018;89(Suppl 1):S267–S290. doi:10.1002/JPER.16-0350
9. Passariello C, Di Nardo D, Testarelli L. Inflammatory periimplant diseases and the periodontal connection question. *Eur J Dent*. 2019;13(1):119–123. doi:10.1055/s-0039-1688525
10. Renvert S, Persson GR, Pirih FQ, Camargo PM. Peri-implant health, peri-implant mucositis, and peri-implantitis: Case definitions and diagnostic considerations. *J Periodontol*. 2018;89(Suppl 1):S304–S312. doi:10.1002/JPER.17-0588
11. Checchi V, Racca F, Bencivenni D, Lo Bianco L. Role of dental implant homecare in mucositis and peri-implantitis prevention: A literature overview. *Open Dent J*. 2019;13:470–477. doi:10.2174/1874210601913010470
12. Jepsen S, Berglundh T, Genco R, et al. Primary prevention of peri-implantitis: Managing peri-implant mucositis. *J Clin Periodontol*. 2015;42(Suppl 16):S152–S157. doi:10.1111/jcpe.12369
13. Lang NP, Wilson TG, Corbet EF. Biological complications with dental implants: Their prevention, diagnosis and treatment. *Clin Oral Implants Res*. 2000;11(Suppl 1):146–155. doi:10.1034/j.1600-0501.2000.011s1146.x
14. Miller SA, Forrest JL. Enhancing your practice through evidence-based decision making: PICO, learning how to ask good questions. *J Evid Based Dent Pract*. 2001;1(2):136–141. doi:10.1067/med.2001.118720
15. Kelly JE, Sanchez MJ; National Center for Health Statistics (U.S.). Periodontal disease and oral hygiene among children. United States. *Vital Health Stat 11. Data from the Health Examination Center*. 1972;117:1–28. PMID:4538240.
16. Meyer S, Giannopoulou C, Courvoisier C, Schimmel M, Müller F, Mombelli A. Experimental mucositis and experimental gingivitis in persons aged 70 or over. Clinical and biological responses. *Clin Oral Implants Res*. 2017;28(8):1005–1012. doi:10.1111/clr.12912
17. Coli P, Sennerby L. Is peri-implant probing causing over-diagnosis and over-treatment of dental implants? *J Clin Med*. 2019;8(8):1123. doi:10.3390/jcm8081123
18. Berglundh T, Armitage G, Araujo MG, et al. Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(Suppl 20):S286–S291. doi:10.1111/jcpe.12957
19. Figuero E, Graziani F, Sanz I, Herrera D, Sanz M. Management of peri-implant mucositis and peri-implantitis. *Periodontol 2000*. 2014;66(1):255–273. doi:10.1111/prd.12049
20. Galofré M, Palao D, Vicario M, Nart J, Violant D. Clinical and microbiological evaluation of the effect of *Lactobacillus reuteri* in the treatment of mucositis and peri-implantitis: A triple-blind randomized clinical trial. *J Periodontal Res*. 2018;53(3):378–390. doi:10.1111/jre.12523

21. Javed F, BinShabaib MS, Alharthi SS, Qadri T. Role of mechanical curettage with and without adjunct antimicrobial photodynamic therapy in the treatment of peri-implant mucositis in cigarette smokers: A randomized controlled clinical trial. *Photodiagnosis Photodyn Ther*. 2017;18:331–334. doi:10.1016/j.pdpdt.2017.04.015
22. Schwarz F, Becker K, Renvert S. Efficacy of air polishing for the non-surgical treatment of peri-implant diseases: A systematic review. *J Clin Periodontol*. 2015;42(10):951–959. doi:10.1111/jcpe.12454
23. Schwarz F, Becker K, Sager M. Efficacy of professionally administered plaque removal with or without adjunctive measures for the treatment of peri-implant mucositis. A systematic review and meta-analysis. *J Clin Periodontol*. 2015;42(Suppl 16):S202–S213. doi:10.1111/jcpe.12349
24. de Tapia B, Mozas C, Valles C, Nart J, Sanz M, Herrera D. Adjunctive effect of modifying the implant-supported prosthesis in the treatment of peri-implant mucositis. *J Clin Periodontol*. 2019;46(10):1050–1060. doi:10.1111/jcpe.13169
25. Wohlfahrt JC, Aass AM, Koldsland OC. Treatment of peri-implant mucositis with a chitosan brush – a pilot randomized clinical trial. *Int J Dent Hyg*. 2019;17(2):170–176. doi:10.1111/idh.12381
26. Ramberg P, Lindhe J, Botticelli D, Botticelli A. The effect of a triclosan dentifrice on mucositis in subjects with dental implants: A six-month clinical study. *J Clin Dent*. 2009;20(3):103–107. PMID:19711612.
27. Hallström H, Persson GR, Lindgren S, Olofsson M, Renvert S. Systemic antibiotics and debridement of peri-implant mucositis. A randomized clinical trial. *J Clin Periodontol*. 2012;39(6):574–581. doi:10.1111/j.1600-051X.2012.01884.x
28. Iorio-Siciliano V, Blasi A, Stratul SI, et al. Anti-infective therapy of peri-implant mucositis with adjunctive delivery of a sodium hypochlorite gel: A 6-month randomized triple-blind controlled clinical trial. *Clin Oral Investig*. 2020;24(6):1971–1979. doi:10.1007/s00784-019-03060-2
29. Lombardo G, Signoriello A, Corrocher G, et al. A topical desiccant agent in association with manual debridement in the initial treatment of peri-implant mucositis: A clinical and microbiological pilot study. *Antibiotics (Basel)*. 2019;8(2):82. doi:10.3390/antibiotics8020082
30. Pulcini A, Bollaín J, Sanz-Sánchez I, et al. Clinical effects of the adjunctive use of a 0.03% chlorhexidine and 0.05% cetylpyridinium chloride mouth rinse in the management of peri-implant diseases: A randomized clinical trial. *J Clin Periodontol*. 2019;46(3):342–353. doi:10.1111/jcpe.13088
31. Pimentel SP, Ribeiro FV, Casarin RC, et al. Triclosan-containing fluoride toothpaste on clinical parameters and osteo-inflammatory mediators when applied in a stent during experimental peri-implant mucositis in smokers. *Clin Oral Implants Res*. 2019;30(2):187–195. doi:10.1111/clr.13405
32. Peña M, Barallat L, Vilarrasa J, Vicario M, Violant D, Nart J. Evaluation of the effect of probiotics in the treatment of peri-implant mucositis: A triple-blind randomized clinical trial. *Clin Oral Investig*. 2019;23(4):1673–1683. doi:10.1007/s00784-018-2578-8
33. Gershenfeld L, Kalos A, Whittle T, Yeung S. Randomized clinical trial of the effects of azithromycin use in the treatment of peri-implantitis. *Aust Dent J*. 2018;63(3):374–381. doi:10.1111/adj.12614
34. Al Rifaiy MQ, Qutub OA, Alasqah MN, Al-Sowygh ZH, Mokeem SA, Alrahlah A. Effectiveness of adjunctive antimicrobial photodynamic therapy in reducing peri-implant inflammatory response in individuals vaping electronic cigarettes: A randomized controlled clinical trial. *Photodiagnosis Photodyn Ther*. 2018;22:132–136. doi:10.1016/j.pdpdt.2018.03.002
35. Ribeiro FV, Casati MZ, Casarin RC, et al. Impact of a triclosan-containing toothpaste during the progression of experimental peri-implant mucositis: Clinical parameters and local pattern of osteo-immunoinflammatory mediators in peri-implant fluid. *J Periodontol*. 2018;89(2):203–212. doi:10.1002/JPER.17-0302
36. Al Ghazal L, O'Sullivan J, Claffey N, Polyzois I. Comparison of two different techniques used for the maintenance of peri-implant soft tissue health: A pilot randomized clinical trial. *Acta Odontol Scand*. 2017;75(7):542–549. doi:10.1080/00016357.2017.1352101
37. Kashefimehr A, Pourabbas R, Faramarzi M, et al. Effects of enamel matrix derivative on non-surgical management of peri-implant mucositis: A double-blind randomized clinical trial. *Clin Oral Investig*. 2017;21(7):2379–2388. doi:10.1007/s00784-016-2033-7
38. Mongardini C, Pilloni A, Farina F, Di Tanna G, Zeza B. Adjunctive efficacy of probiotics in the treatment of experimental peri-implant mucositis with mechanical and photodynamic therapy: A randomized, cross-over clinical trial. *J Clin Periodontol*. 2017;44(4):410–417. doi:10.1111/jcpe.12689
39. Menezes KM, Fernandes-Costa AN, Silva-Neto RD, Calderon PS, Gurgel BCV. Efficacy of 0.12% chlorhexidine gluconate for non-surgical treatment of peri-implant mucositis. *J Periodontol*. 2016;87(11):1305–1313. doi:10.1902/jop.2016.160144
40. Lupi SM, Granati M, Butera A, Collesano V, Rodriguez Y Baena R. Air-abrasive debridement with glycine powder versus manual debridement and chlorhexidine administration for the maintenance of peri-implant health status: A six-month randomized clinical trial. *Int J Dent Hyg*. 2017;15(4):287–294. doi:10.1111/idh.12206
41. Riben-Grundstrom C, Norderyd O, André U, Renvert S. Treatment of peri-implant mucositis using a glycine powder air-polishing or ultrasonic device: A randomized clinical trial. *J Clin Periodontol*. 2015;42(5):462–469. doi:10.1111/jcpe.12395
42. Hallström H, Lindgren S, Twetman S. Effect of a chlorhexidine-containing brush-on gel on peri-implant mucositis. *Int J Dent Hyg*. 2017;15(2):149–153. doi:10.1111/idh.12184
43. Hallström H, Lindgren S, Widén C, Renvert R, Twetman S. Probiotic supplements and debridement of peri-implant mucositis: A randomized controlled trial. *Acta Odontol Scand*. 2016;74(1):60–66. doi:10.3109/00016357.2015.1040065
44. Flichy-Fernández AJ, Ata-Ali J, Alegre-Domingo T, et al. The effect of orally administered probiotic *Lactobacillus reuteri*-containing tablets in peri-implant mucositis: A double-blind randomized controlled trial. *J Periodontal Res*. 2015;50(6):775–785. doi:10.1111/jre.12264
45. McKenna DF, Borzabadi-Farahani A, Lynch E. The effect of subgingival ozone and/or hydrogen peroxide on the development of peri-implant mucositis: A double-blind randomized controlled trial. *Int J Oral Maxillofac Implants*. 2013;28(6):1483–1489. doi:10.11607/jomi.3168
46. Heitz-Mayfield LJA, Salvi GE, Botticelli D, Mombelli A, Faddy M, Lang NP; Implant Complication Research Group. Anti-infective treatment of peri-implant mucositis: A randomised controlled clinical trial. *Clin Oral Implants Res*. 2011;22(3):237–241. doi:10.1111/j.1600-0501.2010.02078.x
47. Serino G, Wada M. Non-surgical mechanical treatment of peri-implant mucositis: The effect of sub-mucosal mechanical instrumentation following supra-mucosal plaque removal. A 7-month prospective single cohort study. *Eur J Oral Implantol*. 2018;11(4):455–466. PMID:30515485.
48. Gomes SC, Corvello P, Romagna R, Müller LH, Melchiors Angst PD, Oppermann RV. How do peri-implant mucositis and gingivitis respond to supragingival biofilm control – an intra-individual longitudinal cohort study. *Eur J Oral Implantol*. 2015;8(1):65–73. PMID:25738180.
49. Zeza B, Farina R, Pilloni A, Mongardini C. Clinical outcomes of experimental gingivitis and peri-implant mucositis treatment with professionally administered plaque removal and photodynamic therapy. *Int J Dent Hyg*. 2018;16(2):e58–e64. doi:10.1111/idh.12302
50. Al Amri MD, Kellesarian SV, Ahmed A, Al-Kheraif AA, Romanos GE, Javed F. Efficacy of periimplant mechanical debridement with and without adjunct antimicrobial photodynamic therapy in patients with type 2 diabetes mellitus. *Photodiagnosis Photodyn Ther*. 2016;14:166–169. doi:10.1016/j.pdpdt.2016.04.015
51. Lerario F, Roncati M, Gariffo A, et al. Non-surgical periodontal treatment of peri-implant diseases with the adjunctive use of diode laser: Preliminary clinical study. *Lasers Med Sci*. 2016;31(1):1–6. doi:10.1007/s10103-015-1785-7
52. Chan D, Pelekos G, Ho D, Cortelini P, Tonetti MS. The depth of the implant mucosal tunnel modifies the development and resolution of experimental peri-implant mucositis: A case–control study. *J Clin Periodontol*. 2019;46(2):248–255. doi:10.1111/jcpe.13066